August 2010

Regulatory Impact Analysis: Amendments to the National Emission Standards for Hazardous Air Pollutants and New Source Performance Standards (NSPS) for the Portland Cement Manufacturing Industry

Final Report

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards (OAQPS)
Air Benefit and Cost Group
(MD-C439-02)
Research Triangle Park, NC 27711

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SECTION 1
INTRODUCTION

The U.S. Environmental Protection Agency (EPA) is finalizing amendments to the National Emission Standards for Hazardous Air Pollutants (NESHAP) from the Portland cement manufacturing industry and New Source Performance Standards (NSPS) for Portland cement plants. The final amendments to the NESHAP add or revise, as applicable, emission limits for mercury (Hg), total hydrocarbons (THC), and particulate matter (PM) from kilns located at a major or an area sources, and hydrochloric acid (HCl) from kilns and located at major sources. EPA is also adopting separate standards for these pollutants that apply during startup, shutdown, and operating modes. Finally, EPA is adopting performance specifications for use of Hg continuous emission monitors (CEMS) and updating recordkeeping and testing requirements. The final amendments to the NSPS add or revise, as applicable, emission limits for particulate matter (PM), opacity, nitrogen oxides (NO_x), and sulfur dioxide (SO_2) for facilities that commence construction, modification, or reconstruction after June 16, 2008. The final rule also includes additional testing and monitoring requirements for affected sources. As part of the regulatory process, EPA is required to develop a regulatory impact analysis (RIA). The RIA includes an economic impact analysis (EIA) and a small entity impacts analysis and documents the RIA methods and results.

1.1 Executive Summary

The key results of the RIA are as follows:

- **Options Analyzed:** EPA's analysis focuses on the results of the final NESHAP and NSPS. We also present additional information on different combinations of the regulatory programs to help stakeholders better understand the size and scope of each. These include
  - final NSPS only,
  - final NESHAP only, and
  - alternative: more stringent NSPS and final NESHAP.

The rest of this summary addresses the results of analyzing the final NESHAP and NSPS.

- **Engineering Cost Analysis:** EPA estimates that total annualized costs with the final NESHAP and NSPS will be $466 million (2005$).
- **Market Analysis:** The partial-equilibrium economic model suggests the average national price for Portland cement could be 5% higher with the NESHAP, or $4.50 per metric ton, while annual domestic production may fall by 11%, or 10 million tons

per year. Because of higher domestic prices, imports rise by 10%, or 3 million metric tons per year.

- **Industry Analysis:** Net industry operating profits fall by $241 million; EPA also identified 10 domestic plants with negative operating profits and significant utilization changes that could temporarily idle until market demand conditions improve. The plants have unit compliance costs close to $8 per ton of clinker capacity and $116 million total change in operating profits. Since these plants account for approximately 8% of domestic capacity, a decision to permanently shut down these plants would reduce domestic supply and could lead to additional projected market price increases and reductions in pollution control costs.

- **Employment Changes**: EPA uses two methods for estimating employment impacts. A simplistic, limited assessment narrowly focused on output changes in the Portland cement industry indicates that the final rule's gross impact on employment is 1,500 job losses. However, this approach inherently overstates job losses, as it is based on the assumption that employment is proportional to output, and because it ignores offsetting general equilibrium and other effects as discussed in detail in Chapter 3. A more sophisticated analytical approach that includes other types of employment effects estimates changes in net employment could range from a loss of 600 to a net gain of 1,300 jobs.

- **Social Cost Analysis:** The estimated social cost is $926 to $950 million (2005$). The range represents the estimated difference in surplus if ten facilities with low estimated post regulation capacity utilization choose to idle or close rather than operate at a low (55.5. percent) capacity utilization. The social cost estimates are significantly higher than the engineering analysis estimates, which estimated annualized costs of $466 million. This is a direct consequence of EPA's assumptions about existing market structure discussed extensively in previous cement industry rulemakings and Section 2, Appendix A, and Appendix B of this RIA. Under baseline conditions without regulation, the existing domestic cement plants are assumed to choose a production level that is less than the level produced under perfect competition. As a result, a preexisting market distortion exists in the markets covered by the final rule (i.e., the observed baseline market price is higher than the [unobserved] market price that a model of perfect competition would predict). The imposition of additional regulatory costs tends to widen the gap between price and marginal cost in these markets and contributes to additional social costs.

- **Energy Impacts:** EPA concludes that the rule when implemented will not have a significant adverse effect on the supply, distribution, or use of energy. The cement industry accounts for less than 0.4% of the U.S. total energy use. EPA estimates the additional add-on controls may increase national electrical demand by 780 million kWh per year and the natural gas use to be 1.0 million MMBTU per year for existing kilns. For new kilns, assuming that of the 16 new kilns to start up by 2013 all 16will add alkaline scrubbers and ACI systems, the electrical demand is estimated to be 199 million kWh per year. This is less than 0.1% of AEO 2010 forecasts of total electricity and natural gas consumption.

- **Small Business Analysis:** Only 4 of the over 40 cement parent companies are small entities. EPA performed a screening analysis for impacts on the 4 small entities by comparing compliance costs to average company revenues. EPA's analysis found that the ratio of compliance cost to company revenue falls below 1% for two of the four small entities (includes a Tribal government). Two small entities would have an annualized cost of between 1% and 3% of sales. No small businesses would have an annualized cost greater than 3% of sales.

- **Benefits Analysis:** In the year of full implementation (2013), EPA estimates that the total monetized benefits of the final NESHAP and NSPS are $7.4 billion to $18 billion and $6.7 billion to $16 billion, at 3% and 7% discount rates, respectively (Table 1-1). All estimates are in 2005 dollars for the year 2013. Using alternate relationships between $PM_{2.5}$ and premature mortality supplied by experts, higher and lower benefits estimates are plausible, but most of the expert-based estimates fall between these estimates. Due to data, methodology, and resource limitations, the benefits from reducing other air pollutants have not been monetized in this analysis, including reducing 4,400 tons of NO_x, 5,200 tons of organic hazardous air pollutants (HAPs), 5,900 tons of HCl, and 16,400 pounds of Hg each year. In addition, ecosystem benefits and visibility benefits have not been monetized in this analysis. These estimates include the energy disbenefits associated with increased electricity usage by the control devices.

- **Net Benefits:** In the year of full implementation (2013), EPA estimates the net benefits of the final NESHAP and NSPS are approximately $6.5 billion to $17 billion and $5.8 billion to $15 billion, at 3% and 7% discount rates, respectively. All estimates are in 2005 dollars for the year 2013.

1.2 Organization of this Report

The remainder of this report supports and details the methodology and the results of the EIA:

- Section 2 presents a profile of the affected industry.

- Section 3 describes the economic impact analysis and energy impacts.

- Section 4 describes the small business impact analysis.

- Section 5 presents the air quality modeling of emission reductions.

- Section 6 presents the benefits analysis.

- Appendix A provides an overview of the economic impact model.

- Appendix B discusses the model of the cement plant's production decision.

- Appendix C presents the social cost methodology.

Table 1-1. Summary of the Monetized Benefits, Social Costs, and Net Benefits for the Final Portland Cement NESHAP in 2013 (millions of 2005$)[a]

Final NESHAP and NSPS						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[b]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[c]	$926	to	$950	$926	to	$950
Net Benefits	$6,500	to	$17,000	$5,800	to	$15,000
Nonmonetized Benefits[d]	4,400 tons of NO_x (includes energy disbenefits) 5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					
Final NSPS only						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[b]	$510	to	$1,300	$460	to	$1,100
Total Social Costs[c]	$72			$72		
Net Benefits	$440	to	$1,200	$390	to	$1,000
Nonmonetized Benefits[d]	6,600 tons of NO_x 520 tons of HCl Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					
Final NESHAP only						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[b]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[c]	$904	to	$930	$904	to	$930
Net Benefits	$6,500	to	$17,000	$5,800	to	$16,000
Nonmonetized Benefits[d]	5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, SO_2 exposure Ecosystem effects Visibility impairment					
Alternative: More Stringent NSPS and Final NESHAP						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[b]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[c]	$955	to	$979	$955	to	$979
Net Benefits	$6,500	to	$17,000	$5,700	to	$15,000
Nonmonetized Benefits[d]	7,800 tons of NO_x (includes energy disbenefits) 5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					

[a] All estimates are for the implementation year (2013) and are rounded to two significant figures.

[b] The total monetized benefits reflect the human health benefits associated with reducing exposure to $PM_{2.5}$ through reductions of directly emitted $PM_{2.5}$ and $PM_{2.5}$ precursors such as SO_2. It is important to note that the monetized benefits include many but not all health effects associated with $PM_{2.5}$ exposure. Benefits are shown as a range from Pope et al. (2002) to Laden et al. (2006). These models assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality because there is no clear scientific evidence that would support the development of differential effects estimates by particle type. The total monetized benefits include the energy disbenefits.

[c] The methodology used to estimate social costs for 1 year in the multimarket model using surplus changes results in the same social costs for both discount rates. Range represents the estimated difference in surplus if ten facilities with low estimated post regulation capacity utilization choose to idle or close rather than operate at a low (55.5 percent) capacity utilization.

[d] Due to data, methodology, and resource limitations, we were unable to monetize the benefits associated with these categories of benefits.

SECTION 2
INDUSTRY PROFILE

Hydraulic cement (primarily Portland cement) is a key component of an important construction material: concrete. Concrete is used in a wide variety of applications (e.g., residential and commercial buildings, public works projects), and cement demand is influenced by national and regional trends in these sectors. Recent data for 2007 show that the U.S. cement industry produced over 90 million metric tons of Portland cement (Department of Interior [DOI], U.S. Geological Survey [USGS], 2008b). The value of total U.S. sales, including imported cement, was about $11.8 billion, with an average value of approximately $100 per metric ton. The vast majority of cement sales went to ready-mixed concrete producers and concrete product manufacturers (88%). Since 2003, the United States has relied on cement imports to meet approximately 20% to 23% of its consumption needs. However, this share dropped to approximately 17% in 2007 as overall construction demand for cement fell (DOI, USGS, 2008b).

The remainder of this section provides an introduction to the Portland cement industry. The purpose is to give the reader a general understanding of the technical and economic aspects of the industry that must be addressed in the economic impact analysis. Section 2.1 provides an overview of the production processes and costs data. Section 2.2 discusses the uses, consumers, and substitutes for cement. Section 2.3 summarizes the organization of the Portland cement industry. The industry profile concludes with a discussion of historical market data and the current industry outlook.

2.1 The Supply Side

2.1.1 Production Process

As shown in Figure 2-1, the manufacturing process of an integrated cement plant includes

- quarrying and crushing the raw materials,
- grinding the carefully proportioned materials to a high degree of fineness,
- firing the raw materials mixture in a rotary kiln to produce clinker, and
- grinding the resulting clinker to a fine powder and mixing with gypsum to produce cement.

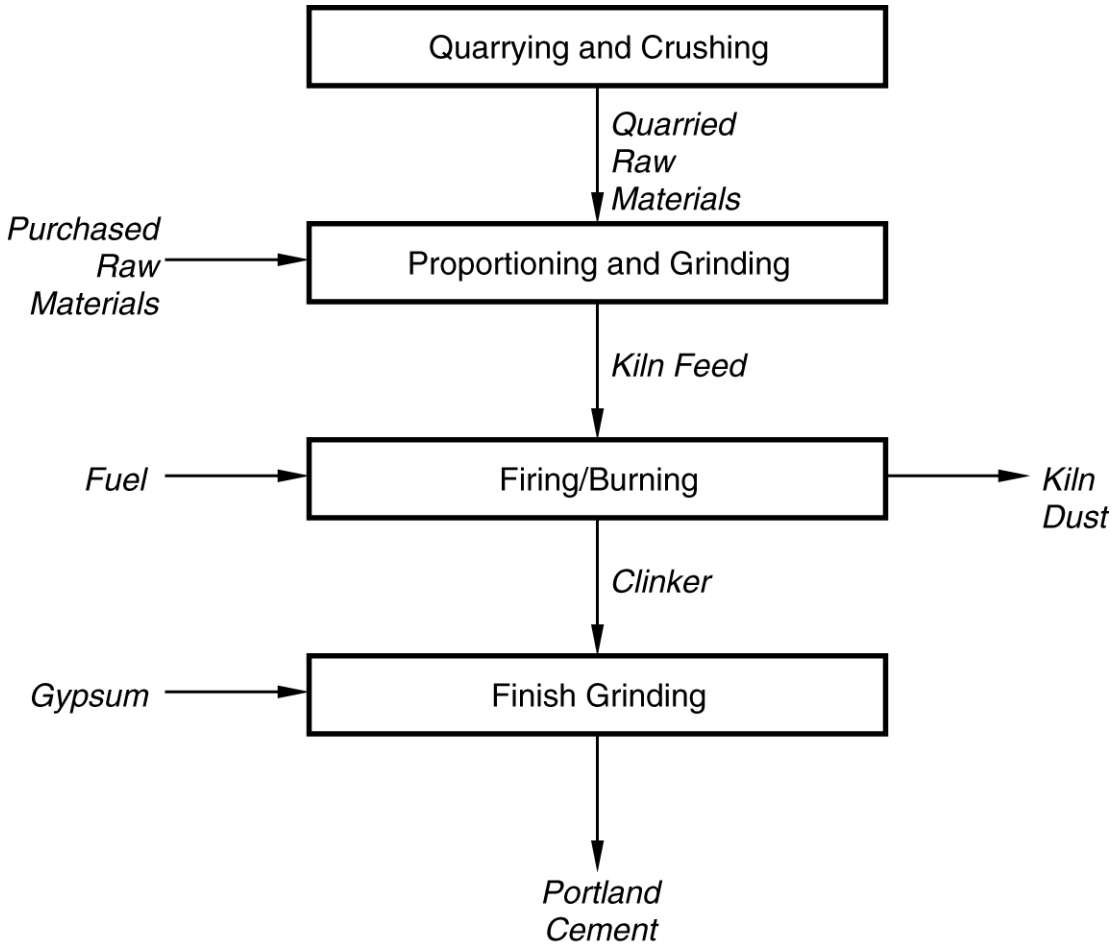

Figure 2-1. Simplified Flow Sheet of Clinker and Cement Manufacture

There are two processes for manufacturing cement: the wet process and the dry process. In the wet process, water is added to the raw materials during the blending process and before feeding the mixture into the rotary kiln. In contrast, the dry process feeds the blended materials directly into the rotary kiln in a dry state. Newer dry process plants also use preheater and precalciner technologies that partially heat and calcine the blended raw materials before they enter the rotary kiln. These technologies can increase the overall energy efficiency of the cement plant and reduce production costs.

The fuel efficiency differences between the wet and dry processes have led to a substantial decline in clinker capacity provided by the wet process over the last 3 decades. Historical data show capacity shares falling from 52% in 1980 to approximately 22% in 2000 (Van Oss and Padovani, 2002). Data also show that the number of wet process plants fell from 32 in 2000 to 23 in 2005 (DOI, USGS, 2007).

2.1.2 Types of Portland Cement

Portland cement manufacturers produce a variety of types of cement in the United States designed to meet different requirements. The American Society for Testing Materials (ASTM) specification C-150 provides for eight types of Portland cement: five standard types (I, II, III, IV, V) and three additional types that include air-entraining properties (IA, IIA, IIIA) (PCA, 2008a). We describe these below.

Types I and IA: These types are the usual product used in general concrete construction, most commonly known as gray cement because of its color.

Types II and IIA: These types are intended for use when moderate heat of hydration is required or for general concrete construction exposed to moderate sulfate action.

Type III and IIIA: These types are made from raw materials with a lime-to-silica ratio higher than that of Type I cement and are ground finer than Type I cements. They contain a higher proportion of tricalcium silicate than regular Portland cements.

Type IV: This type contains a lower percentage of tricalcium silicate and tricalcium aluminate than Type I, thus lowering the heat evolution. Consequently, the percentage of tetracalcium aluminoferrite is increased. Type IV cements are produced to attain a low heat of hydration.

Type V: This type resists sulfates better than the other four types.

As shown in Table 2-1, the vast majority of Portland cement shipments[1] in 2005 were Types I and II grey cement. However, Type V (sulfate-resisting) is a growing market (DOI, USGS, 2007a); since 2000, Type V cement has increased its share of shipments from 4% to 15%. Shipment shares for other types of cement remained constant during this period.

2.1.3 Production Costs

Portland cement is produced using a combination of variable inputs such as raw materials, labor, electricity, and fuel. U.S. Census data for the cement industry (North American Industry Classification System [NAICS] 32731: cement manufacturing) provides an initial overview of aggregated industry expenditures on these inputs (Department of Commerce [DOC], Bureau of the Census, 2010). In 2007, the total value of shipments was $10.6 billion, and the industry spent approximately $1.7 billion on materials, parts, and packaging, or 16% of the value of shipments. Total compensation for all employees (includes payroll and fringe benefits)

[1] USGS notes these shipment data include cement imports (primarily Types I, II, and V).

Table 2-1. Portland Cement Shipped from Plants in the United States to Domestic Customers, by Type[a, b]

Type	2000	Share	2005	Share
General use and moderate heat (Types I and II) (gray)[c]	90,644	88%	93,900	77%
High early strength (Type III)	3,815	4%	3,960	3%
Sulfate resisting (Type V)[c]	4,453	4%	18,100	15%
White[d]	894	1%	1,190	1%
Blended	1,296	1%	3,160	3%
Expansive and regulated fast setting	60	0%	6	0%
Other[e]	1,786	2%	1,997	2%
Total[f]	102,947	100%	122,000	100%

[a] Includes imported cement.

[b] Data are rounded to no more than three significant digits; may not add to totals shown.

[c] Cements classified as Type II/V hybrids are now commonly reported as Type V.

[d] Mostly Types I and II but may include Types III through V and block varieties.

[e] Includes block, oil well, low heat (Type IV), waterproof, and other Portland cements.

[f] Data are based on an annual survey of plants and importers.

Sources: U.S. Department of the Interior, U.S. Geological Survey. 2007a. *2005 Minerals Yearbook, Cement.* Washington, DC: U.S. Department of the Interior. Table 15.

U.S. Department of the Interior, U.S. Geological Survey. 2002. *2001 Minerals Yearbook, Cement.* Washington, DC: U.S. Department of the Interior. Table 15.

amounted to $1.4 billion (13%).[1] Fuels and electricity expenditures were approximately $1.7 billion (16%).

2.1.3.1 Raw Material Costs

According to the USGS, approximately 159.7 million tons of raw materials were required to produce approximately 95.5 million tons of cement in 2005 or 1.67 tons of raw materials per ton of cement. Table 2-2 summarizes the amount of raw material inputs used per ton of cement produced in the United States between 2000 and 2005. As the data show, the amount of raw materials required to produce one ton of cement has remained essentially constant during this 6-year period.

[1] Wages paid to production workers were $0.8 billion (8% of the value of shipments) at an average hourly rate of $27.

Table 2-2. Raw Material Input Ratios for the U.S. Cement Industry: 2000 to 2005

	2000	2001	2002	2003	2004	2005
Raw material input (10^3 metric tons)	144,949	147,300	153,100	150,500	158,200	159,700
Cement production (10^3 metric tons)	85,178	86,000	86,817	89,592	94,014	95,488
Metric tons of raw material input per ton of cement	1.70	1.71	1.76	1.68	1.68	1.67

Sources: U.S. Department of the Interior, U.S. Geological Survey. 2002–2007a. *2001–2005 Minerals Yearbook, Cement*. Table 6. Washington, DC: U.S. Department of the Interior.

U.S. Department of the Interior, U.S. Geological Survey. 2002–2007a. *2001–2005 Minerals Yearbook, Cement*. Table 3. Washington, DC: U.S. Department of the Interior.

The price of these raw materials varies across regions. Table 2-3 lists the average price of raw materials per metric ton by state. In 2005, the prices of raw materials were highest in Hawaii where they sold for an average of $13.34 per metric ton. The prices of raw materials were lowest in Michigan, where they sold for an average of $3.89 per metric ton.

2.1.3.2 Labor Costs

In 2005, the Portland Cement Association (PCA) reported labor productivity measures (in terms of metric tons of cement per employee hour)[1] for 2000 to 2005 in its U.S. and Canadian Labor-Energy Input Survey. Using these data, we computed a measure of labor hour requirements to produce cement (see Table 2-4). As these data show, wet process plants are typically more labor intensive, requiring approximately 45% more labor hours to produce a metric ton of cement than dry process plants.

In addition, labor productivity has been improving more quickly in dry process plants than in those using a wet manufacturing process. Between 2000 and 2005, labor requirements decreased by 15% in dry process plants, while in wet process plants labor requirements remained constant. As a result, the wet process labor costs relative to dry process plants labor costs have risen in recent years (Figure 2-2).[2]

[1] Throughout this report, we use PCA's method to calculate labor and energy efficiency. This measure is a weighted sum of clinker and finished cement production. Weights for labor are 85% clinker and 15% finished cement production. Weights for energy are 92% clinker and 8% finished cement production (PCA, 2005).

[2] The labor costs reported in Figure 2-3 were calculated by first multiplying the number of employee hours per metric ton of cement reported in Table 2-4 by the average hourly earnings of production workers for each year (BLS, 2007a and 2007b). Next, these cost estimates were adjusted for inflation and expressed in 2005 dollars by using the consumer price index (CPI) (BLS, 2008).

Table 2-3. Raw Material Costs by Market and State: 2005

State(s)	Price of Raw Materials ($/metric ton)[a]	State(s)	Price of Raw Materials ($/metric ton)[a]
AK	6.60	MT	$4.76
AL	6.57	NC	$8.59
AR	$6.29	ND	$4.45
AZ	$5.75	NE	$7.10
CA	$8.37	NH	$8.02
CO	$6.85	NJ	$7.04
CT	$9.19	NM	$6.67
DE	$6.89	NV	$7.17
FL	$8.67	NY	$8.44
GA	$7.63	OH	$5.82
HI	$13.34	OK	$5.67
IA	$7.27	OR	$6.01
ID	$5.37	PA	$6.67
IL	$7.16	RI	$7.74
IN	$5.40	SC	$7.61
KS	$7.20	SD	$4.60
KY	$7.24	TN	$7.55
LA	$8.18	TX	$6.15
MA	$9.19	UT	$5.58
MD	$8.28	VA	$9.03
ME	$6.85	VT	$6.75
MI	$3.89	WA	$6.92
MN	$8.30	WI	$5.83
MO	$7.37	WV	$6.86
MS	$11.90	WY	$5.68

Source: U.S. Department of the Interior, U.S. Geological Survey. 2007b. *2005 Minerals Yearbook, Crushed Stone.* Table 4. Washington, DC: U.S. Department of the Interior.

Table 2-4. Labor Productivity Measures for the U.S. Cement Industry by Process Type: 2000 to 2005 (employee hours per metric ton)

Year	2000	2001	2002	2003	2004	2005
All plants	0.394	0.388	0.360	0.347	0.338	0.338
Wet process	0.469	0.457	0.450	0.465	0.452	0.463
Dry process	0.376	0.375	0.342	0.328	0.318	0.318

Source: Portland Cement Association. December 2005. U.S. and Canadian Labor-Energy Input Survey 2005. Skokie, IL: PCA's Economic Research Department.

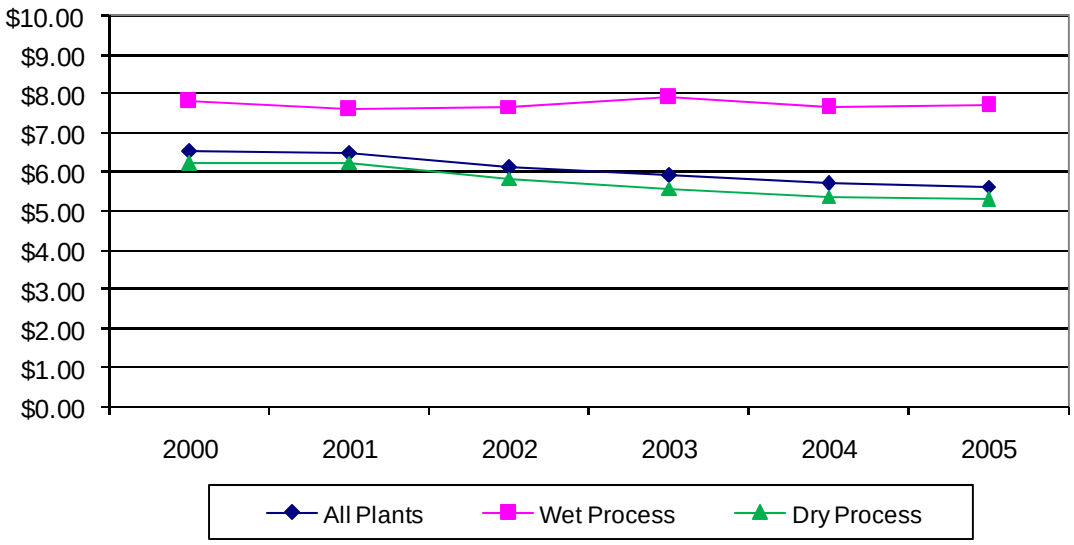

Figure 2-2. Labor Costs per Metric Ton of Cement ($2005)

Sources: Portland Cement Association. December 2005. *U.S. and Canadian Labor-Energy Input Survey 2005*. Skokie, IL: PCA's Economic Research Department.

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2007a. "Current Employment Statistics (National): Customizable Data Tables" Available at <http://www.bls.gov/ces/>. As obtained on March 14, 2008.

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2008. "Consumer Price Index All Items – U.S. City Average Data: Customizable Data Tables." Available at <http://www.bls.gov/cpi/>. As obtained on March 14, 2008.

2.1.3.3 Energy Costs

Figure 2-3 provides a detailed breakdown of U.S. energy consumption in 2005. As this figure shows, the vast majority of energy in U.S. cement plants is derived from coal and coke (75%). The remaining 25% of energy consumption is derived from electricity, waste, natural gas, and petroleum products.

PCA also reported energy consumption data by type of U.S. cement plant (in terms of millions of BTUs per metric ton of cement) (see Table 2-5). As these data show, wet process plants are typically more energy intensive, consuming approximately 44% more energy per ton of cement than dry process plants. In addition, the trends in energy consumption continue to show that dry plants have become more energy efficient than wet process plants. Between 2000 and 2005, energy consumption per ton of cement in dry process plants *decreased* by 5%; in contrast, wet process plants' energy consumption increased slightly during this period.

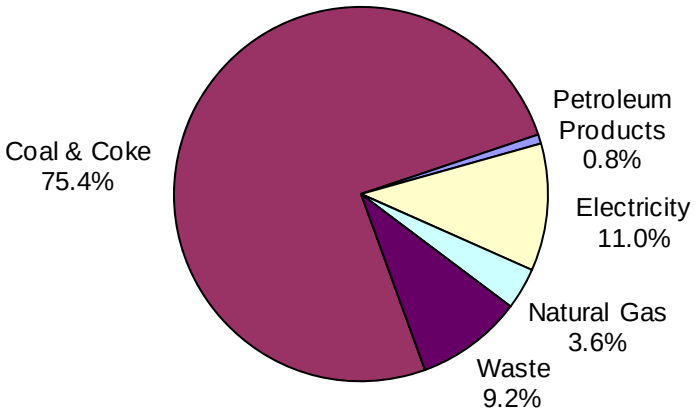

Figure 2-3. Distribution of Energy Consumption

Source: Portland Cement Association. December 2005. *U.S. and Canadian Labor-Energy Input Survey 2005*. Skokie, IL: PCA's Economic Research Department.

Table 2-5. Energy Consumption by Type of U.S. Cement Plant (million BTU per metric ton)

Year	2000	2001	2002	2003	2004	2005
All plants	4.982	4.93	4.858	4.762	4.755	4.699
Wet process	6.25	6.442	6.676	6.647	6.807	6.387
Dry process	4.673	4.655	4.498	4.433	4.407	4.433

Source: Portland Cement Association. December 2005. *U.S. and Canadian Labor-Energy Input Survey 2005*. Skokie, IL: PCA's Economic Research Department.

2.2 The Demand Side

The demand for Portland cement is considered a "derived" demand because it depends on the construction demands for its end product—concrete. A recent study by the U.S. International Trade Commission suggests that 0.192 metric tons of grey Portland cement were used per $1,000 of construction in 1998 (USITC, 2006). Given cement prices at this time (approximately $75 per metric ton), Portland cement costs represented only a small share of the total value of construction expenditures (less than 2%).

Concrete is used in a wide variety of construction applications, including residential and commercial buildings, and public works projects such as the national highway system. As shown in Figure 2-4, ready-mixed concrete producers have historically accounted for over half of the Portland cement consumption. Although government and miscellaneous expenditures saw substantial increases in the early 1990s, their consumption share returned to pre-1990s levels after 1996. The latest USGS use data show that ready-mixed concrete producers accounted for

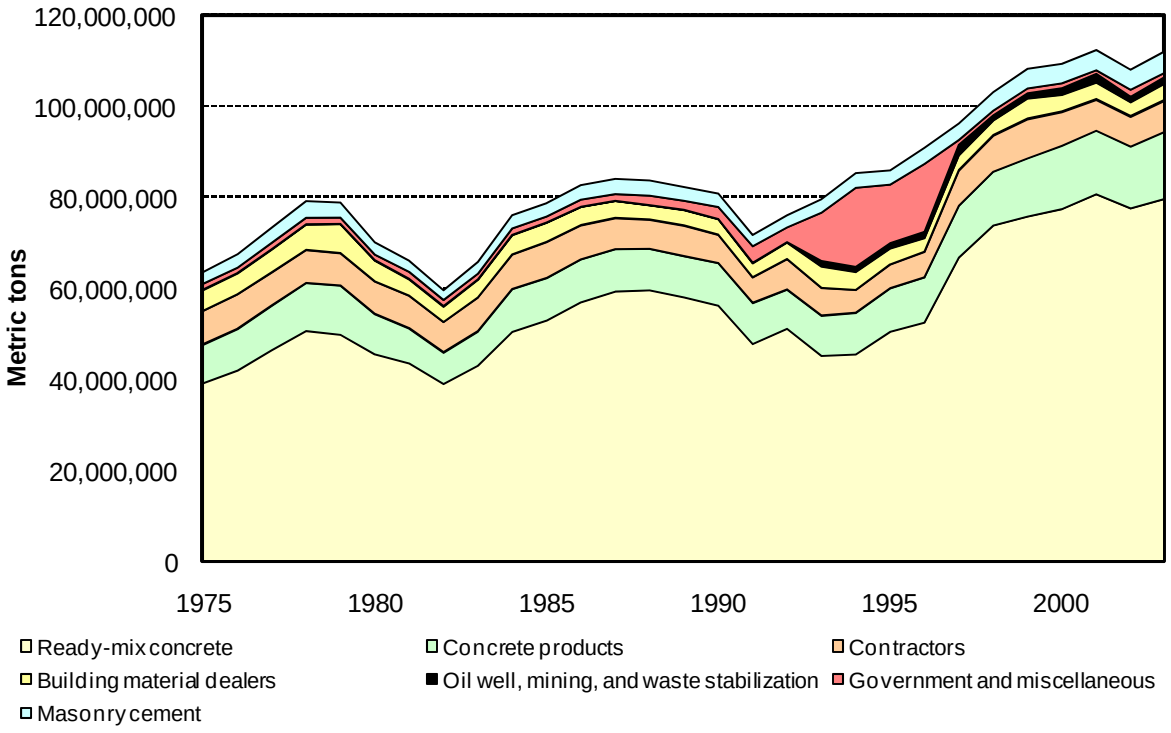

Figure 2-4. End Uses of Cement: 1975 to 2003

Source: Kelly, T. and G. Matos. 2007a. "Historical Statistics for Mineral and Material Commodities in the United States: Cement End Use Statistics." U.S. Geological Survey Data Series 140, Version 1.2. Available at http://minerals.usgs.gov/ds/2005/140/.

74% of cement sales in 2005, followed by concrete product manufacturers (14%), contractors (6%), and other (6%) (Kelly and Matos, 2007a).

Cement competes with other construction materials such as steel, asphalt, and lumber. Lumber is the primary substitute in the residential construction market, while steel is the primary substitute in commercial applications. Asphalt is a key substitute in transportation projects such as road and parking lot surfacing. However, concrete has advantages over these substitutes because it tends to be available locally and has lower long-term maintenance costs (Van Oss and Padovani, 2002).

The PCA regularly reports price trends for these competing building materials (PCA, 2008b). As shown in Figure 2-5, steel and asphalt have risen sharply relative to cement since 2003 while lumber has declined.

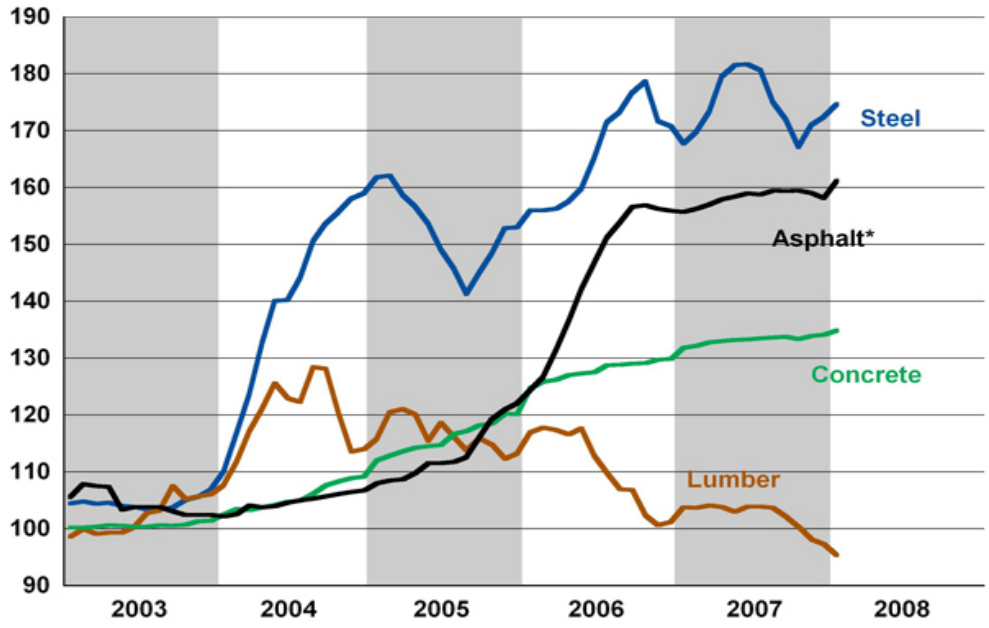

Figure 2-5. Producer Price Indices for Competitive Building Materials: 2003 to 2008

Source: Portland Cement Association. 2008b. "Market Research: Producer Price Indices—Competitive Building Materials." Available at <http://www.cement.org/market/>.

2.3 Industry Organization

2.3.1 Market Structure

A review and description of market characteristics (i.e., degree of concentration, entry barriers, and product differentiation) can enhance our understanding of how U.S. cement markets operate. These characteristics provide indicators of a firm's ability to influence market prices by varying the quantity of cement it sells. For example, in markets with large numbers of sellers and identical products, firms are unlikely to be able to influence market prices via their production decisions (i.e., they are "price takers"). However, in markets with few firms, significant barriers to entry (e.g., licenses, legal restrictions, or high fixed costs), or products that are similar but can be differentiated, the firm may have some degree of market power (i.e., set or significantly influence market prices).

Cement sales are often concentrated locally among a small number of firms for two reasons: high transportation costs and production economies of scale.[1] Transportation costs significantly influence where cement is ultimately sold; high transportation costs relative to unit value provide incentives to produce and sell cement locally in regional markets (USITC, 2006).

[1] The 2002 Economic Census reports that the national Herfindahl-Hirschman Index (HHI) for cement (North American Industry Classification System [NAICS] 32731) is 568. However, this measure is likely not representative of actual concentration that exists in regional markets.

To support this claim, the empirical literature has typically pointed to Census of Transportation data showing over 80% of cement shipments were made within a 200-mile radius (Jans and Rosenbaum, 1997)[1] and reported evidence of high transportation costs per dollar of product value from case studies (Ryan, 2006). The cement industry is also very capital intensive and entry requires substantial investments. In additional, large plants are typically more economical because they can produce cement at lower unit costs; this reduces entry incentives for small-sized cement plants. Using recent data for planned capacity expansions between 2008 and 2012, the PCA reports these expansions will cost $5.9 billion and add 25 million metric tons (PCA, 2007), or $240 per metric ton, of new capacity.

For a given construction application, consumers are likely to view cement produced by different firms as very good substitutes. American Society for Testing and Materials (ASTM) specifications tend to ensure uniform quality, and recent industry reviews (USITC, 2006) suggest that there is little or no brand loyalty that allows firms to differentiate their products.

2.3.2 Manufacturing Plants

During 2005, 107 cement manufacturing plants with 186 cement kilns were operating in the United States. This section describes the location, age, production capacity, and employment of these manufacturing facilities. Section 2.3.2 concludes with a discussion of future trends. Section 2.3.3 provides a detailed discussion of the characteristics of the firms owning these facilities.

2.3.2.1 Location

Table 2-6 summarizes the geographic location of cement kilns in the United States and clinker capacity. The top five states in order of clinker capacity are California, Texas, Pennsylvania, Florida, and Alabama. Together these states account for 75 (40%) of the kilns in the United States and 41 million metric tons (44%) of clinker capacity. Figure 2-6 provides a graphical depiction of the number of kilns distributed by state.

Fourteen states (Alaska, Hawaii, Connecticut, Louisiana, New Hampshire, North Dakota, Wisconsin, Delaware, Massachusetts, New Jersey, Rhode Island, Minnesota, North Carolina, and Vermont) and the District of Columbia had no clinker-producing facilities in 2005.

[1] A recent USITC study of California cement markets found more than 75% of gray Portland cement shipments in the state were shipped to customers within 200 miles of the cement producer (USITC, 2006).

Table 2-6. Number of Kilns and Clinker Capacity by State: 2005

	No. Kilns	Clinker Capacity (10^3 metric tons per year)
AK	0	
AL	5	5,375
AR	3	831
AZ	8	2,809
CA	20	12,392
CO	2	2,117
CT	0	
DE	0	
FL	7	5,489
GA	2	1,020
HI	0	
IA	4	2,672
ID	2	260
IL	8	2,770
IN	8	3,191
KS	9	2,835
KY	1	1,365
LA	0	
MA	0	
MD	4	2,538
ME	1	392
MI	8	4,243
MN	0	
MO	6	5,169
MS	1	419
MT	2	573
NC	0	
ND		
NE	2	845
NH	0	
NJ	0	
NM	2	432
NV	2	452
NY	4	2,886
OH	3	1,115
OK	7	1,869
OR	1	816

(continued)

Table 2-6. Number of Kilns and Clinker Capacity by State: 2005 (continued)

	No. Kilns	Clinker Capacity (10^3 metric tons per year)
PA	21	6,414
RI	0	
SC	6	3,480
SD	3	851
TN	2	1,438
TX	22	11,688
UT	2	1,514
VA	1	1,120
VT	0	
WA	2	1,100
WI	0	
WV	3	708
WY	2	597
Total	186	93,785

Source: Portland Cement Association (PCA). 2004. U.S. and Canadian Portland Cement Industry: Plant Information Summary. Skokie, IL: PCA's Economic Research Department.

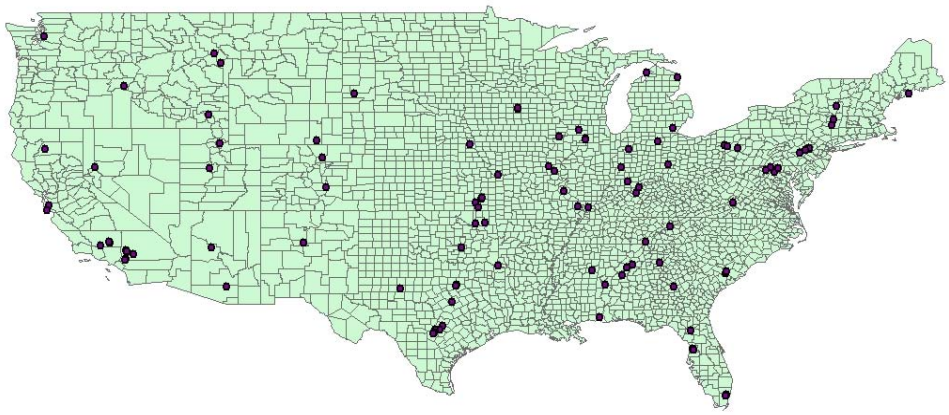

Figure 2-6. Distribution of Cement Kilns in the United States

Source: Portland Cement Association (PCA). December 2004. *U.S. and Canadian Portland Cement Industry: Plant Information Summary*. Skokie, IL: Portland Cement Association Economic Research Department.

2.3.2.2 Age

In 2005, 72% (134) of all kilns in the United States used the dry manufacturing process, and it accounted for 83% (78 million metric tons) of national clinker capacity. The growing prevalence of the dry process among cement manufacturers is part of a long-term trend. As the data in Table 2-7 indicate, no new wet clinker capacity has been added within the past 30 years.

Table 2-7. Number of Kilns and Clinker Capacity by Age and Process Type

	No. Kilns	Clinker Capacity (10^3 metric tons per year)	Average Annual Capacity per Kiln
Total			
0–10	26	28,144	1,082.5
11–15	3	2,176	725.3
16–20	5	3,345	669.0
21–25	16	14,982	936.4
26–30	18	11,843	657.9
31–35	16	5,786	361.6
36–40	21	9,285	442.1
41–45	29	8,971	309.3
46–50	32	6,564	205.1
51–55	6	991	165.2
56–60	6	800	133.3
60+	8	898	112.3
Total	186	93,785	504.2
Dry Process			
0–10	26	28,144	1,082.5
11–15	3	2,176	725.3
16–20	5	3,345	669.0
21–25	16	14,982	936.4
26–30	18	11,843	657.9
31–35	10	3,962	396.2
36–40	12	5,498	458.2
41–45	14	3,800	271.4
46–50	16	2,651	165.7
51–55	4	682	170.5
56–60	6	800	133.3
60+	4	328	82.0
Total	134	78,211	583.7
Wet Process			
0–10	0		
11–15	0		
16–20	0		
21–25	0		
26–30	0		
31–35	6	1,824	304.0
36–40	9	3,787	420.8
41–45	15	5,171	344.7

(continued)

Table 2-7. Number of Kilns and Clinker Capacity by Age and Process Type (continued)

	No. Kilns	Clinker Capacity (10^3 metric tons per year)	Average Annual Capacity per Kiln
Wet Process (cont.)			
46–50	16	3,913	244.6
51–55	2	309	154.5
56–60	0		
60+	4	570	142.5
Total	52	15,574	299.5

Source: Portland Cement Association (PCA). 2004. U.S. and Canadian Portland Cement Industry: Plant Information Summary. Skokie, IL: PCA's Economic Research Department.

All 68 kilns that have become operational within the past 30 years use the dry manufacturing process. These new kilns account for 64% (60 million metric tons) of national clinker capacity.

2.3.2.3 Production Capacity and Utilization

Between 2000 and 2005, apparent annual clinker capacity grew approximately 17%, while clinker production grew by approximately 14% (Table 2-8). Because capacity tends to grow more rapidly than production, total capacity utilization decreased slightly in this period from 87.5% in 2000 to 85.4% in 2005.

Table 2-8. Clinker Capacity, Production, and Capacity Utilization in the United States: 2000 to 2005

	2000	2001	2002	2003	2004	2005
Apparent annual capacity (10^3 metric tons)	89,264	100,360	101,000	102,000	105,000	104,000
Production (10^3 metric tons)	78,138	79,979	82,959	83,315	88,190	88,783
Capacity utilization (%)	87.5%	79.7%	82.1%	81.7%	84.0%	85.4%

Source: U.S. Department of the Interior, U.S. Geological Survey. 2000–2005. *Minerals Yearbook, Cement*. Table 5. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/>. As obtained on March 14, 2008.

Much of the vast majority of the growth in clinker capacity came in 2001 when existing Portland cement plants completed major capacity upgrade projects, resulting in a 12% increase in clinker capacity over the previous year (USGS, 2002). As a result, capacity utilization fell to 79.7% that year. After 2001, clinker capacity grew an average of 1% each year, while production grew an average of 2%. As a result, capacity utilization has risen slowly since 2001. However,

throughout these movements in clinker capacity and production, capacity utilization tended to remain between 80% and 85%.

Capacity utilization often varies by geographic region as a result of fluctuations in regional construction activity. For example, 2005 data show that Idaho, Montana, and Nevada shared a capacity utilization rate of 95.5%—well above the national average. In contrast, South Carolina used only 64.5% of its clinker capacity. Table 2-9 provides a complete listing of capacity utilization rates by state in 2005.

2.3.2.4 Employment

Each year, the Annual Survey of Manufactures (ASM) collects employment, payroll, sales, and other data for all manufacturing establishments. Table 2-10 summarizes the employment data collected by the ASM for the cement manufacturing industry (NAICS 327310) from 2000 to 2005. As these data indicate, total employment fell approximately 2% over this 6-year period, from approximately 17,000 employees in 2000 to 16,900 in 2005.

2.3.2.5 Trends

As previously discussed, clinker capacity has been increasing at a slower pace since 2001. However, according to the PCA, the cement industry has announced that it will increase clinker capacity by nearly 25 million metric tons between 2007 and 2012. This represents a 27% increase over U.S. 2006 clinker capacity and amounts to a $5.9 billion investment (PCA, 2007).

In addition to these expected capacity expansions, likely changes in U.S. specifications allowing the use of limestone in Portland cement could also increase production capacity. According to the PCA, domestic cement supply could increase by as much as 2 million additional tons by 2012. Increases in EPA production variances could also add another 1.1 million metric tons of domestic supply (PCA, 2007).

2.3.3 Firm Characteristics

EPA has reviewed industry information and publicly available sales and employment databases to identify the chain of ownership by accounting for subsidiaries, divisions, and joint ventures to appropriately group companies by size. Table 2-11 provides sales and employment data for 27 ultimate parent companies operating Portland cement manufacturing plants in 2005.

Table 2-9. Capacity Utilization Rates by State: 2005

State	USGS Geographic Area	Utilization Rate (percent)
AL	Alabama	86.7
AR	Arkansas and Oklahoma	90.9
AZ	Arizona and New Mexico	87
CA	California, northern and southern	88.8
CO	Colorado and Wyoming	79.5
FL	Florida	85.9
GA	Georgia, Virginia, West Virginia	78.4
IA	Iowa, Nebraska, South Dakota	85.5
ID	Idaho, Montana, Nevada, Utah	95.5
IL	Illinois	91.4
IN	Indiana	86.8
KS	Kansas	89.1
KY	Kentucky, Mississippi, Tennessee	87.4
MD	Maryland	89.1
ME	Maine and New York	83.6
MI	Michigan	85.5
MO	Missouri	90.3
MS	Kentucky, Mississippi, Tennessee	87.4
MT	Idaho, Montana, Nevada, Utah	95.5
NE	Iowa, Nebraska, South Dakota	85.5
NM	Arizona and New Mexico	87
NV	Idaho, Montana, Nevada, Utah	95.5
NY	Maine and New York	83.6
OH	Ohio	84.7
OK	Arkansas and Oklahoma	90.9
OR	Oregon and Washington	83.3
PA	Pennsylvania, eastern and western	83.7
SC	South Carolina	64.5

Source: U.S. Department of the Interior, U.S. Geological Survey. 2007b. *2005 Minerals Yearbook, Cement*. Table 5. Washington, DC: U.S. Department of the Interior.

Table 2-10. Cement Manufacturing Employment (NAICS 327310): 2000 to 2005

Year	Number of Employees
2000	17,175
2001	17,220
2002	17,660
2003	17,352
2004	16,883
2005	16,877

Sources: U.S. Department of Commerce, Bureau of the Census. 2006. *2005 Annual Survey of Manufacture*s. M05(AS)-1. Washington, DC: Government Printing Office. Available at <http://www.census.gov/prod/2003pubs/m01as-1.pdf>. As obtained on March 14, 2008.

U.S. Department of Commerce, Bureau of the Census. 2003. *2001 Annual Survey of Manufacture*s. M05(AS)-1. Washington, DC: Government Printing Office. Available at <http://www.census.gov/prod/2003pubs/m01as-1.pdf>. As obtained on March 14, 2008.

2.3.3.1 Distribution of Small and Large Companies

Firms are grouped into small and large categories using Small Business Administration (SBA) general size standard definitions for NAICS codes. These size standards are presented either by number of employees or by annual receipt levels, depending on the NAICS code. The manufacture of Portland cement is covered by NAICS code 327310 for cement manufacturing. Thus, according to SBA size standards, firms owning Portland cement manufacturing plants are categorized as small if the total number of employees at the firm is less than 750; otherwise, the firm is classified as large. As shown in Table 2-11, potentially affected firms range in size from 160 to 71,000 employees. A total of 4 firms, or 15%, are categorized as small, while the remaining 23 firms, or 75%, are large.[1]

2.3.3.2 Capacity Share

As shown in Table 2-11, the leading companies in terms of capacity at the end of 2005 were Holcim (U.S.) Inc.; CEMEX, Inc.; Lafarge North America, Inc.; Buzzi Unicem USA, Inc.; HeidelbergCement AG (owner of Lehigh Cement Co.); Ash Grove Cement Co.; Texas Industries, Inc.; Italcementi S.p.A.; Taiheiyo Cement Corporation; Titan Cement; and VICAT. The top 5 had about 57% of total U.S. clinker capacity, and the top 10 accounted for 83% of total capacity. Small companies accounted for less than 5% of clinker capacity.

[1] In cases where no employment data were available, we used information from previous EPA analyses to determine firm size.

Table 2-11. Ultimate Parent Company Summary Data: 2005

Ultimate Parent Name	Annual Sales (10^6)	Employ-ment	Type	Small Business	Plants	Kilns	Clinker Capacity (10^3 metric tons per year)	Capacity Share
Holcim, Inc	$14,034	59,901	Public	No	14	17	13,089	14.0%
CEMEX, S.A. de C.V.	$18,290	26,679	Public	No	13	21	12,447	13.3%
Lafarge S.A.	$22,325	71,000	Public	No	13	23	12,281	13.1%
BUZZI UNICEM SpA	$3,495	11,815	Private	No	10	19	8,129	8.7%
HeidelbergCement AG	$12,182	45,958	Public	No	10	13	7,786	8.3%
Ash Grove Cement Company	$1,190	2,600	Private	No	9	15	6,687	7.1%
Texas Industries, Inc.	$944	2,680	Public	No	4	15	5,075	5.4%
Italcementi S.p.A.	$5,921	20,313	Public	No	6	16	4,442	4.7%
Taiheiyo Cement Corporation	$7,710	2,061	Private	No	3	7	3,375	3.6%
Titan Cement	$1,589	1,834	Public	No	2	2	2,612	2.8%
VICAT	$2,137	6,015	Public	No	2	2	1,933	2.1%
Eagle Materials	$922	1,600	Public	No	3	5	1,651	1.8%
Mitsubishi Cement Corporation	$1,134	NA	Joint venture	No	1	1	1,543	1.6%
Rinker Materials	$4,140	11,193	Private	No	2	2	1,533	1.6%
Hanson America Holdings	$3,000	14,872	Private	No	1	1	1,497	1.6%
Salt River Materials Group [a]	$150[b]	<750	Tribal Govern ment	Yes	1	4	1,477	1.6%
Grupo Cementos de Chihuahua, S.A. de C.V.	$663	2,591	Public	No	2	5	1,283	1.4%
Cementos Portland Valderrivas, S.A.	$1,159	2,674	Public	No	2	6	1,257	1.3%
Zachary Construction	$152	1,200	Private	No	1	2	868	0.9%
RMC Pacific Materials	$160	800	Private	No	1	1	812	0.9%

(continued)

Table 2-11. Ultimate Parent Company Summary Data: 2005 (continued)

Ultimate Parent Name	Annual Sales (10^6)	Employ-ment	Type	Small Business	Plants	Kilns	Clinker Capacity (10^3 metric tons per year)	Capacity Share
Monarch Cement Company	$154	600	Public	Yes	1	2	787	0.8%
Florida Rock Industries	$1,368	3,464	Public	No	1	1	726	0.8%
Votorantim Group and Anderson Columbia Company	$9,518	30,572	Joint venture	No	1	1	682	0.7%
Dyckerhoff AG	$1,876	6,958	Public	No	1	1	586	0.6%
Continental Cement Company, LLC	$50[b]	<750	Private	Yes	1	1	549	0.6%
Cementos Del Norte	NA	NA	Private	No	1	1	392	0.4%
Snyder Associate Companies	$29	350	Private	Yes	1	2	286	0.3%

[a] Enterprise is owned by Salt River Pima-Maricopa Indian Community.

[b] EPA estimate.

Sources: Dun & Bradstreet, Inc. 2007. D&B million dollar directory. Bethlehem, PA.
LexisNexis. LexisNexis Academic [electronic resource]. Dayton, OH: LexisNexis.

2.3.3.3 Company Revenue and Ownership Type

Cement manufacturing is a capital-intensive industry. The vast majority of stakeholders are large global companies with sales exceeding $1 billion. In 2005, ultimate parent company sales ranged from $30 million to $22.3 billion (Table 2-11), with average (median) sales of $4,565 ($1,589) million. Small companies accounted for 0.3% share by sales. Ultimate parent companies were either privately or publicly owned or jointly operated by several companies. A majority of the companies (52%) were publicly owned. Private companies had a slightly smaller share (41%), and only two (or 7%) were joint ventures.

2.4 Markets

Portland cement is produced and consumed domestically as well as traded internationally. The United States meets a substantial fraction of its cement needs through imports; in contrast, it exports only a small fraction of domestically produced cement to other countries. We provide value, quantity, and price trends over the past decade for Portland cement when detailed statistics

are available. In the case of international trade, we can report data only for hydraulic cement, which includes Portland and masonry cement.

2.4.1 Market Volumes

2.4.1.1 Domestic Production

In 2007, the domestic shipments of Portland cement were 90.6 million metric tons, reflecting an 8.5% increase from 2000 and, more recently, a 3% decrease from 2006 (see Table 2-12). Year-end stocks remained relatively level during this period at 7.4 million metric tons. Stocks fell slightly by 5% since 2006 and equaled 8.9 million tons in 2007. As Table 2-12 shows, shipments to customers increased steadily since 2000, reaching 128 million tons in 2006. However, affected by declines in the housing market, the shipments fell by 9% in 2007.

Table 2-12. Historical U.S. Cement Statistics (10^6 metric tons)

	2000	2001	2002	2003	2004	2005	2006	2007
Production								
Clinker	78.1	78.5	82.0	81.9	86.7	87.4	88.6	87.2
Portland cement	83.5	84.5	85.3	88.1	92.4	93.9	93.2	90.6
Masonry cement	4.3	4.5	4.4	4.7	5.0	5.4	5.0	4.9
Total cement	87.8	88.9	89.7	92.8	97.4	99.3	98.2	95.5
Shipments to customers	110.0	113.1	110.0	112.9	120.7	127.4	127.9	116.0
Stocks, cement, year end	7.6	6.6	7.6	6.6	6.7	7.4	9.4	8.9

Sources: U.S. Department of the Interior, U.S. Geological Survey. 2008b. *Minerals Commodity Summaries, Cement 2008*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2008-cemen.pdf>.

U.S. Department of the Interior, U.S. Geological Survey. 2003. *2002 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

2.4.1.2 International Trade

Cement imports are a significant share of domestic consumption (approximately 20%); they also grew by 30% from 2000 to 2006 (see Table 2-13). Major importing countries in 2007 included Canada (18% of total imports in 2006), China (16%), and Thailand (11%) (DOI, USGS, 2008b). In 2007, the falling value of the dollar and construction activity declines in the housing market tempered the quantity of import demanded. As a result, the share of U.S. consumption met by imports fell to its lowest level in 10 years.

Table 2-13. U.S. Cement Trade Data: 2000 to 2007

	2000	2001	2002	2003	2004	2005	2006	2007
Exports (10^6 metric tons)	0.7	0.7	0.9	0.8	0.7	0.8	1.5	1.9
Imports (10^6 metric tons)	24.6	23.6	22.5	21.0	25.4	30.4	32.1	21.3
Net import share of apparent consumption (%)	20.0	21.0	19.0	20.0	21.0	23.0	23.0	17.0

Sources: U.S. Department of the Interior, U.S. Geological Survey. 2008b. *Minerals Commodity Summaries, Cement 2008*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2008-cemen.pdf>.

U.S. Department of the Interior, U.S. Geological Survey. 2003. *2002 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/>.

During the period from 2000 to 2005, U.S. exports remained relatively constant at about 800,000 tons and typically did not exceed 1% of production. However, the level of U.S. exports has increased during the last 2 years. In 2007, U.S. exports totaled 1.9 million metric tons. The vast majority of U.S. exports of hydraulic cement are supplied to Canada: U.S. producers shipped a total of 650,000 tons to Canada in 2005, or 85% of total U.S. exports. The remaining fraction of U.S. exports in 2005 went to the Bahamas, Mexico, and 33 other countries around the world (DOI, USGS, 2008b).

2.4.2 Market Prices

Correcting for the effects of inflation, we find that the real price of cement per metric ton (2005 dollars) has typically ranged between $75 and $95 since 1990 (see Figure 2-7). However, data for the last 2 years suggest the average price of cement is at its highest level in over 2 decades (approximately $100). Because of transportation constraints, there are regional differences in the price of cement across states. For example, remote locations such as Alaska and Hawaii had the highest deviation from the national average ($48 in 2005) (see Figure 2-8). In the contiguous states, prices in Arizona, New Mexico, and California were higher than the national averages, while prices in Texas, Indiana, and South Carolina were among the lowest.

2.4.3 Future Projections

Although estimates of future cement demand are not publicly available, the Energy Information Administration provides projections for the real value of shipments for the stone, clay, and glass industry in its *AEO* (DOE, 2007). The forecasted annual average growth rate for 2005 to 2030 is approximately 1.7%.

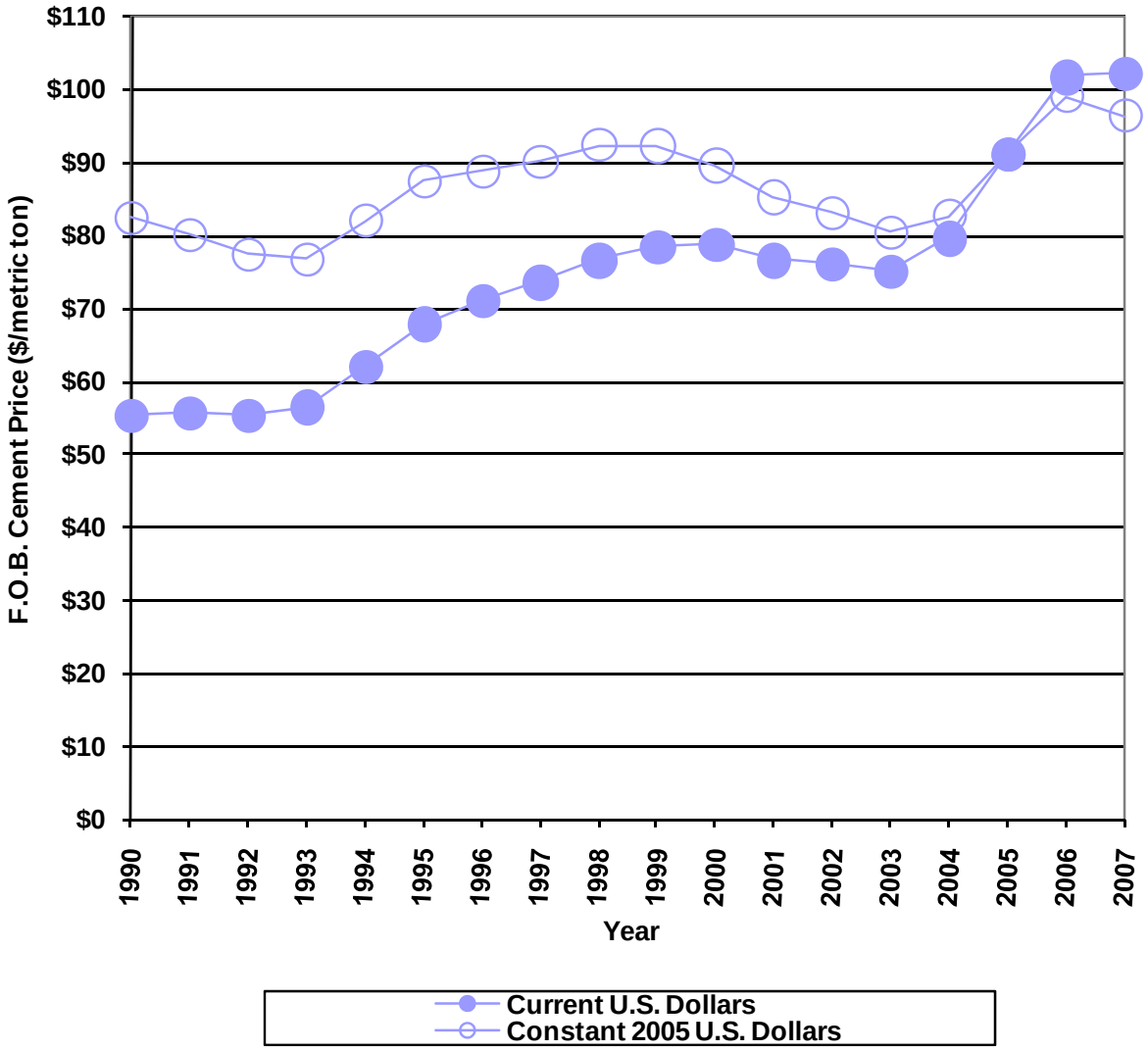

Figure 2-7. Historical U.S. Cement Price

Sources: 1990–2003: Kelly, T. and G. Matos. 2007b. "Historical Statistics for Mineral and Material Commodities in the United States: Cement Supply and Demand Statistics." U.S. Geological Survey Data Series 140, Version 1.2. Available at <http://minerals.usgs.gov/ds/2005/140/>. Last modified April 11, 2006.

2004–2007: U.S. Department of the Interior, U.S. Geological Survey. 2008b. *Minerals Commodity Summaries, Cement 2008*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2008-cemen.pdf>.

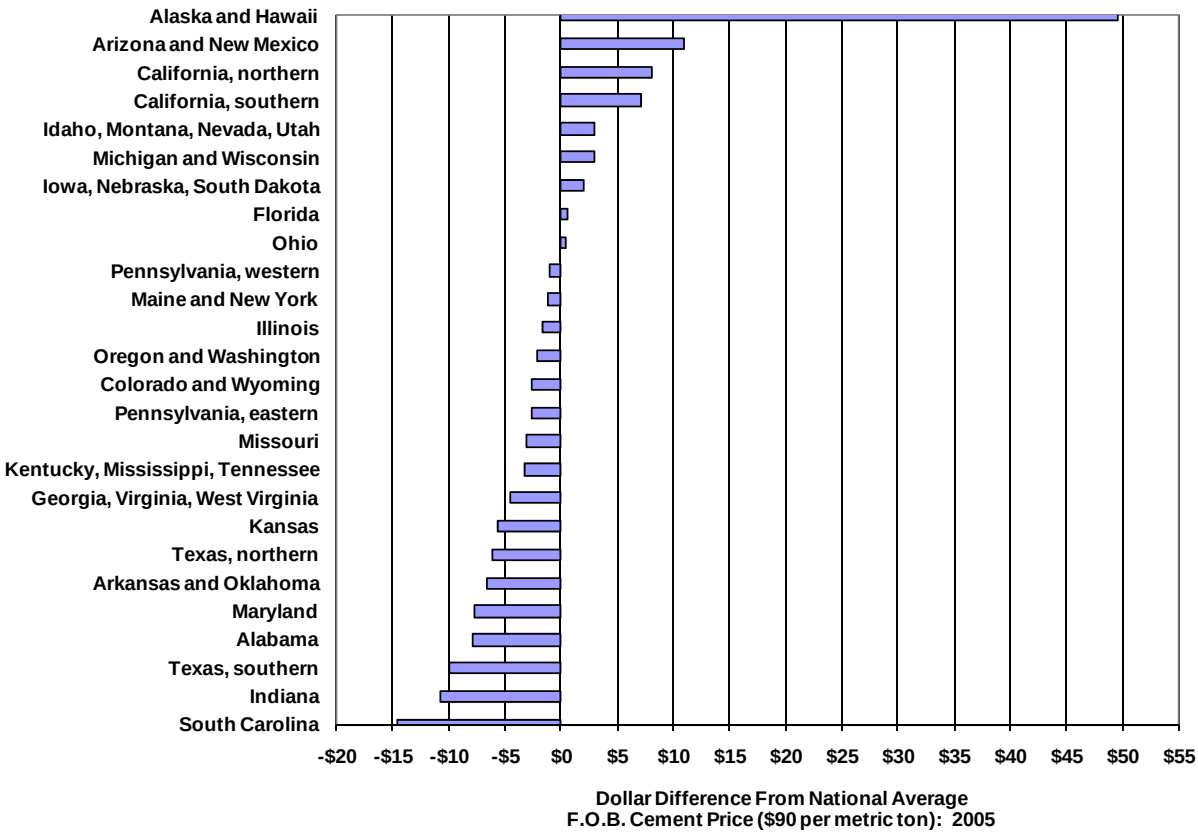

Figure 2-8. Deviation from National Average Cement Price per Metric Ton by Region: 2005

Source: U.S. Department of the Interior, U.S. Geological Survey. 2007a. *2005 Minerals Yearbook, Cement.* Washington, DC: U.S. Department of the Interior. Table 11. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

SECTION 3
ECONOMIC IMPACT ANALYSIS

EPA prepares an EIA to provide decision makers with a measure of the social costs of using resources to comply with a program (EPA, 2000). The social costs can then be compared with estimated social benefits (as presented in Section 5). As noted in EPA's (2000) *Guidelines for Preparing Economic Analyses*, several tools are available to estimate social costs and range from simple direct compliance cost methods to the development of a more complex market analysis that estimates market changes (e.g., price and consumption) and economic welfare changes (e.g., changes in consumer and producer surplus).

The Office of Air Quality Planning and Standards (OAQPS) has adopted the standard industry-level analysis described in the Office's resource manual (EPA, 1999a). This approach is consistent with previous EPA analyses of the Portland cement industry (EPA, 1998; EPA, 1999b, and 2009a) and uses a single-period static partial-equilibrium model to compare pre-policy cement market baselines with expected post-policy outcomes in these markets. The benchmark time horizon for the analysis is the intermediate run where producers have some constraints on their flexibility to adjust factors of production. This time horizon allows us to capture important transitory impacts of the program on existing producers. Key measures in this analysis include

- market-level effects (market prices, changes in domestic production and consumption, and international trade),
- industry-level effects (changes in (i.e. operating profits) and employment),
- facility-level effects (plant utilization changes), and
- social costs (changes in producer and consumer surplus).

Absent forecasts and the uncertainties of future economic baselines, the partial-equilibrium market analysis can only cover a subset of plants presumed to be operating in conditions similar to 2005. Thus, this analysis does not reflect changes in the state of the US economy which may occur by the analysis year of 2013 which could significantly influence the quantity of cement needed. As shown in the following sections, the market analysis covers $378 million of the total $466 million in regulatory program costs, or 81%; simulated post policy outcomes described throughout Section 3.2 should be interpreted in light of this modeling choice. EPA analyzed the remaining $88 million in NESHAP and NSPS regulatory program costs "outside" of the partial equilibrium market analyses using direct compliance costs methods (see Section 3.3). EPA provides complete social cost accounting in the section describing the

social cost estimates (Section 3.4) and provides a discussion of its overall assessment (Section 3.5).

3.1 Regulatory Program Costs

EPA is finalizing amendments to the NESHAP from the Portland cement manufacturing industry and (NSPS for Portland cement plants. The final amendments to the NESHAP add or revise, as applicable, emission limits for Hg, THC, and PM from kilns located at a major or an area sources, and HCl from kilns and located at major sources. EPA is also adopting separate standards for these pollutants which apply during startup, shutdown, and operating modes. Finally, EPA is adopting performance specifications for use of mercury CEMS and updating recordkeeping and testing requirements. The final amendments to the NSPS add or revise, as applicable, emission limits for particulate matter (PM), opacity, nitrogen oxides (NO_x), and sulfur dioxide (SO_2) for facilities that commence construction, modification, or reconstruction after June 16, 2008. The final rule also includes additional testing and monitoring requirements for affected sources. Although EPA's analysis focuses on the final NESHAP and NSPS engineering cost estimates, EPA also presents additional information on different combinations of the regulatory programs. This information helps stakeholders better understand the size and scope of the each. These include

- final NSPS only,
- final NESHAP only, and
- alternative: more stringent NSPS and final NESHAP.

For the year 2013, EPA's engineering cost analysis estimates the total annualized costs of the final NESHAP and NSPS are $466 million (in 2005 dollars) (see Table 3-1). These costs include a variety of pollution control expenditures: equipment installation, operating and maintenance, recordkeeping, and performance-testing activities. Capital costs are annualized at a discount rate of 7% over the expected life of the control equipment which is 20 years for all devices except RTOs which are 15 years. The majority of the costs ($455 million, or 98%, are associated with the final NESHAP. The remaining costs ($11 million) are associated with the final NSPS limits for SO_2 and NO_x. Figure 3-1 illustrates the distribution of annualized compliance costs per metric ton of clinker capacity by different combinations of the regulatory programs. In Table 3-2, we report state-level summary statistics for total annualized compliance costs per metric ton of clinker capacity for the final NESHAP and NSPS to highlight any regional differences in control costs.

Table 3-1. Summary of Direct Total Annualized Compliance Costs (million, 2005$)

Description	Total Annualized Compliance Costs	
	Final NSPS Only	**More Stringent NSPS Only**
Total:	$40[a]	$56[a]
	Final NESHAP Only	
Partial Equilibrium Analysis (136 Kilns)	$378	
NSPS kilns (7 kilns)	$29	
Other kilns (13 kilns)	$48	
Total:	$455	
	Final NESHAP and NSPS	**Final NESHAP and More Stringent NSPS**
136 Kilns	$378	$378
20 Kilns	$88	$104
Total:	$466	$482

[a] The final NSPS only also includes the $29 million in NESHAP costs for 7 kilns. The 7 kilns will also incur an additional $11 in compliance costs to meet the final NSPS limits for SO_2 and NO_x. Alternatively, the 7 kilns would also incur an additional $27 in compliance costs to meet the stringent NSPS limits for SO_2 and NO_x.

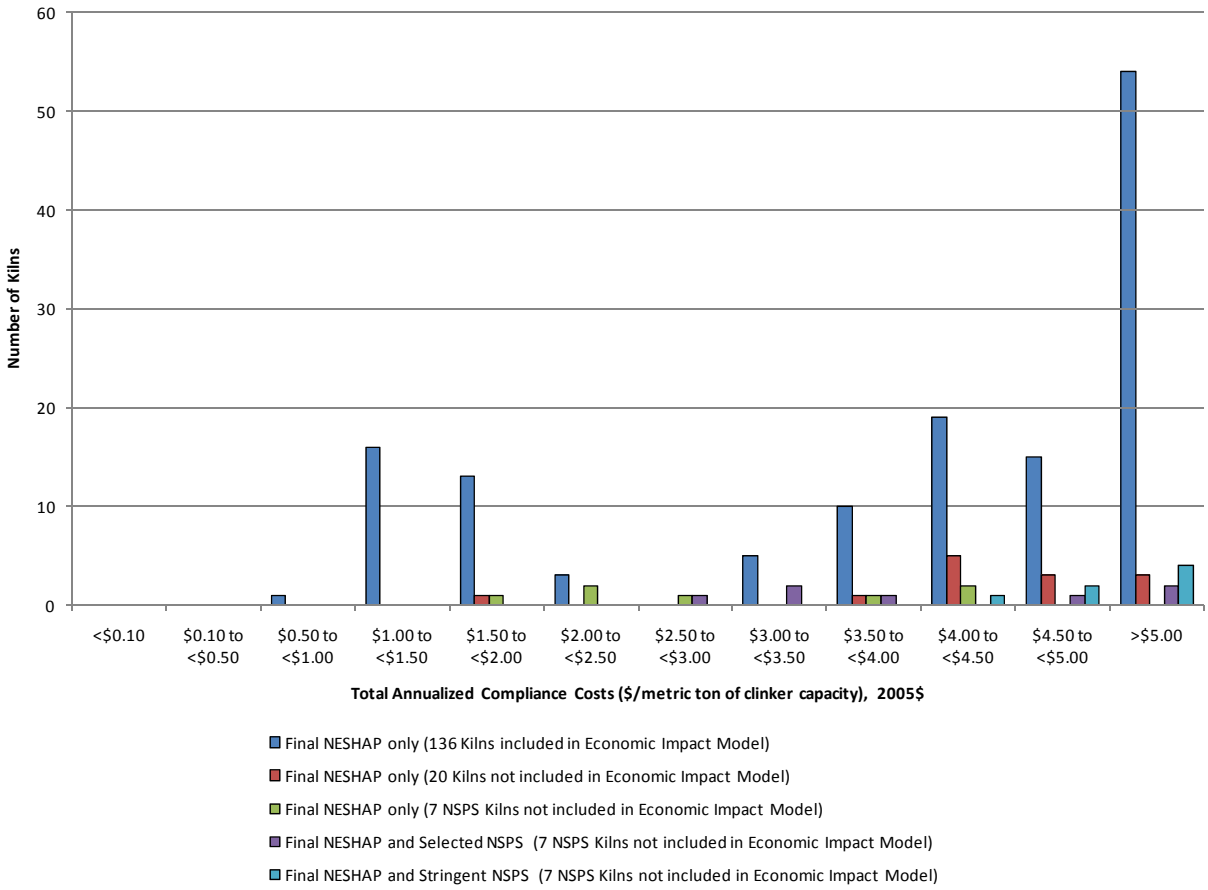

Figure 3-1. Range of Per-Ton Total Annualized Compliance Costs (2005$)

Table 3-2. Range of Per-ton Total Annualized Compliance Costs by State (2005$)

	Data		
ST	Average ($/ ton of clinker capacity)	Minimum ($/ ton of clinker capacity)	Maximum ($/ ton of clinker capacity)
AL	$3	$1	$5
AZ	$3	$1	$6
CA	$4	$3	$5
CO	$2	$1	$3
FL	$3	$1	$5
GA	$1	$1	$1
IA	$6	$4	$8
ID	$10	$9	$10
IL	$6	$1	$8
IN	$9	$5	$14
KS	$6	$6	$6
KY	$4	$4	$4
MD	$6	$3	$9
ME	$1	$1	$1
MI	$5	$4	$6
MO	$5	$4	$5
MT	$2	$2	$2
NE	$6	$5	$6
NM	$2	$2	$2
NV	$2	$2	$2
NY	$3	$1	$4
OH	$5	$5	$5
OK	$8	$4	$13
OR	$4	$4	$4
PA	$5	$2	$7
SC	$4	$4	$4
SD	$2	$1	$2
TN	$3	$1	$5
TX	$5	$1	$8
UT	$5	$1	$9
VA	$4	$4	$4
WA	$1	$1	$2
WV	$7	$6	$8
WY	$7	$5	$8
U.S.	$5	$1	$14

Note: Includes Final NESHAP only for 136 kilns included in economic impact model.

3.2 Partial-Equilibrium Analysis

Field Co

The partial-equilibrium analysis develops a cement market model that simulates how stakeholders (consumers and firms) might respond to the additional regulatory program costs. In this section, we provide an overview of the economic model used during proposal (EPA, 2009). Appendix A provides additional details about economic model updates made since proposal, model equations, and parameters.

3.2.1 Regional Structure and Baseline Data

Cement sales are often concentrated locally among a small number of firms for two reasons: high transportation costs and production economies of scale.[1] Transportation costs significantly influence where cement is ultimately sold; high transportation costs relative to unit value provide incentives to produce and sell cement locally in regional markets (USITC, 2006). To support this claim, the empirical literature has typically pointed to Census of Transportation data showing over 80% of cement shipments were made within a 200-mile radius (Jans and Rosenbaum, 1997)[2] and reported evidence of high transportation costs per dollar of product value from case studies (Ryan, 2006). Based on this literature, the Agency assumes that the U.S. Portland cement industry is divided into a number of independent regional markets with each having a single market-clearing price.

The freight-on-board (f.o.b.) price of Portland cement for each regional market is derived as the production weighted average of the state level f.o.b. prices reported by the USGS for cement (see Table 3-3). The production of Portland cement within each market is the sum of estimated individual kiln production levels (EPA, 2009) and include adjustments described in Appendix A (see Table 3-4). We obtained estimates of Portland cement imports from the USGS and mapped them to each market based on the port of entry.

3.2.2 Near-Term Cement Plant Production Decisions

A cement company acts in the best interest of its shareholders and maximizes profits. When deciding whether to make another ton of cement, the company considers the *production effect* on profits by comparing the current market price of cement and the marginal production cost; if price is above marginal production cost, producing and selling the extra ton of cement increase profit. The company continues to produce additional cement until the profit from

[1] The 2002 Economic Census reports that the national Herfindahl-Hirschman Index (HHI) for cement—North American Industry Classification System (NAICS) 32731—is 568. However, this measure is likely not representative of actual concentration that exists in regional markets.

[2] A recent USITC study of California cement markets found more than 75% of gray Portland cement shipments in the state were shipped to customers within 200 miles of the cement producer (USITC, 2006).

Table 3-3. Portland Cement Prices by Market ($/metric tons): 2005

Market	Price ($/metric ton)
Atlanta	$81
Baltimore/Philadelphia	$82
Birmingham	$83
Chicago	$67
Cincinnati	$84
Dallas	$75
Denver	$89
Detroit	$93
Florida	$91
Kansas City	$86
Los Angeles	$78
Minneapolis	$92
New York/Boston	$89
Phoenix	$83
Pittsburgh	$88
St. Louis	$84
Salt Lake City	$91
San Antonio	$82
San Francisco	$97
Seattle	$88

producing an extra ton of cement is zero (price equals marginal cost) or capacity constraints are reached. The decision rule is consistent with the assumption of pure competition.

Although perfect competition is widely accepted for modeling many industries regardless of the model time horizon (EPA, 2000), the cement industry has two characteristics that influenced EPA's modeling choice relating to market structure. First, high transportation costs and other production economics tend to limit the number of sellers (particularly over a short time horizon), so each seller has a substantial regional market share. Timely market entry is also constrained by the high capital costs that involve purchases and construction of large rotary kilns that are not readily movable or transferable to other uses.[3] Second, cement producers offer similar or identical products. American Society for Testing and Materials (ASTM) specifications tend to ensure uniform quality, and recent industry reviews (USITC, 2006) suggest that there is little or no brand loyalty that allows firms to differentiate their products.

[3] In addition, large plants are typically more economical because they can produce cement at lower unit costs; this reduces entry incentives for smaller capacity cement plants.

Table 3-4. Portland Cement Markets (10^6 metric tons): 2005

Market	U.S. Production	Imports	Total
Atlanta	5.8	2.3	8.1
Baltimore/Philadelphia	7.8	0.6	8.5
Birmingham	5.9	2.2	8.1
Chicago	4.7	0.2	4.9
Cincinnati	3.7	0.0	3.7
Dallas	8.1	2.4	10.5
Denver	3.4	0.0	3.4
Detroit	3.8	1.3	5.2
Florida	5.5	5.8	11.4
Kansas City	5.0	0.0	5.0
Los Angeles	10.6	3.8	14.4
Minneapolis	1.7	0.4	2.1
New York/Boston	3.2	2.8	6.0
Phoenix	4.3	0.0	4.3
Pittsburgh	1.5	1.6	3.1
St. Louis	6.0	0.0	6.0
Salt Lake City	2.4	0.1	2.4
San Antonio	5.5	4.6	10.0
San Francisco	3.4	2.8	6.2
Seattle	1.1	2.5	3.6

Given entry barriers, product characteristics, and the need to understand important near-term/transitory stakeholder outcomes, EPA continued to use the economic impact model designed for previous analyses (EPA, 1998, 1999b, 2009). The model considers how regional markets may operate in near-term time horizons when 1) the number of companies is limited and 2) the companies sell similar or identical products.[4] Under these circumstances, the short-run production decision rule that a cement company makes differs from pure competition. The company continues to consider the *production effect* described above; however, the company adds another dimension to the decision-making process by also considering the *market price effect* that is associated with producing an additional ton of cement. Given the small number of cement producers, adding an extra ton of cement to the regional market may *lower* the market

[4] This economic model is formally known as a multi-firm Cournot oligopoly model.

cement price and reduce the profits on all the other cement sold. If the price effect is large enough, companies may find it more profitable to reduce production below the levels implied by pure competition. As a result, short-run regional market prices tend to be higher than marginal production costs (i.e., there may be a preexisting market distortion within cement markets *prior* to regulation).[5] The size of the existing distortion depends on the seller's market share and how responsive cement consumers are to changes in the cement price. Economic theory suggests the market distortion will typically be higher the smaller the number of sellers and when the quantity demanded is less sensitive to price (i.e., the demand elasticity is inelastic) (see Appendix A).

3.2.3 Economic Impact Model Results

3.2.3.1 Market-Level Results

Market-level impacts include the regional price and quantity adjustments for Portland cement, including the changes in imports for the appropriate regions. As shown in Table 3-5, the average national price for Portland cement increases by 5%, or $4.50 per metric ton, while overall U.S. cement consumption falls by approximately 5%. Domestic production falls by 11%, or 10 million tons per year. Cement imports increase in response to higher domestic cement prices; imports increase by 10%, or 3 million metric tons.

Table 3-5. National-Level Market Impacts: 2005

		Changes from Baseline	
	Baseline	**Absolute**	**Percent**
Market Price ($/metric ton)	$83.70	$4.50	5.4%
Market Output (million metric tons)	126	−6	−4.8%
Domestic production	93	−10	−10.8%
Imports	33	3	10.0%

As shown in Table 3-6, price increases are the highest in regions with high compliance costs per metric ton. For example, the Cincinnati market price increase ($10 per metric ton) also includes kilns with higher average compliance costs and a kiln with the highest per-unit

[5] This ultimately influences the partial-equilibrium model's estimates of the social cost of the regulatory program since bigger existing market distortions tend to widen the gap between price and marginal cost in these markets and lead to higher deadweight loss estimates than under the case of perfectly competitive markets. The Office of Management and Budget (OMB) explicitly mentions the need to consider market power–related welfare costs in evaluating regulations under Executive Order 12866 (EPA, 1999a).

Table 3-6. Regional Compliance Costs and Market Price Changes ($/metric ton of cement): 2005

Market	Incremental Compliance Costs ($/metric ton of estimated cement production)			Baseline Price	Market Price Change	
	Mean	Minimum	Maximum		Absolute	Percent
Atlanta	$3.60	$1.10	$5.90	$81.30	$2.80	3.4%
Baltimore/Philadelphia	$6.20	$1.20	$10.00	$81.70	$6.10	7.5%
Birmingham	$3.60	$1.10	$4.80	$82.60	$3.80	4.6%
Chicago	$6.80	$0.90	$10.10	$66.90	$4.80	7.2%
Cincinnati	$8.10	$4.00	$14.10	$84.20	$10.40	12.4%
Dallas	$5.60	$3.50	$8.50	$75.10	$4.90	6.5%
Denver	$3.00	$1.00	$8.10	$88.70	$6.30	7.1%
Detroit	$6.50	$4.00	$10.30	$92.70	$4.20	4.5%
Florida	$3.40	$1.20	$5.50	$90.70	$3.50	3.9%
Kansas City	$8.60	$3.80	$13.80	$86.10	$8.20	9.5%
Los Angeles	$6.00	$3.20	$13.10	$78.20	$4.30	5.5%
Minneapolis	$6.30	$4.50	$8.80	$92.20	$8.50	9.2%
New York/Boston	$2.50	$1.00	$4.50	$89.00	$1.80	2.0%
Phoenix	$1.90	$1.00	$6.00	$83.10	$4.20	5.1%
Pittsburgh	$7.60	$6.90	$8.00	$88.00	$4.60	5.2%
St. Louis	$4.80	$3.80	$5.60	$84.10	$4.50	5.4%
Salt Lake City	$5.90	$1.60	$9.90	$91.40	$10.40	11.4%
San Antonio	$4.00	$0.80	$7.70	$82.30	$3.30	4.0%
San Francisco	$3.10	$1.00	$5.00	$96.90	$3.30	3.4%
Seattle	$1.20	$1.00	$1.40	$88.00	$0.70	0.8%
Grand Total	$5.20	$0.80	$14.10	$83.90	$4.50	5.4%

compliance costs ($14 per metric ton).[6] It is important to note that EPA uses a time horizon where transportation costs between regions are high enough that interregional trade is unlikely to occur, at least in the short run. The regional differences in unit compliance costs and the

[6] The per-unit compliance costs were calculated by dividing the total annualized cost per kiln by the kiln's estimated cement production within the economic impact model.

significant simulated changes in relative regional prices suggest domestic cement plants may be more likely to consider short-run shipments of cement between regional markets. Choices would depend on the additional benefits of selling cement to these markets and the costs of transporting the cement outside the regional market. Although EPA has not quantified this effect, additional flexibility would tend to temper prices increases in some of these markets.

Imports also tend to limit price increases in certain regions. This tends to reinforce U.S. production declines because cement plants have more difficulty passing on compliance costs in the form of higher prices when compared with similar plants operating in regions without import competition. Because imports are only modeled for markets with imports in the baseline without regulation, Table 3-7 separates the results into markets with and without imports as well as providing the results for all markets. As shown in Table 3-7, median price increases in regions with imports are lower than the median price increases in regions without import competition. In some regions with imports, the reductions in U.S. production are significant. As shown in Table 3-7, the maximum simulated U.S. regional production change is 23%. To the extent there are any unobserved constraints on import supply that are not captured in the import supply elasticity parameter, price and U.S. production adjustments for regional markets with imports would tend to become more similar to regional markets without imports.

3.2.3.2 Industry-Level Results

As shown in Table 3-8, compliance costs vary by cement plant, and this variation suggests some plants will be more adversely affected than others. To assess these differences, EPA collected industry operating profit data and identified plants with operating profit increases and losses. Absent plant-specific data, EPA assumed each plant's baseline profits were consistent with the median operating profit margin reported by the PCA (2008c, Table 44). In 2005, this value was $18 per metric ton, or 16%. Using this assumption, total operating profits for 59 plants (58%) decrease by $387 million with regulation. These plants tend to have higher per ton compliance costs. The remaining plants' compliance burden is offset by higher regional cement prices, and total plant operating profits increase by $147 million. These 44 plants have lower unit compliance costs compared with their competitors.

Table 3-7. Summary of Regional Market Impacts

	Regional Markets		
	With Imports	**Without Imports**	**All Markets**
Change in Market Price			
Absolute ($/metric ton)			
Mean	$4.70	$6.40	$4.50
Median	$4.20	$5.40	$4.40
Minimum	$0.70	$4.20	$0.70
Maximum	$10.40	$10.40	$10.40
Percentage of baseline price			
Mean	5.5%	7.5%	5.4%
Median	4.9%	6.2%	5.3%
Minimum	0.8%	5.0%	0.8%
Maximum	11.4%	12.4%	12.4%
Change in Domestic Production			
Absolute (thousand metric tons)			
Mean	−559	−271	−501
Median	−421	−247	−372
Minimum	−74	−189	−74
Maximum	−1,539	−403	−1,539
Percentage of baseline production			
Mean	−11.8%	−6.6%	−10.8%
Median	−11.6%	−5.5%	−10.4%
Minimum	−6.8%	−4.4%	−4.4%
Maximum	−22.8%	−10.9%	−22.8%

EPA notes that since conducting this analysis, one high mercury-emitting plant has invested in control technology estimated to reduce emissions by approximately 85 percent. The current analysis does not include these actual costs in the baseline but rather estimates aggregate compliance costs based on the averaging methodologies applied to all other modeled plants. In addition, because this investment occurred after the analysis was conducted, the baseline benefits likewise do not include the approximately 85% emissions reduction. Finally, EPA did not estimate the change in social costs that would occur if the 2 high mercury-emitting plants were to shut down, because the Agency believes these plants will ultimately be able to meet the emissions limit by applying multiple mercury controls, which were accounted for in the cost

analysis. EPA also acknowledges that if these 2 high mercury-emitting plants ultimately are able to meet the emissions limit, they will not likely be able to do so by the required compliance date.

Within the group of plants with operating losses, EPA identified 10 domestic plants with negative operating profits and significant utilization changes that could temporarily idle until market demand conditions improve (see Table 3-9). The plants have unit compliance costs close to $8 per ton; they account for approximately 8% of domestic capacity. These plants are modeled as continuing to operate despite low capacity utilization and short run negative profits. The model results for them are included in the summary results for Tables 3-5, 3-6, 3-7, and 3-8 but are also reported separately in Table 3-9.

If the plant owners did decide to permanently shut down these plants, the reduction in domestic supply would lead to additional projected market price increases. This would lead to an increased production at other plants, a possible increase in imports (depending if the plant that chooses to close is in a market where imports are anticipated) and a decrease in control cost. This scenario cannot be easily modeled. In an effort to bound this effect, the price increase needed to reduce national consumption by the amount of production that would be lost if the ten plants dropped from 55.5% capacity utilization to 0.0% capacity utilization was estimated using the demand elasticity of 0.88. This ignores changes in other plants is response to an increased potential market share and increases in imports. Both of these would tend diminish the price increase. The predicted price change was multiplied by the change in production associated with the ten plants dropping capacity utilization to zero and multiplied by one half to estimate the change in surplus associated with the price and quantity change. This gave a result of a $10 million increase in social cost. This number was then reduced by the avoided pollution control cost of $34 million at the ten plants because if the plants were to idle or shut down, they would not incur compliance costs. This resulted in a net reduction of $24 million in social cost when these firms idle or shut down as compared to the modeled scenario, where firms continue to operate a low capacity but incur compliance costs. Because of the method of estimating this adjustment it cannot be distributed between producer and consumer surplus. An estimate of the social cost is provided with and without this adjustment.

Table 3-8. Distribution of Industry 2005

	Changes in Total Operating Profit:		
	Plants with Loss	**Plants with Gain**	**All Plants**
Number	58	44	102
Cement Capacity (million metric tons)			

Total	55,202	38,145	93,346
Average per plant	952	867	915
Compliance Costs			
Total (thousand)	$308,740	$68,806	$377,546
Average ($/metric cement)	$5.59	$1.80	$4.04
Capacity Utilization (percent)			
Baseline	100.3%	98.7%	99.6%
With regulation	81.0%	100.3%	88.9%
Change in total operating profits (million)	−$387	$147	−$241

Table 3-9. Cement Plants with Significant Utilization Changes 2005

	Total
Number	10
Cement Capacity (thousand metric tons)	
Total	7,815
Average per plant	782
Compliance Costs	
Total (thousand)	$62,222
Average ($/metric ton)	$7.96
Capacity Utilization (%)	
Baseline	99.0%
With regulation	55.5%
Change in Operating Profit (million)	−$116

3.2.3.3 Job Effects

Precise job effect estimates cannot be estimated with certainty and the economic literature does not give clear evidence on the effect of regulation on job effects. Several empirical studies, including Morgenstern et al.,suggest the net employment decline is zero or economically small (e.g., Cole and Elliot, 2007; Berman and Bui, 2001). However, others show the job effects are not trivial (Henderson, 1996; Greenstone, 2002).

EPA has most often estimated employment changes associated with plant closures due to environmental regulation or changes in output for the regulated industry (EPA, 1999a; EPA, 2000). This partial equilibrium approach focuses only on the "demand" portion of the projected change in employment and neglects other employment changes. EPA provides this estimate because it employs the most detailed modeling for the industry being regulated even if it does not capture all types of employment impacts. In addition to the employment effects identified by Morgenstern et al., we also expect that the substitutes for cement (e.g., asphalt) would expand production as consumers shift away from cement to other products. This would also lead to increased employment in those industries. Focusing only on the "demand effect", it can be seen that the estimate from the historical approach is within the range presented by the Morgenstern "demand effect" portion. This strengthens our comfort in the reasonableness of both estimates. In April of this year, EPA started including an estimate based on the Morgenstern approach because it is thought to be a broader measure of the employment impacts of this type of environmental regulation. Thus, this analysis goes beyond what EPA has typically done, and

uses Morgenstern et al. (2002) to provide the basis for the estimates. Morgenstern et al. (2002) model three economic mechanisms by which pollution abatement activities can indirectly influence jobs:

- higher production costs raise market prices, higher prices reduce consumption, and employment within an industry falls ("demand effect");
- pollution abatement activities require additional labor services to produce the same level of output ("cost effect"); and
- postregulation production technologies may be more or less labor intensive (i.e., more/less labor is required per dollar of output) ("factor-shift effect").

This transfer of results from the Morgenstern study is uncertain but avoids ignoring the "cost effect" and the "factor-shift effect" In examinining job effects. EPA selected this paper because the parameter estimates provide a transparent and tractable way to transfer estimates for an employment effects analysis. Simialr estimates were not available from other studies.

- Using the historical approach, we calculated "demand effect" employment changes by assuming that the number of jobs declines proportionally with the economic model's simulated output changes. As shown in Table 3-10, using this limited approach, the employment falls by an 1,500 jobs, or approximately −10%.[7] By comparison, using the Morgenstern approach, we estimate that the net employment effects could range between 600 job losses to 1,300 job gains.

EPA has solely used this historical estimate in the past as a measure of the projected employment change associated with a regulation. However there are a number of serious shortcomings with this approach. First, and foremost, the historical approach only looks at the employment effects on the regulated industry from reduced output. Second, to arrive at that estimate, EPA needed to string together a number of strong assumptions. The employment impacts are independent of the performance of the overall economy. This rule takes effect in three years. If the economy is strong, the demand for cement strong, it is unlikely that any contraction in the industry will take place, even with the regulation. Second, we assume that all plants have the same limited ability to pass on the higher costs. In reality, plants should be modeled as oligopolists for each of their regional markets. Finally, EPA assumed that employment is directly proportional to output. This is unlikely, and biases the results towards higher employment losses. The Morgenstern methodology is a more complete consideration of probable impacts of a regulation on the economy.

[7] To place this reduction in context, it is a similar to the decline experienced during the latest economic downturn; approximately 2,000 jobs (see Appendix A, Table A-3).

Table 3-10. Job Losses/Gains Associated with the Final Rule

Method	1,000 Jobs
Partial equilibrium model (demand effect only)	**−1.5**
Literature-based estimate (net effect [A + B + C below])	**0.3 (−0.6 to +1.3)**
A. Literature-based estimate: Demand effect	−0.8 (−1.7 to +0.1)
B. Literature-based estimate: Cost effect	0.5 (+0.2 to +0.9)
C. Literature-based estimate: Factor shift effect	0.6 (+0 to +1.2)

We calculated a similar "demand effect" estimate that used the Morgenstern paper. EPA selected this paper because the parameter estimates (expressed in jobs per million [$1987] of environmental compliance expenditures) provide a transparent and tractable way to transfer estimates for an employment effects analysis. Similar estimates were not available from other studies. To do this, we multiplied the point estimate for the total demand effect (−3.56 jobs per million [$1987] of environmental compliance expenditure) by the total environmental compliance expenditures used in the partial equilibrium model. For example, the jobs effect estimate is estimated to be 807 jobs (−3.56 × $378 million × 0.6).[8] The timeframe for EPA's regulatory analysis focuses on a single year effect, by contrast the Morganstern analysis used annualized inputs, and translates to annualized impacts. Demand effect results are provided in Table 3-10. It is not appropriate to substitute the data from that approach in to the Morgenstern due to the incompatibilities of the underlying data. Since the result from the historical approach is within the confidence bounds for the Morgenstern results for the "demand effect", we are comfortable that the more general Morgenstern result is a good representation of the change in employment.

We also present the results of using the Morgenstern paper to estimate employment "cost" and "factor-shift" effects. Although using the Morgenstern parameters to estimate these "cost" and "factor-shift" employment changes is uncertain, it is helpful to compare the potential job gains from these effects to the job losses associated with the "demand" effect. Table 3-10 shows that using the "cost" and "factor shift" employment effects may offset employment loss

[8] Since Morgenstern's analysis reports environmental expenditures in 1987 dollars, we make an inflation adjustment to the engineering cost analysis using the consumer price index (195.3/113.6) = 0.6)

estimates using either "demand" effect employment losses. The 95% confidence intervals are shown for all of the estimates based on the Morgenstern parameters. As shown, at the 95% confidence level, we cannot be certain if net employment changes are positive or negative.

Although the Morgenstern paper provides additional information about the potential job effects of environmental protection programs, there are several qualifications EPA considered as part of the analysis. First, EPA has used the weighted average parameter estimates for a narrow set of manufacturing industries (pulp and paper, plastics, petroleum, and steel). Absent other data and estimates, this approach seems reasonable and the estimates come from a respected peer-reviewed source. However, EPA acknowledges the final rule covers an industry not considered in the original empirical study. By transferring the estimates to the cement sector, we make the assumption that estimates are similar in size. In addition, EPA assumes also that Morgenstern et al.'s estimates derived from the 1979–1991 are still applicable for policy taking place in 2013, almost 20 years later. Second, the economic impact model only considers near-term employment effects in the cement industry where production technologies are fixed. As a result, the economic impact model places more emphasis on the short-term "demand effect," whereas the Morgenstern paper emphasizes other important long-term responses. For example, positive job gains associated with "factor shift effects" are more plausible when production choices become more flexible over time and industries can substitute labor for other production inputs. Third, the Morgenstern paper estimates rely on sector demand elasticities that are different (typically bigger) from the demand elasticity parameter used in the cement model. As a result, the demand effects are not directly comparable with the demand effects estimated by the cement model. Fourth, Morgenstern identifies the industry average as economically and statistically insignificant effect (i.e., the point estimates are small, measured imprecisely, and not distinguishable from zero). EPA acknowledges this fact and has reported the 95% confidence intervals in Table 3-10. Fifth, Morgenstern's methodology assumes large plants bear most of the regulatory costs. By transferring the estimates, EPA assumes a similar distribution of regulatory costs by plant size and that the regulatory burden does not disproportionately fall on smaller plants.

3.3 Other Economic Analyses: Direct Compliance Cost Methods

Field Co

In addition to the market-level partial equilibrium analysis, EPA developed a separate economic analysis for the remaining 20 kilns that EPA anticipates will be affected by the final rule. These costs ($88 million, or 19%) were not included in the economic impact model analysis because of uncertainties and difficulties with developing an appropriate set of baseline cement market conditions for future years.

The total annualized costs for two white cement kilns are $2 million, or approximately $9 per metric ton of cement production. Using reported 2005 data from the USGS on the average mill net value of white cement ($176 per metric ton), this cost represents 5% of the product value.

EPA also conducted sales tests for 18 other kilns that were not included in the partial equilibrium analysis. The total annualized NESHAP cost for these 18 kilns is approximately $75 million. The median cost per ton is approximately $3.80 and ranges from $1.90 to $4.40 per ton of cement production. In addition, 7 of these 18 kilns would face an additional control cost above the NESHAP (approximately $1 dollar per metric ton) to meet the NSPS limits for SO_2 and NO_x.

The USGS reports that the real price of cement per metric ton (2005 dollars) has typically ranged between $75 and $100 since 1990. A sales test using these price data shows cost-to-sales ratios (CSRs) could range between 2% and 6%

Sales Test Ratio = Control Costs ($/ton)/F.O.B Cement Prices ($/ton).

From 2000 to 2006, the PCA reports that the average operating profit rates for the industry ranged from 17 to 21% (PCA, 2008c). If these profit data are representative of operating profit rates for new kilns, kilns could potentially significantly reduce their operating profit rates. As a result, companies may have the incentive to look for less expensive alternatives to meet the emission standards. If these alternatives are limited or not cost effective, the final rule may lead companies to consider delaying rates of construction of new kilns until market conditions change (e.g., increases in demand that lead to rising cement prices) to cover additional control costs.

3.4 Social Cost Estimates

Field Co

For the kilns modeled in our partial equilibrium model, the market adjustments in price and quantity were used to estimate the changes in aggregate economic welfare using applied welfare economics principles (see Appendix C). Higher cement prices and reduced consumption lead to consumer welfare losses ($540 million). Domestic producers (in aggregate) experience a net loss of $239 million. As noted in the previous section, individual domestic producers may gain or lose depending on the change in compliance costs versus the change in the regional market prices. The total domestic surplus loss (consumer and producers) totals $792 million.

For the kilns not modeled in our partial equilibrium model, the $88 million in engineering costs were multiplied by 1.8 to approximate the likely additional social cost associated with oligopoly market response. Thus the social cost estimate for the 20 kilns not in

the partial equilibrium model is $158 million. Because of the approximation used, we cannot estimate how the $158 million is distributed between consumers and producers.

Table 3-11. Distribution of Social Costs (10^6): 2005

Description	Social Cost Estimates: Final NESHAP and NSPS	Social Cost Estimates: Final NESHAP and More Stringent NSPS	EIA Social Cost Method
Change in consumer surplus	$551	$551	Partial-equilibrium model (baseline year 2005)
Change in domestic producer surplus	$241	$241	Partial-equilibrium model (baseline year 2005)
20 Kilns	$158	$187	Direct Compliance Method (scaled by 1.8 for oligopoly)
Change in domestic surplus	$950	$979	Combined methods
Adjustment if ten low utilization facilities idle or close (net negative because shut down facilities will not incur compliance costs).	-$24	-$24	
Total:	**$926-$950**	**$955-$979**	**Change with and without adjustment**
	Final NSPS Only	**More Stringent NSPS Only**	
Total:	**$72**	**$101**	**Direct compliance cost method** (scaled by 1.8 for oligopoly)
	Final NESHAP Only		
Change in consumer surplus	$551		Partial-equilibrium model (baseline year 2005)
Change in domestic producer surplus	$241		Partial-equilibrium model (baseline year 2005)
NSPS kilns (7 kilns)	$52		Direct compliance cost method (scaled by 1.8 for oligopoly)
Other kilns (13 kilns)	$86		Direct compliance cost method (scaled by 1.8 for oligopoly)
Change in domestic surplus	$930		Combined methods
Adjustment if ten low utilization facilities idle or close	$-24		
Total:	**$904-$930**		**Change with and without adjustment**

The estimated social cost of the final rule is $926-950 million. This estimate includes the results for existing kilns included in the partial-equilibrium analysis ($792 million),the final NESHAP direct compliance costs 20 kilns not included in the economic impact model, ($77 million), and the additional NSPS direct compliance cost for 7 kilns coming on line in the future ($11 million). The social estimates are significantly higher than the engineering analysis estimate of annualized costs totaling $466 million. This is a direct consequence of EPA's assumptions about existing market structure discussed extensively in previous cement industry rulemakings and in Section 2 and Appendix B of this RIA. Under baseline conditions without regulation, the existing domestic cement plants are assumed to choose a production level that is less than the level produced under perfect competition. As a result, a preexisting market distortion exists in the cement markets covered by the final rule (i.e., the observed baseline market price is higher than the [unobserved] market price that a model of perfect competition would predict). The imposition of additional regulatory costs tends to widen the gap between price and marginal cost in these markets and contributes to additional social costs. The above social costs for 2013 include annualized capital costs over the expected lifetime of the equipment and an opportunity cost of capital (7%) discount rate. To facilitate comparisons of benefits and costs when estimates vary of time across multiple years, EPA typically estimates a "consumption equivalent" present value measure of costs. This could be computed using a consumption rate of interest of 3% and 7%. However, this calculation was not necessary since the cost and benefit analyses only produce estimates for a single year (OAQPS, 1999a).

3.5 Energy Impacts

Executive Order 13211 (66 FR 28355, May 22, 2001) provides that agencies will prepare and submit to the Administrator of the Office of Information and Regulatory Affairs, OMB, a Statement of Energy Effects for certain actions identified as "significant energy actions." Section 4(b) of Executive Order 13211 defines "significant energy actions" as any action by an agency (normally published in the *Federal Register*) that promulgates or is expected to lead to the promulgation of a rule or regulation, including notices of inquiry, advance notices of final rulemaking, and notices of final rulemaking: (1) (i) that is a significant regulatory action under Executive Order 12866 or any successor order, and (ii) is likely to have a significant adverse effect on the supply, distribution, or use of energy; or (2) that is designated by the Administrator of the Office of Information and Regulatory Affairs as a significant energy action.

This rule is not a significant energy action as designated by the Administrator of the Office of Information and Regulatory Affairs because it is not likely to have a significant adverse

impact on the supply, distribution, or use of energy. EPA has prepared an analysis of energy impacts that explains this conclusion below.

To enhance understanding regarding the regulation's influence on energy consumption, EPA examined publicly available data describing the cement sector's energy consumption. The *AEO 2010* (DOE, 2010) provides energy consumption data. As shown in Table 3-12, this industry accounts for approximately 0.4% of the U.S. total energy consumption. As a result, any

Table 3-12. U.S. Cement Sector Energy Consumption (Trillion BTUs)[a]: 2013

	Quantity	Share of Total Energy Use
Residual fuel oil	0.9	0.00%
Distillate fuel oil	10.8	0.00%
Petroleum coke	47.3	0.10%
Other petroleum[b]	30.2	0.00%
Petroleum subtotal	89.2	0.10%
Natural gas	19.8	0.00%
Steam coal	206.6	0.20%
Metallurgical coal	6.8	0.00%
Coal subtotal	213.4	0.20%
Purchased electricity	38.9	0.00%
Total	399.44	0.40%
Delivered Energy Use	72,407	72.20%
Total Energy Use	100,592	100.00%

[a] Fuel consumption includes consumption for combined heat and power.

[b] Includes petroleum coke, lubricants, and miscellaneous petroleum products.

Source: U.S. Department of Energy, Energy Information Administration. 2010. Supplemental Tables to the Annual Energy Outlook 2010. Table 10 and Table 39. Available at <http://www.eia.doe.gov/oiaf/aeo/supplement/supref.html>.

energy consumption changes attributable to the regulatory program should not significantly influence the supply, distribution, or use of energy. EPA has also estimated the amount of additional electricity consumption associated with add-on controls. The analysis shows the additional national electrical demand to be 780 million kWh per year and the natural gas use to be 1.2 million MMBTU per year for existing kilns. For new kilns, assuming that of the 16 new kilns to start up by 2013, all 16 will add alkaline scrubbers and ACI systems, the electrical demand is estimated to be 199 million kWh per year. This is less than 0.1% of *AEO 2010* forecasts of total electricity and natural gas use.

3.6 Assessment

Although the economic analyses presented in this section cannot provide precise estimates of the final NESHAP's and NSPS's economic impacts, the evidence presented in this section suggests that the economic impacts may be significant across several dimensions (price, consumption, production, and international trade). There are several broad issues we emphasize as stakeholders review the analysis. First, OAQPS's partial equilibrium analysis of NESHAPs has traditionally been designed to assess small (marginal) changes in industry conditions. The

overall engineering cost analysis estimates are significant relative to the size of the U.S. cement market; EPA acknowledges that use of demand and import supply elasticities can be tenuous in these cases because the exact functional relationships (demand and supply) are less certain when simulated outcomes move further away from the observed pre-policy equilibrium. Second, the partial equilibrium assumes that transportation costs between regions are high enough that interregional trade is unlikely to occur, at least in the short run. Allowing interregional trade would expand the cement market definitions and increase the number of producers in each market. As discussed above, as the number of producers in a market increases, the production decision becomes more consistent with decisions made in pure competition; the additional trading opportunities may tend to moderate the relative price changes simulated within the model. Third, as discussed earlier in this section, the choice of market structure increases the agency's social cost estimate; it is almost 2 times higher than a model that assumes perfect competition. Therefore, the analysis may overstate the social costs of the rule. EPA continues to believe the market structure is reasonable and provides an upper-bound social cost estimate for the following reasons: (1) high transportation costs and other production economics tend to limit the number of sellers (particularly over a short time horizon), so each seller has a substantial regional market share; (2) timely market entry is also constrained by the high capital costs that involve purchases and construction of large rotary kilns that are not readily movable or transferable to other uses[9]; (3) cement producers offer very similar or identical products; and (4) the Office of Management and Budget (OMB) explicitly mentions the need to consider market power–related welfare costs in evaluating regulations under Executive Order 12866.

[9] In addition, large plants are typically more economical because they can produce cement at lower unit costs; this reduces entry incentives for smaller capacity cement plants.

SECTION 4
SMALL BUSINESS IMPACT ANALYSIS

The Regulatory Flexibility Act (RFA) generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a *significant* economic impact on a *substantial* number of small entities (SISNOSE). The first step in this assessment was to determine whether the rule will have SISNOSE. To make this determination, EPA used a screening and market analysis to indicate whether EPA can certify the rule as not having a SISNOSE. The elements of this analysis included

- identifying affected small entities,
- selecting and describing the measures and economic impact thresholds used in the analysis, and
- completing the assessment and determining the SISNOSE certification category.

4.1 Identify Affected Small Entities

For the purposes of assessing the impacts of the final rule on small entities, small entity is defined as (1) a small business as defined by the Small Business Administration's regulations at 13 CFR 121.201; according to these size standards, ultimate parent companies owning Portland cement manufacturing plants are categorized as small if the total number of employees at the firm is fewer than 750 (see Table 4-1 for list); (2) a small governmental jurisdiction that is a government of a city, county, town, school district, or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise that is independently owned and operated and is not dominant in its field. As reported in Section 2, EPA has identified four small entities (see Table 4-1). One of the four entities is owned by a small Tribal government (Salt River Pima-Maricopa Indian Community). The remaining three entities are small businesses.

4.2 Sales and Revenue Test Screening Analysis

In the next step of the analysis, EPA assessed how the regulatory program may influence the profitability of ultimate parent companies by comparing pollution control costs to total sales (i.e., a "sales" test). To do this, we divided an ultimate parent company's total annualized compliance costs by its reported revenue:

Table 4-1. Small Entity Analysis

Owner	Entity Type	Annual Sales (10^6)	Employees	Plants	Kilns	Clinker Capacity (10^3 metric tons per year)	Cost-to-Sales Ratio
Salt River Materials Group[a]	Tribal government	$184[b]	NA	1	1	1,477	0.7%
Monarch Cement Company	Business	$154	600	1	2	787	3.0%
Continental Cement Company, LLC	Business	$93[c]	<750	1	1	1,164	0.0%
Snyder Associate Companies	Business	$29	350	1	2	286	2.0%

[a] Enterprise is owned by Salt River Pima-Maricopa Indian Community.

[b] EPA estimate. Estimate uses revenue data for four of the six enterprises owned by Salt River Pima-Maricopa Indian Community.

[c] EPA estimate. Estimate uses cement production levels and average market prices.

$$CSR = \frac{\sum_{i}^{n} TACC}{TR_j} \tag{4.1}$$

where

CSR = cost-to-sales ratio,

TACC = total annualized compliance costs,

i = index of the number of affected plants owned by company j,

n = number of affected plants, and

TR_j = total sales from all operations of ultimate parent company j or annual government revenue.

The results of the screening analysis, presented in Table 4-1, show that no small businesses have a CSR greater than 3%. Two small business have an estimated CSR between 1 and 3%.

4.3 Additional Market Analysis

In additional to the screening analysis, EPA also examined small entity effects after accounting for market adjustments. Under this assumption, the entities recover some of the regulatory program costs as the market price adjusts in response to higher cement production costs. Even after accounting for these adjustments, small entity operating profits fall by less than 1 million.

4.4 Assessment

After considering the economic impact of this final rule on small entities, EPA has determined it will not have a significant economic impact on the four small entities. No small companies have cost-to-sales ratios greater than 3% and only 4 of the over 40 cement companies are small entities.

SECTION 5
AIR QUALITY MODELING OF EMISSION REDUCTIONS

5.1 Synopsis

This section describes the air quality modeling performed by EPA in support of the Portland cement NESHAP and NSPS. A national scale air quality modeling analysis was performed to estimate the impact of the sector emissions changes on future years: annual and 24-hour $PM_{2.5}$ concentrations, total Hg deposition, as well as visibility impairment. Air quality benefits are estimated with the Comprehensive Air Quality Model with Extensions (CAMx) model. CAMx simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone, PM, and air toxics. In addition to the CAMx model, the modeling platform includes the emissions, meteorology, and initial and boundary condition data which are inputs to this model.

Emissions and air quality modeling decisions are made early in the analytical process. For this reason, it is important to note that the inventories used in the air quality modeling and the benefits modeling are slightly different than the final adjusted cement kiln sector inventories presented in the RIA. However, the air quality inventories and the final rule inventories are generally consistent, so the air quality modeling adequately reflects the effects of the rule.

The 2005-based CAMx modeling platform was used as the basis for the air quality modeling for this final rule. This platform represents a structured system of connected modeling-related tools and data that provide a consistent and transparent basis for assessing the air quality response to projected changes in emissions. The base year of data used to construct this platform includes emissions and meteorology for 2005. The platform is intended to support a variety of regulatory and research model applications and analyses. This modeling platform and analysis is described fully below. Additional details about the modeling system are available in a separate technical support document: Air Quality Modeling Technical Support Document: National Emission Standards for Hazardous Air Pollutants from the Portland Cement Manufacturing Industry (U.S. EPA, 2010c).

5.2 Photochemical Model Background

CAMx version 5.10 is a freely available computer model that simulates the formation and fate of photochemical oxidants, primary and secondary PM concentrations, and air toxics, over regional and urban spatial scales for given input sets of meteorological conditions and emissions. CAMx includes numerous science modules that simulate the emission, production, decay,

deposition and transport of organic and inorganic gas-phase and particle-phase pollutants in the atmosphere (Nobel, McDonald-Buller et al., 2001; Baker and Scheff, 2007; Russell, 2008).

CAMx is applied with ISORROPIA inorganic chemistry (Nenes et al., 1999), a semivolatile equilibrium scheme to partition condensable organic gases between gas and particle phase (Strader et al., 1999), Regional Acid Deposition Model (RADM) aqueous phase chemistry (Chang et al., 1987), and Carbon Bond 05 (CB05) gas-phase chemistry module (Gery et al., 1989; ENVIRON, 2008). All modeling domains were modeled for the entire year of 2005. Data from the entire year were used when looking at the estimation of $PM_{2.5}$, total Hg deposition, and visibility impacts from the regulation.

5.3 Model Domain and Grid Resolution

The modeling analyses were performed for a domain covering the continental United States, as shown in Figure 5-1. This domain has a parent horizontal grid of 36 km with two finer-scale 12 km grids over portions of the eastern and western United States. The model extends vertically from the surface to 100 millibars (approximately 15 km) using a sigma-pressure coordinate system. Air quality conditions at the outer boundary of the 36 km domain were taken from a global model and did not change over the simulations. In turn, the 36 km grid was only used to establish the incoming air quality concentrations along the boundaries of the 12 km grids. Only the finer grid data were used in determining the impacts of the emission standard program changes. Table 5-1 provides some basic geographic information regarding the photochemical model domains.

5.4 Emissions Input Data

The emissions data used in the base year and future reference and future emissions adjustment case are based on the 2005 v4 platform. The emissions cases use some different emissions data than the official v4 platform to use data intended only for the rule development and not for general use. Unlike the 2005 v4 platform, the configuration for this modeling application included some additional HAPs and a cement kiln sector emissions inventory more consistent with the engineering analysis of potential control options.

The 2013 reference case is intended to represent the emissions associated with growth and controls in that year. The U.S. EGU point source emissions estimates for the future year reference and control case are based on an Integrated Planning Model (IPM) run for criteria pollutants, HCl, and Hg in 2013 (although HCl was not modeled). Both control and growth factors were applied to a subset of the 2005 non-EGU point and nonpoint to create the 2013

reference case. The 2002 v3.1 platform 2020 projection factors were the starting point for most of the 2013 SMOKE-based projections.

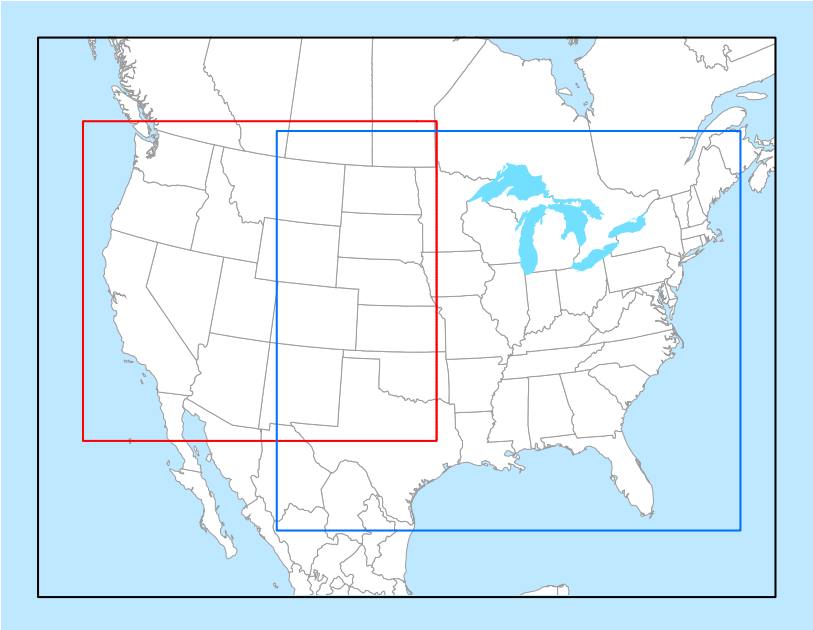

Figure 5-1. Map of the Photochemical Modeling Domain[a]

[a] The black outer box denotes the 36 km national modeling domain; the red inner box is the 12 km western U.S. grid; and the blue inner box is the 12 km eastern U.S. grid.

Table 5-1. Geographic Elements of Domains Used in Photochemical Modeling

	Photochemical Modeling Configuration		
	National Grid	**Western U.S. Fine Grid**	**Eastern U.S. Fine Grid**
Map Projection	Lambert Conformal Projection		
Grid Resolution	36 km	12 km	12 km
Coordinate Center	97 deg W, 40 deg N		
True Latitudes	33 deg N and 45 deg N		
Dimensions	148 x 112 x 14	213 x 192 x 14	279 x 240 x 14
Vertical extent	14 Layers: Surface to 100 millibar level		

The 2013 reference scenario for the cement kiln sector assumed no growth or control for the industry from the 2005 sector emissions estimates with the exception that facilities that closed between 2005 and 2010 were removed from the 2013 inventory. The length of time

required to conduct emissions and photochemical modeling precludes using the final facility-specific emissions estimates based on controls implemented for this rule. A 2013 "control" or emissions adjustment case was developed by removing all Portland cement sector emissions from the 2013 baseline inventory. This "zero-out" of the sector creates a policy space where potential controls would be maximized at all locations. Since this is unrealistic, the air quality estimates from the 2013 "zero-out" or "control" case are adjusted to reflect nationwide estimates of control percentages by pollutant. It is important to note that the scenario without cement kilns includes the zeroing-out of emissions from hazardous waste kilns. Out of 181 kilns nationwide, there are 14 hazardous waste kilns, which represent 10 to 20% of total kiln emissions. This leads to a slight overestimate of the reduction in $PM_{2.5}$ levels and mercury deposition.

Table 5-2. Cement Kiln Emissions in 2005 Base and Estimated Future Year (2013) in tons per year

Specie	2005	2013
Nitrogen Oxides	216,525	199,391
Volatile Organic Compounds	8,817	8,419
Sulfur Dioxide	158,560	149,013
Primary$PM_{2.5}$	16,758	15,403
$PM_{2.5}$ Mercury	0.8	0.7
Reactive Gas Phase Mercury	6.2	6.0
Elemental Mercury	3.8	3.6

The air quality estimates associated with 2013 zero-out of the cement kiln sector are adjusted nationally to reflect various options.

- A 90% reduction in mercury emissions for the NSPS and NESHAP , more stringent NSPS and NESHAP, and NESHAP only
- 82% reductions in SO_X and 86% reductions in primarily emitted $PM_{2.5}$ for the NSPS and NESHAP, more stringent NSPS and NESHAP, and NESHAP only
- 6% reductions in SO_X and 5% reductions in primarily emitted $PM_{2.5}$ for NSPS only

As part of the analysis for this rulemaking, the modeling system was used to calculate daily and annual $PM_{2.5}$ concentrations, annual total Hg deposition levels, and visibility impairment. Model predictions are used in a relative sense to estimate scenario-specific future-

year design values of $PM_{2.5}$ and ozone. Specifically, we compare a 2013 reference scenario, a scenario without the cement kiln controls, to a 2013 control scenario that includes the adjustments to the cement kiln sector. This is done by calculating the simulated air quality ratios between any particular future year simulation and the 2005 base. These predicted ratios are then applied to ambient base year design values. The design value projection methodology used here followed EPA guidance for such analyses (U.S. EPA, 2007). Additionally, the raw model outputs are also used in a relative sense as inputs to the health and welfare impact functions of the benefits analysis. Only model predictions for Hg deposition were analyzed using absolute model changes, although these parameters also considered percentage changes between the control case and two future baselines.

5.5 Model Results: Air Quality Impacts

As described above, we performed a series of air quality modeling simulations for the continental United States to assess the impacts of emissions adjustments to the Portland cement kiln sector. We looked at impacts on future ambient $PM_{2.5}$, total Hg deposition levels, and visibility impairment. In this section, we present information on current and projected levels of pollution for 2013.

This section summarizes the results of our modeling of differences in total Hg deposition impacts in the future based on changes to the cement kiln emissions. Specifically, we compare a 2013 reference scenario to a 2013 emissions change scenario (approximating a nationwide 90% reduction to mercury emissions). Model results for the eastern and central United States indicate that total Hg deposition (wet and dry forms) would be reduced by a total of 63,518 $\mu g/m^2$. A reduction of 26,047 $\mu g/m^2$ is estimated for the western United States. The reductions to total annual Hg deposition estimated by the photochemical model show that the reductions tend to be greatest nearest the sources.

This section summarizes the results of our modeling of annual average $PM_{2.5}$ air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2013 reference scenario to a 2013 control scenario. The modeling assessment indicates that a decrease up to 0.3 $\mu g/m^3$ in annual $PM_{2.5}$ design values is possible given an area's proximity to controlled sources and the amount of reduced sulfur dioxide emissions. The median reduction over all monitor locations is 0.09 $\mu g/m^3$. An annual $PM_{2.5}$ design value is the concentration that determines whether a monitoring site meets the annual NAAQS for $PM_{2.5}$. The full details involved in calculating an annual $PM_{2.5}$ design value are given in Appendix N of 40 CFR part 50.

Projected air quality benefits are estimated using procedures outlined by EPA modeling guidance (U.S. EPA, 2007).

This section summarizes the results of our modeling of 24-hour average $PM_{2.5}$ air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2013 reference scenario to a 2013 control scenario. The modeling assessment indicates that a decrease up to 0.5 $\mu g/m^3$ in 24-hour average $PM_{2.5}$ design values at most monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced sulfur dioxide emissions. The median reduction over all monitor locations is 0.1 $\mu g/m^3$. A 24-hour $PM_{2.5}$ design value is the concentration that determines whether a monitoring site meets the 24-hour NAAQS for $PM_{2.5}$. The full details involved in calculating a 24-hour $PM_{2.5}$ design value are given in Appendix N of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by EPA modeling guidance (U.S. EPA, 2007).

Air quality modeling conducted for this final rule was used to project visibility conditions in 138 mandatory Class I federal areas across the United States in 2013 (U.S. EPA, 2007). The level of visibility impairment in an area is based on the light-extinction coefficient and a unitless visibility index, called a "deciview," that is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. Higher deciview values are indicative of worse visibility. Thus, an improvement in visibility is a decrease in deciview value. The modeling assessment indicates that a decrease up to 0.31 deciviews in annual 20% worst visibility days is possible given an area's proximity to controlled sources and the amount of reduced sulfur dioxide emissions. Median reductions are 0.01 deciviews to the 20% worst days and 20% best days over all monitor locations.

5.6 Limitations (Uncertainties) Associated with the Air Quality Modeling

Any deficiencies with the emissions or meteorological inputs may lead to control scenario estimates that may not fully characterize the source contribution mix at a receptor location. This application used a complete year of meteorology to capture the variety of meteorological formation regimes conducive to eleveted pollution. However, it is possible that the meteorology used for these model applications may not represent all elevated pollution formation regimes at every individual receptor location in the continental United States.

SECTION 6
BENEFITS OF EMISSIONS REDUCTIONS

6.1 Synopsis

In this section, we provide an estimate of the monetized benefits associated with reducing exposure to particulate matter (PM) for the final Portland Cement NESHAP and NSPS. The PM reductions are the result of emission limits on PM as well as emission limits on other pollutants, including hazardous air pollutants (HAPs) for the NESHAP and criteria pollutants for the NSPS. The total $PM_{2.5}$ reductions are the consequence of the technologies installed to meet these multiple limits. These estimates include the number of cases of avoided morbidity and premature mortality among populations exposed to $PM_{2.5}$, as well as the monetized value of those avoided cases. Using a 3% discount rate, we estimate the total monetized benefits of the final Cement NESHAP and NSPS to be $7.4 billion to $18 billion in the implementation year (2013). Using a 7% discount rate, we estimate the total monetized benefits of the final Cement NESHAP and NSPS to be $6.7 billion to $17 billion in the implementation year. All estimates are in 2005$. These estimates include the energy disbenefits associated with increased electricity usage by the control devices.

These monetized estimates reflect EPA's most current interpretation of the scientific literature and several methodology updates introduced in the proposal analysis. In addition, these estimates incorporate an array of improvements since the proposal, including cement sector-specific air quality modeling data, revised value-of-a-statistical-life (VSL), lowest measure level (LML) assessment, qualitative benefits for ecosystems and HAPs, and mercury deposition maps. Higher or lower estimates of benefits are possible using other assumptions; examples of this are provided in Figure 6-1. Data, resource, and methodological limitations prevented EPA from monetizing the benefits from several important benefit categories, including benefits from reducing hazardous air pollutants, ecosystem effects, and visibility impairment. The benefits from reducing other air pollutants have not been monetized in this analysis, including reducing 4,400 tons of NO_x, 5,800 tons of HCl, 5,200 tons of organic HAPs, and 16,400 pounds of mercury each year.

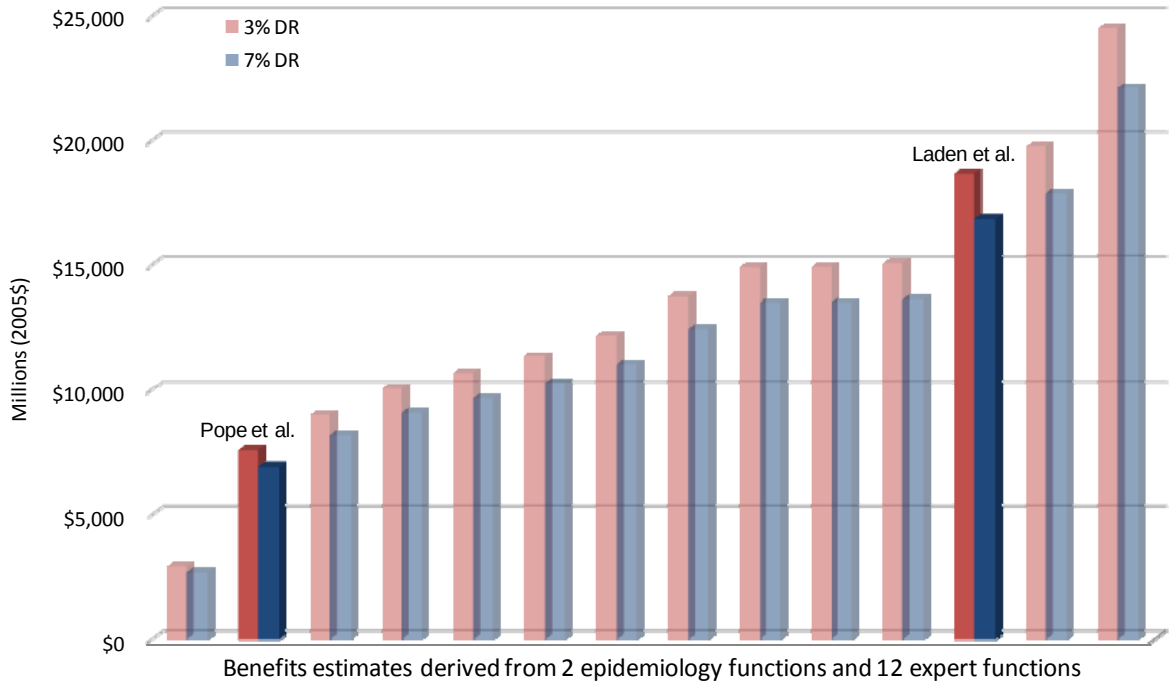

Figure 6-1. Total Monetized $PM_{2.5}$ Benefits for the Final Cement NESHAP and NSPS in 2013 [a]

[a] This graph shows the estimated benefits at discount rates of 3% and 7% using effect coefficients derived from the Pope et al. study and the Laden et al study, as well as 12 effect coefficients derived from EPA's expert elicitation on PM mortality. The results shown are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. These estimates do not include benefits from reducing HAP emissions, but they do include the energy disbenefits. Due to data, methodology, and resource limitations, we were unable to monetize the benefits associated with several categories of benefits, including exposure to HAPs, NO_2, and SO_2, ecosystem effects, and visibility effects.

6.2 Calculation of $PM_{2.5}$ Human Health Benefits

In addition to pollutants we cannot monetize, this rulemaking would reduce emissions of $PM_{2.5}$ and SO_2. Because SO_2 is also a precursor to $PM_{2.5}$, reducing and SO_2 emissions would also reduce $PM_{2.5}$ formation, human exposure, and the incidence of $PM_{2.5}$-related health effects. The PM reductions are the result of emission limits on PM as well as emission limits on other pollutants, including hazardous air pollutants for the NESHAP and criteria pollutants for the NSPS. The total $PM_{2.5}$ reductions are the consequence of the technologies installed to meet these multiple limits.

6.2.1 Methodology Improvements since Proposal

This benefits analysis incorporates an array of policy and technical improvements since the proposal RIA in 2009 (U.S. EPA, 2009a), including:

1. *Cement sector-specific air quality modeling data*. The benefits estimates for this final analysis are based on air quality data modeled by CAMx that reflect the emissions from the cement sector and the reductions anticipated as a result of this rule. This data provides a superior representation of the geographic distribution of the emission reductions and resulting ambient concentrations than the national average benefit-per-ton estimates used in the proposal. For more information regarding the modeling inputs and assumptions, please see Section 5 of this RIA.

2. *Use of a revised Value of Statistical Life (VSL)*. The Agency continues to update its guidance on valuing mortality risk reductions and until a final report is available, EPA now uses a single, peer-reviewed mean VSL estimate of $6.3 million (2000$). We discuss this issue in more detail in Section 6.2.5.

3. *Lowest Measured Level (LML) assessment*. Consistent with the rationale outlined in the proposal RIA, EPA now estimates PM-related mortality without assuming an arbitrary threshold in the concentration-response function. Consistent with recent scientific advice, we are replacing the previous threshold sensitivity analysis with a new LML assessment to highlight the uncertainty associated with benefits estimated at low air quality levels. We discuss this issue in more detail in Section 6.2.4 and provide the results of this LML assessment in Section 6.3.

4. *Qualitative benefits for ecosystems and HAPs.* Data, resource, and methodological limitations prevented EPA from quantifying or monetizing the benefits from several important benefit categories, including benefits from reducing toxic air pollutant emissions, ecosystem effects, and visibility impairment. Instead, we provide a qualitative description of the benefits anticipated as a result of the emission reductions from this rule. These unquantified benefits are described in Section 6.5.

5. *Mercury deposition*. The air quality modeling data provide an estimate of the reduction in mercury deposition associated with the mercury emission reductions anticipated as a result of this rule. We provide maps of the reduced mercury deposition in Section 6.3.2.1. Due to time and resource limitations, we were unable to model mercury methylation, bioaccumulation in fish tissue, and human consumption of mercury-contaminated fish that would be needed in order to estimate the human health benefits from reducing mercury emissions.

6.2.2 Benefits Analysis Approach

We follow a "damage-function" approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all non-overlapping

health and welfare endpoints. The "damage-function" approach is the standard method for assessing costs and benefits of environmental quality programs and has been used in several recent published analyses (Levy et al., 2009; Hubbell et al., 2009; Tagaris et al., 2009).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. For changes in PM, a health impact analysis (HIA) must first be conducted to convert air quality changes into effects that can be assigned dollar values. For this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to PM. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, including the benefits associated with decreasing deposition of sulfur to terrestrial and aquatic ecosystems, but we are unable to place an economic value on these changes due to time and resource limitations.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure directly either the health outcomes or their values for regulatory analyses. Thus, similar to Kunzli et al. (2001) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the socio-demographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

6.2.3 Health Impact Analysis (HIA)

The HIA quantifies the changes in the incidence of adverse health impacts resulting from changes in human exposure to $PM_{2.5}$ air quality. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts resulting from population-level changes in exposure to pollutants (Levy et al. 2009). Analysts have applied the HIA approach to estimate human health impacts resulting from hypothetical changes in pollutant levels (Hubbell et al. 2005; Davidson et al. 2007, Tagaris et al. 2009). For this analysis, we used the environmental Benefits Mapping and Analysis Program (BenMAP), which is a PC-based tool that can systematize health impact analyses by applying a database of key input parameters, including health impact functions and population projections.[1]

[1] For this analysis, we used BenMAP version 3.0 (Abt Associates, 2008). This model is available for free download on the Internet at <http://www.epa.gov/air/benmap>.

The HIA approach used in this analysis involves three basic steps: (1) utilizing CAMx-generated projections of $PM_{2.5}$ air quality and estimating the change in the spatial distribution of the ambient air quality; (2) determining the subsequent change in population-level exposure; (3) calculating health impacts by applying concentration-response relationships drawn from the epidemiological literature (Hubbell et al. 2009) to this change in population exposure.

A typical health impact function might look as follows:

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

where y_0 is the baseline incidence rate for the health endpoint being quantified (for example, a health impact function quantifying changes in mortality would use the baseline, or background, mortality rate for the given population of interest); *Pop* is the population affected by the change in air quality; Δx is the change in air quality; and β is the effect coefficient drawn from the epidemiological study. For this analysis, we systematize the HIA calculation process using BenMAP's library of existing air quality monitoring data, population data and health impact functions. Figure 6-2 provides a simplified overview of this approach, and Figure 6-3 identifies the data inputs and outputs for the BenMAP model.

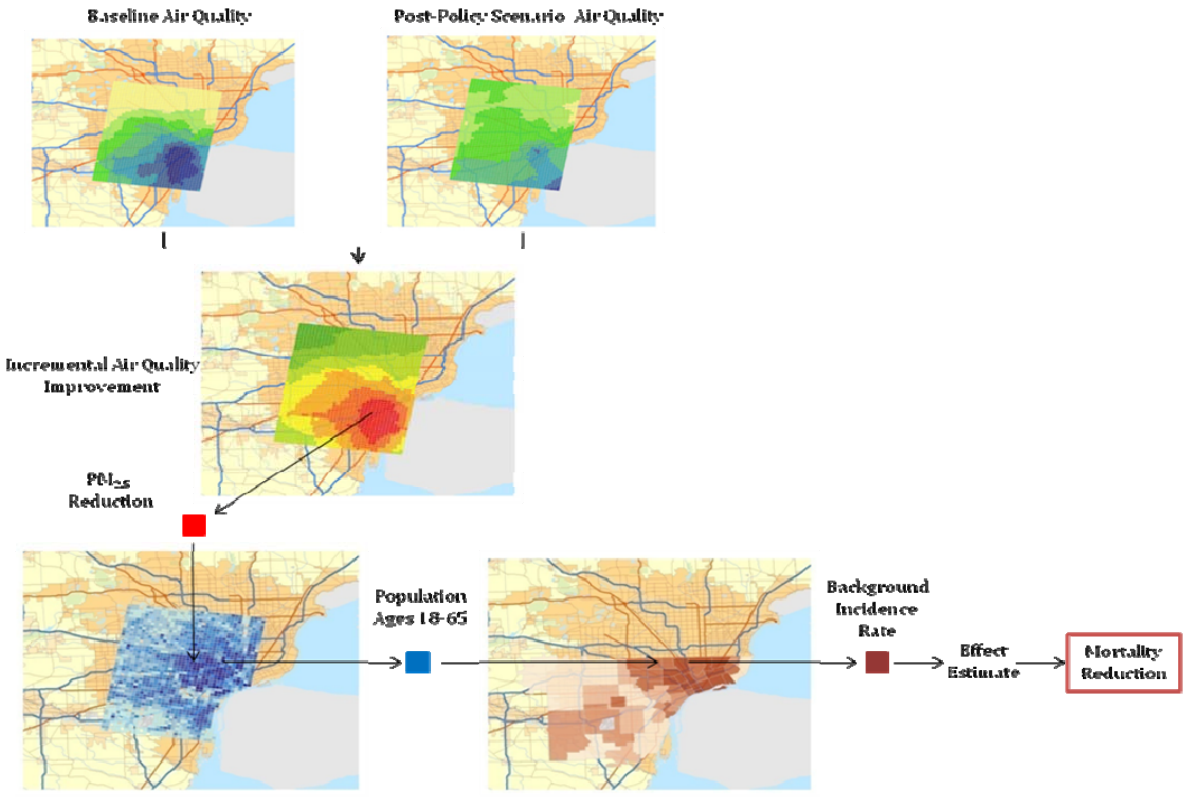

Figure 6-2. Illustration of BenMAP Approach

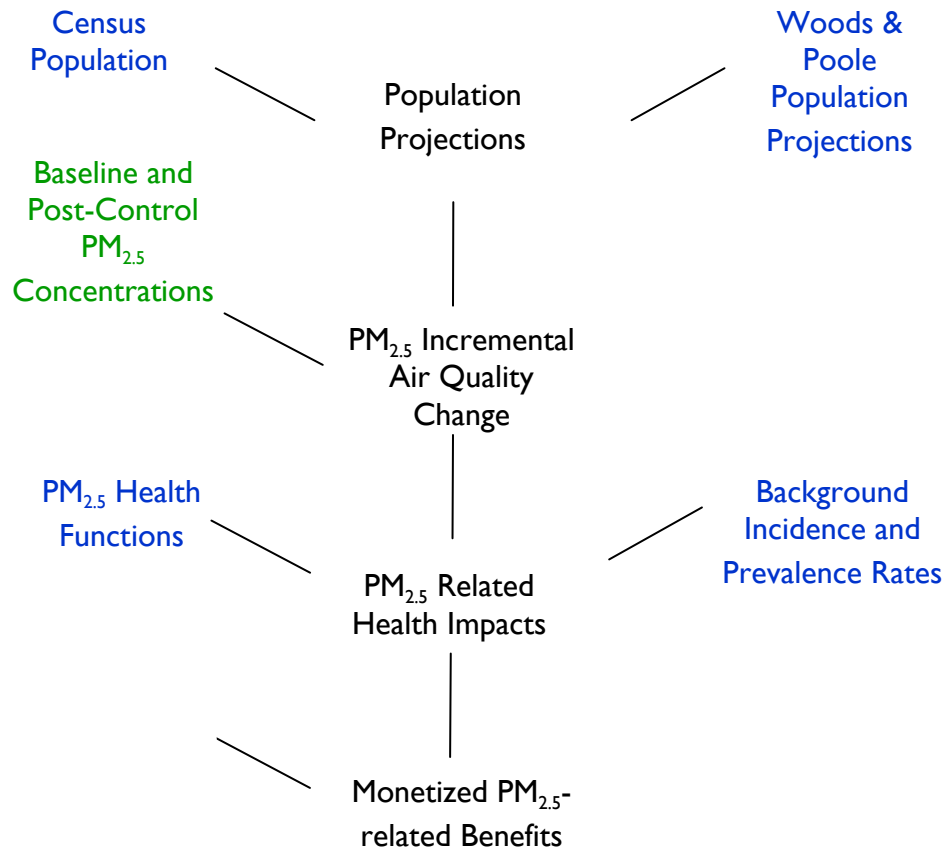

Figure 6-3. Data inputs and outputs for the BenMAP model

The benefits estimates in this analysis were derived using modified versions of the health impact functions used in the PM NAAQS Regulatory Impact Analysis (RIA) (U.S. EPA, 2006). While many of the functions are identical to those used in the PM NAAQS RIA, we have updated a few of the underlying assumptions over the last few years. For a detailed description of the underlying functions, studies, baseline incidence rates, and population data used in this analysis, please refer to Chapter 5 of the recently proposed Transport Rule (U.S. EPA, 2010a). Table 6-1 identifies which human health and welfare endpoints are included in the monetized benefits and which endpoints are unquantified. In summary, the monetized PM benefits include premature mortality and 11 morbidity endpoints.

Table 6-1. Human Health and Welfare Effects of Pollutants Affected

Pollutant/ Effect	*Quantified and monetized in primary estimate*	*Unquantified*
PM: health[a]	Premature mortality based on cohort study estimates[b] Premature mortality based on expert elicitation estimates Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarctions) Lower and upper respiratory illness Minor restricted activity days Work loss days Asthma exacerbations (among asthmatic populations Respiratory symptoms (among asthmatic populations) Infant mortality	Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Non-asthma respiratory emergency room visits UVb exposure (+/-)[c]
PM: welfare		Visibility in Class I areas in SE, SW, and CA regions Visibility in residential areas Visibility in non-class I areas and class 1 areas in NW, NE, and Central regions UVb exposure (+/-)[c] Global climate impacts[c]
SO_2: health		Respiratory hospital admissions Asthma emergency room visits Asthma exacerbation Acute respiratory symptoms Premature mortality Pulmonary function
SO_X: welfare		Commercial fishing and forestry from acidic deposition effects Recreation in terrestrial and aquatic ecosystems from acid deposition effects Increased mercury methylation
Mercury: health		Incidence of neurological disorders Incidence of learning disabilities Incidences in developmental delays Potential cardiovascular effects including: --Altered blood pressure regulation --Increased heart rate variability --Incidences of Myocardial infarction Potential reproductive effects
Mercury: welfare		Impact on birds and mammals (e.g. reproductive effects) Impacts to commercial, subsistence and recreational fishing

[a] In addition to primary economic endpoints, there are a number of biological responses that have been associated with PM health effects including morphological changes and altered host defense mechanisms. The public health impact of these biological responses may be partly represented by our quantified endpoints.

[b] Cohort estimates are designed to examine the effects of long term exposures to ambient pollution, but relative risk estimates may also incorporate some effects due to shorter term exposures (see Kunzli et al., 2001 for a discussion of this issue). While some of the effects of short term exposure are likely to be captured by the cohort estimates, there may be additional premature mortality from short term PM exposure not captured in the cohort estimates included in the primary analysis.

[c] May result in benefits or disbenefits.

6.2.4 Estimating $PM_{2.5}$-related premature mortality

Consistent with the proposal RIA for this rule (U.S. EPA, 2009a), the $PM_{2.5}$ benefits estimates utilize the concentration-response functions as reported in the epidemiology literature, as well as the 12 functions obtained in EPA's expert elicitation study as a characterization of uncertainty.

- One estimate is based on the concentration-response (C-R) function developed from the extended analysis of American Cancer Society (ACS) cohort, as reported in Pope et al. (2002), a study that EPA has previously used to generate its primary benefits estimate. When calculating the estimate, EPA applied the effect coefficient as reported in the study without an adjustment for assumed concentration threshold of 10 $\mu g/m^3$ as was done in recent (2006-2009) Office of Air and Radiation RIAs.

- One estimate is based on the C-R function developed from the extended analysis of the Harvard Six Cities cohort, as reported by Laden et al. (2006). This study, published after the completion of the Staff Paper for the 2006 $PM_{2.5}$ NAAQS, has been used as an alternative estimate in the $PM_{2.5}$ NAAQS RIA and $PM_{2.5}$ benefits estimates in RIAs completed since the $PM_{2.5}$ NAAQS. When calculating the estimate, EPA applied the effect coefficient as reported in the study without an adjustment for assumed concentration threshold of 10 $\mu g/m^3$ as was done in recent (2006-2009) RIAs.

- Twelve estimates are based on the C-R functions from EPA's expert elicitation study (IEc, 2006; Roman et al., 2008) on the $PM_{2.5}$ -mortality relationship and interpreted for benefits analysis in EPA's final RIA for the $PM_{2.5}$ NAAQS. For that study, twelve experts (labeled A through L) provided independent estimates of the $PM_{2.5}$ -mortality concentration-response function. EPA practice has been to develop independent estimates of $PM_{2.5}$ -mortality estimates corresponding to the concentration-response function provided by each of the twelve experts, to better characterize the degree of variability in the expert responses.

The effect coefficients are drawn from epidemiology studies examining two large population cohorts: the American Cancer Society cohort (Pope et al., 2002) and the Harvard Six Cities cohort (Laden et al., 2006).[2] These are logical choices for anchor points in our presentation because, while both studies are well designed and peer reviewed, there are strengths and weaknesses inherent in each, which we believe argues for using both studies to generate benefits estimates. Previously, EPA had calculated benefits based on these two empirical studies, but derived the range of benefits, including the minimum and maximum results, from an expert elicitation of the relationship between exposure to $PM_{2.5}$ and premature mortality (Roman et al.,

[2] These two studies specify multi-pollutant models that control for SO_2, among other pollutants.

2006).[3] Within this assessment, we include the benefits estimates derived from the concentration-response function provided by each of the twelve experts to better characterize the uncertainty in the concentration-response function for mortality and the degree of variability in the expert responses. Because the experts used these cohort studies to inform their concentration-response functions, benefits estimates using these functions generally fall between results using these epidemiology studies (see Figure 6-1). In general, the expert elicitation results support the conclusion that the benefits of $PM_{2.5}$ control are very likely to be substantial.

EPA strives to use the best available science to support our benefits analyses, and we recognize that interpretation of the science regarding air pollution and health is dynamic and evolving. This analysis continues to use the updated assumptions first applied in the proposal RIA for this rule (U.S. EPA, 2009a), including the updated population dataset in BenMAP 3.0 and the functions directly from the epidemiology studies without an adjustment for an assumed threshold.[4] Removing the threshold assumption is a key difference between the method used in this analysis of PM benefits and the methods used in RIAs prior to the proposal RIA for this rule, and we now calculate incremental benefits down to the lowest modeled $PM_{2.5}$ air quality levels.[5] Prior to the proposal RIA for this rule, EPA presented the results using an assumed threshold at 10 $\mu g/m^3$ in the PM-mortality health impact function as the primary PM-related benefits results. Using a threshold of 10 $\mu g/m^3$ was an arbitrary choice, and we could have assumed thresholds at other points in the lower end of the observed range the analysis. Since the proposal RIA for this rule, EPA included a sensitivity analysis with an assumed threshold at 10 $\mu g/m^3$ to illustrate that the fraction of benefits that occur at lower air pollution concentration levels are inherently more uncertain.

In the proposal RIA for this rule, EPA solicited comment on the use of the no-threshold model for benefits analysis within the preamble.[6] Based on our review of the public comments as well as the current body of scientific literature, EPA now estimates PM-related mortality without applying an assumed concentration threshold. EPA's Integrated Science Assessment for Particulate Matter (U.S. EPA, 2009b), which was recently reviewed by EPA's Clean Air

[3] Please see the Section 5.2 of the proposal RIA for this rule for more information regarding the change in the presentation of benefits estimates.

[4] The benefits methodology has also been updated since the proposal RIA to incorporate a revised VSL, as discussed in the next section.

[5] It is important to note that uncertainty regarding the shape of the concentration-response function is conceptually distinct from an assumed threshold. An assumed threshold (below which there are no health effects) is a discontinuity, which is a specific example of non-linearity.

[6] The comment period for the proposed rule closed on September 4, 2009 (Docket ID No. EPA–HQ–OAR–2002–0051 available at http://www.regulations.gov). All public comments received as well as the responses to those comments are available in this docket.

Scientific Advisory Committee (U.S. EPA-SAB, 2009a; U.S. EPA-SAB, 2009b), concluded that the scientific literature consistently finds that a no-threshold log-linear model most adequately portrays the PM-mortality concentration-response relationship while recognizing potential uncertainty about the exact shape of the concentration-response function. Since then, the Health Effects Subcommittee (U.S. EPA-SAB, 2010) of EPA's Council concluded, "The HES fully supports EPA's decision to use a no-threshold model to estimate mortality reductions. This decision is supported by the data, which are quite consistent in showing effects down to the lowest measured levels. Analyses of cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. Therefore, there is no evidence to support a truncation of the CRF." In conjunction with the underlying scientific literature, this document provided a basis for reconsidering the application of thresholds in $PM_{2.5}$ concentration-response functions used in EPA's RIAs. For a summary of these scientific review statements and the panel members please consult the Technical Support Document (TSD) entitled *Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for PM-related Mortality* (U.S. EPA, 2010b), which is provided in Appendix D of this RIA.

Consistent with recent scientific advice, we are replacing the previous threshold sensitivity analysis with a new "Lowest Measured Level" (LML) assessment. This approach summarizes the distribution of avoided PM mortality impacts according to the baseline $PM_{2.5}$ levels experienced by the population receiving the $PM_{2.5}$ mortality benefit. In the results section, we identify on the figures the lowest air quality levels measured in each of the primary cohort studies that estimate PM-related mortality. This information allows readers to determine the portion of PM-related mortality benefits occurring above or below the LML of each study; in general, our confidence in the estimated PM mortality decreases as we consider air quality levels further below the LML in the two epidemiological studies.

While an LML assessment provides some insight into the level of uncertainty in the estimated PM mortality benefits, EPA does not view the LML as a threshold and continues to quantify PM-related mortality impacts using a full range of modeled air quality concentrations. Unlike an assumed threshold, which is a modeling assumption that reduces the magnitude of the estimated health impacts, the LML is a characterization of the fraction of benefits that are more uncertain. It is important to emphasize that just because we have greater confidence in the benefits above the LML, this does not mean that we have no confidence that benefits occur below the LML.

Analyses of these cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. As we model mortality impacts among populations exposed to levels of $PM_{2.5}$ that are successively lower than the LML of each study, our confidence in the results diminishes. As air pollution emissions continue to decrease over time, there will be more people in areas where we do not have published epidemiology studies. However, each successive cohort study has shown evidence of effects at successively lower levels of $PM_{2.5}$. As more large cohort studies follow populations over time, we will likely have more studies with lower LML as air quality levels continue to improve. Even in the absence of a definable threshold, we have more confidence in the benefits estimates above the LML of the large cohort studies. To account for the uncertainty in each of the studies that we base our mortality estimates on, we provide the LML for each of the cohort studies. However, the finding of effects at the lowest LML from recent studies indicate that confidence in $PM_{2.5}$-related mortality effects down to at least 7.5 $\mu g/m^3$ is high.

For these rules the SO_2 reductions represent a large fraction of the total benefits from reducing $PM_{2.5}$, but it is not possible to isolate the portion if the total benefits attributable to the emission reductions of SO_2 resulting from the application of HCl controls. The benefits models assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality because there is no clear scientific evidence that would support the development of differential effects estimates by particle type.

6.2.5 Economic valuation of health impacts

After quantifying the change in adverse health impacts, the final step is to estimate the economic value of these avoided impacts. Please refer to Table 5-11 in the recently proposed Transport Rule (U.S. EPA, 2010a) for a detailed description of the underlying valuation functions and the monetized unit values for each endpoint incorporated into this analysis.[7] The monetized mortality benefits dominate the total benefits estimates.

[7] To comply with Circular A-4, EPA provides monetized benefits using discount rates of 3% and 7% (OMB, 2003). These benefits are estimated for a specific analysis year (i.e., 2013), and most of the PM benefits occur within that year with two exceptions: acute myocardial infarctions (AMIs) and premature mortality. For AMIs, we assume 5 years of follow-up medical costs and lost wages. For premature mortality, we assume that there is a "cessation" lag between PM exposures and the total realization of changes in health effects. Although the structure of the lag is uncertain, EPA follows the advice of the SAB-HES to assume a segmented lag structure characterized by 30% of mortality reductions in the first year, 50% over years 2 to 5, and 20% over the years 6 to 20 after the reduction in $PM_{2.5}$ (U.S. EPA-SAB, 2004). Changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths. Therefore, discounting only affects the AMI costs after the analysis year and the valuation of premature mortalities that occur after the analysis year. As such, the monetized benefits using a 7% discount rate are only approximately 10% less than the monetized benefits using a 3% discount rate.

As is the nature of RIAs, the assumptions and methods used to estimate air quality benefits evolve over time to reflect the Agency's most current interpretation of the scientific and economic literature. For a period of time (2004–2006), the Office of Air and Radiation (OAR) valued mortality risk reductions using a value-of-a-statistical-life (VSL) estimate derived from a limited analysis of some of the available studies. OAR arrived at a VSL using a range of $1 million to $10 million (2000$) consistent with two meta-analyses of the wage-risk literature. The $1 million value represented the lower end of the interquartile range from the Mrozek and Taylor (2002) meta-analysis of 33 studies. The $10 million value represented the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis of 43 studies. The mean estimate of $5.5 million (2000$)[8] was also consistent with the mean VSL of $5.4 million estimated in the Kochi et al. (2006) meta-analysis. However, the Agency neither changed its official guidance on the use of VSL in rule-makings nor subjected the interim estimate to a scientific peer-review process through the Science Advisory Board (SAB) or other peer-review group.

During this time, the Agency continued work to update its guidance on valuing mortality risk reductions, including commissioning a report from meta-analytic experts to evaluate methodological questions raised by EPA and the SAB on combining estimates from the various data sources. In addition, the Agency consulted several times with the Science Advisory Board Environmental Economics Advisory Committee (SAB-EEAC) on the issue. With input from the meta-analytic experts, the SAB-EEAC advised the Agency to update its guidance using specific, appropriate meta-analytic techniques to combine estimates from unique data sources and different studies, including those using different methodologies (i.e., wage-risk and stated preference) (U.S. EPA-SAB, 2007).

Until updated guidance is available, the Agency determined that a single, peer-reviewed estimate applied consistently best reflects the SAB-EEAC advice it has received. Therefore, the Agency has decided to apply the VSL that was vetted and endorsed by the SAB in the Guidelines for Preparing Economic Analyses (U.S. EPA, 2000)[9] while the Agency continues its efforts to update its guidance on this issue. This approach calculates a mean value across VSL estimates derived from 26 labor market and contingent valuation studies published between 1974 and

[8]After adjusting the VSL to account for a different currency year (2005$) and to account for income growth to 2015, the $5.5 million VSL is $7.2 million.

[9]In the (draft) update of the Economic Guidelines (U.S. EPA, 2006), EPA retained the VSL endorsed by the SAB with the understanding that further updates to the mortality risk valuation guidance would be forthcoming in the near future. Therefore, this report does not represent final agency policy.

1991. The mean VSL across these studies is $6.3 million (2000$).[10] The Agency is committed to using scientifically sound, appropriately reviewed evidence in valuing mortality risk reductions and has made significant progress in responding to the SAB-EEAC's specific recommendations.

6.3 Health Benefits Results

Table 6-2 provides a summary of the monetized $PM_{2.5}$ benefits for the final Portland Cement NESHAP and NSPS using the anchor points of Pope et al. and Laden et al. as well as the results from the expert elicitation on PM mortality at discount rates of 3% and 7%. Table 6-3 provides a summary of the reductions in health incidences as a result of the pollution reductions for the final Portland Cement NESHAP and NSPS. Table 6-4 compares the monetized $PM_{2.5}$ benefits attributable to the final NSPS only, the final NESHAP only, and the more stringent NSPS and final NESHAP. Figure 6-4 illustrates the relative breakdown of the monetized $PM_{2.5}$ health benefits. Figure 6-5 provides a graphical representation of all 14 of the $PM_{2.5}$ benefits, at both a 3 percent and 7% discount rate.

The very large proportion of the avoided PM-related impacts we estimate in this analysis occur among populations exposed at or above the lowest LML of the cohort studies (Figures 6-6 and 6-7), increasing our confidence in the PM mortality analysis. Figure 6-6 shows a bar chart of the percentage of the estimated mortalities at each $PM_{2.5}$ level. Figure 6-7 shows a cumulative distribution function of the same data. Both figures identify the LML for each of the major cohort studies.

Using the Pope et al. (2002) study, approximately 94% of the mortality impacts occur among populations with baseline exposure to annual mean $PM_{2.5}$ levels at or above the LML of 7.5 $\mu g/m^3$. Using the Laden et al. (2006) study, 40% of the mortality impacts occur at or above the LML of 10 $\mu g/m^3$. As we model mortality impacts among populations exposed to levels of $PM_{2.5}$ that are successively lower than the LML of the lowest cohort study, our confidence in the results diminishes. However, the analysis above confirms that the great majority of the impacts occur at or above the lowest cohort study's LML. It is important to emphasize that we have high confidence in $PM_{2.5}$-related effects down to the lowest LML of the major cohort studies.

[10] In this analysis, we adjust the VSL to account for a different currency year (2005$) and to account for income growth to 2015. After applying these adjustments to the $6.3 million value, the VSL is $8.3 million.

Table 6-2. Summary of Monetized Benefits Estimates for Final Cement NESHAP and NSPS in 2013 (millions of 2005$)[a]

	3%	7%
Based on Epidemiology Literature		
Pope et al.	$7,600 ($620--$23,000)	$6,900 ($560--$21,000)
Laden et al.	$19,000 ($1,600--$55,000)	$17,000 ($1,500--$49,000)
Based on Expert Elicitation		
Expert A	$20,000 ($1,100--$65,000)	$18,000 ($1,000--$59,000)
Expert B	$15,000 ($550--$61,000)	$13,000 ($500--$55,000)
Expert C	$15,000 ($870--$57,000)	$14,000 ($790--$52,000)
Expert D	$11,000 ($690--$34,000)	$9,700 ($620--$31,000)
Expert E	$24,000 ($2,100--$73,000)	$22,000 ($1,900--$66,000)
Expert F	$14,000 ($1,300--$41,000)	$12,000 ($1,200--$37,000)
Expert G	$9,000 ($56--$33,000)	$8,200 ($53--$30,000)
Expert H	$11,000 ($75--$44,000)	$10,000 ($71--$40,000)
Expert I	$15,000 ($820--$50,000)	$13,000 ($740--$45,000)
Expert J	$12,000 ($900--$47,000)	$11,000 ($810--$42,000)
Expert K	$2,900 ($56--$19,000)	$2,700 ($53--$17,000)
Expert L	$10,000 ($370--$39,000)	$9,100 ($330--$35,000)

[a] All estimates are for the implementation year (2013), and are rounded to two significant figures so numbers may not sum across columns. All fine particles are assumed to have equivalent health effects. These estimates do not include benefits from reducing HAP emissions, and they do not include the energy disbenefits described in the next section.

Table 6-3. Summary of Reductions in Health Incidences and Monetized Benefits from $PM_{2.5}$ Benefits for the Final Cement NESHAP and NSPS in 2013 (95th percentile confidence interval)[a]

Health Endpoint	Incidence	3% Discount (millions of 2005$)	7% Discount (millions of 2005$)
Avoided Premature Mortality			
Pope et al. (ACS cohort)	960 (320--1,600)	$7,000 ($0,560--$21,000)	$6,300 ($0,500--$19,000)
Laden et al. (H6C cohort)	2,500 (1,200--3,700)	$18,000 ($1,600--$53,000)	$16,000 ($1,400--$47,000)
Woodruff et al. (Infant Mortality)	4 (-4--13)	$35 (-$38--$160)	$35 (-$38--$160)
Avoided Morbidity			
Chronic Bronchitis	650 (70--1,200)	$19 ($1.1--$90)	$19 ($1.10--$90.00)
Acute Myocardial Infarction	1,500 (470--2,600)	$11 ($2.0--$27)	$11 ($1.8--$26)
Hospital Admissions, Respiratory	240 (100--360)	$0.21 ($0.10--$0.31)	$0.21 ($0.10--$0.31)
Hospital Admissions, Cardiovascular	500 (360--590)	$0.90 ($0.47--$1.20)	$0.90 ($0.47--$1.2)
Emergency Room Visits, Respiratory	1,000 (550--1,500)	$0.03 ($0.01--$0.04)	$0.03 ($0.01--$0.04)
Acute Bronchitis	1,500 (-200--3,200)	$0.01 ($0.00--$0.02)	$0.01 ($0.00--$0.02)
Work Loss Days	130,000 (110,000--140,000)	$1.2 ($1.1--$1.4)	$1.2 ($1.1--$1.4)
Asthma Exacerbation	17,000 (1,200--52,000)	$0.06 ($0.00--$0.21)	$0.06 ($0.00--$0.21)
Minor Restricted Activity Days	750,000 (620,000--880,000)	$3.0 ($1.6--$4.6)	$3.0 ($1.6--$4.6)
Lower Respiratory Symptoms	18,000 (7,800--28,000)	$0.02 ($0.01--$0.05)	$0.02 ($0.01--$0.05)
Upper Respiratory Symptoms	14,000 (3,400--24,000)	$0.03 ($0.01--$0.07)	$0.03 ($0.01--$0.07)

[a] All estimates are for the analysis year (2013) and are rounded to whole numbers with two significant figures. All fine particles are assumed to have equivalent health effects. These estimates do not include benefits from reducing HAP emissions, and they do not include the energy disbenefits described in the next section.

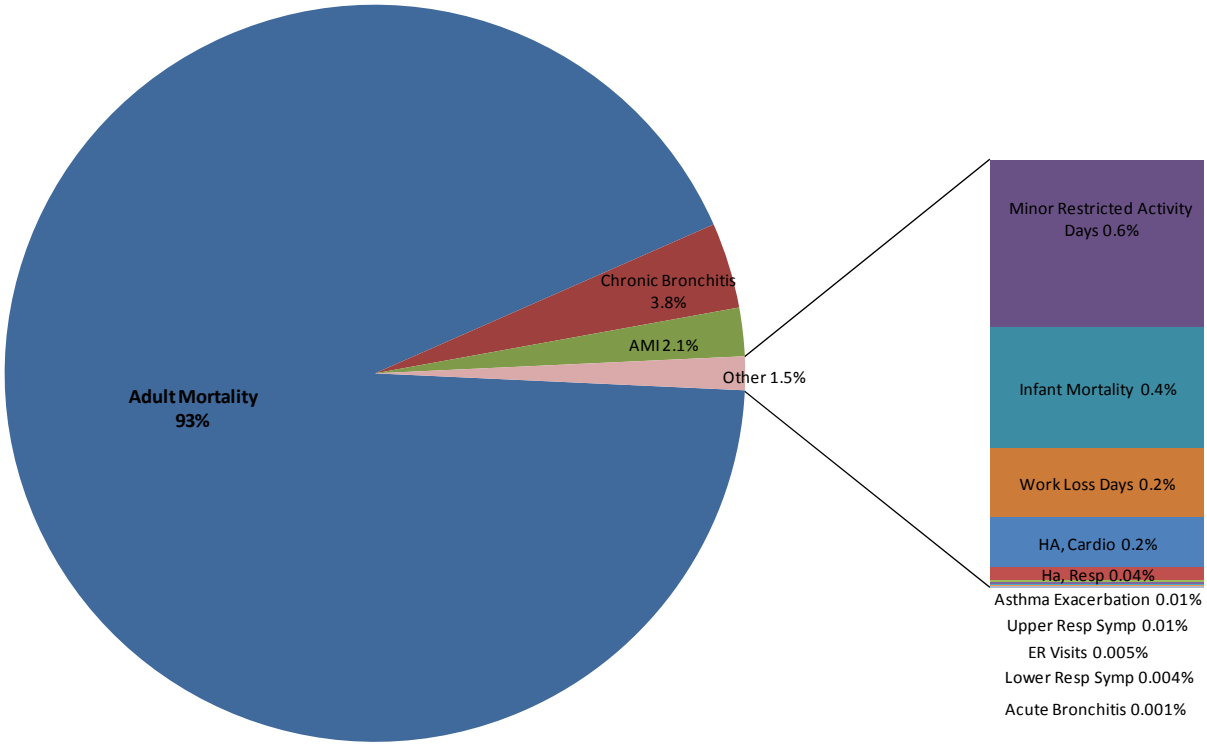

Figure 6-4. Breakdown of Monetized $PM_{2.5}$ Health Benefits using Mortality Function from Pope et al. (2002)[a]

[a] This pie chart breakdown is illustrative, using the results based on Pope et al. (2002) as an example. Using the Laden et al. (2006) function for premature mortality, the percentage of total monetized benefits due to adult mortality would be 97%. This chart shows the breakdown using a 3% discount rate, and the results would be similar if a 7% discount rate was used. The monetized estimates do not include benefits from reducing HAP emissions or NO_x, and they do not include the energy disbenefits described in the next section.

Table 6-4. Comparison of Monetized Benefits and Emission Reductions for Final Cement NESHAP and NSPS in 2013 (2005$)[a]

		Final NESHAP and NSPS	Final NSPS only	Final NESHAP only	Final NESHAP and Stringent NSPS
3%	Pope	$7,600	$510	$7,600	$7,600
	Laden	$19,000	$1,300	$19,000	$19,000
7%	Pope	$6,900	$460	$6,900	$6,900
	Laden	$17,000	$1,100	$17,000	$17,000
Emission Reductions	PM (tpy)	11,000	590	11,000	11,000
	SO_2 (tpy)	124,000	9,000	124,000	124,000
	NO_x (tpy)	6,600	6,600	0	11,000
	HCl (tpy)	5,900	520	5,900	5,900
	Organic HAPs (tpy)	5,200	0	5,200	5,200
	Hg (pounds)	16,400	0	16,400	16,400

[a] All estimates are for the analysis year (2013) and are rounded to whole numbers with two significant figures. All fine particles are assumed to have equivalent health effects. The monetized estimates do not include benefits from reducing HAP emissions or NO_x, and they do not include the energy disbenefits described in the next section.

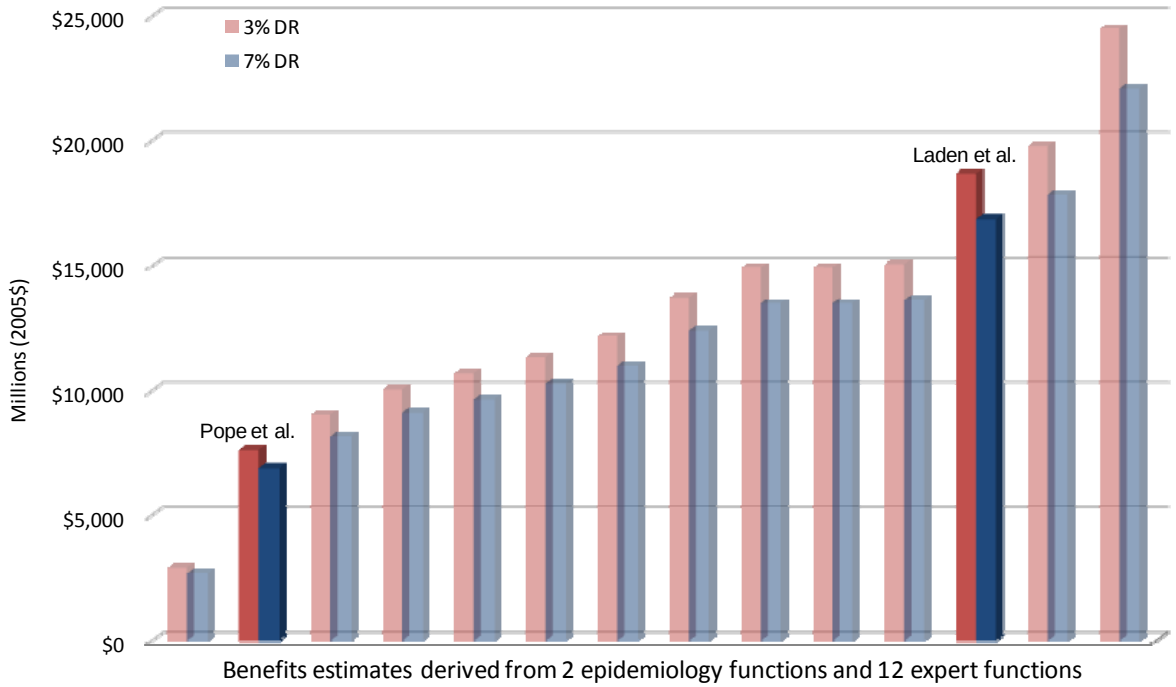

Figure 6-5. Total Monetized $PM_{2.5}$ Benefits for the Final Cement NESHAP and NSPS in 2013[a]

[a] This graph shows the estimated benefits at discount rates of 3% and 7% using effect coefficients derived from the Pope et al. study and the Laden et al study, as well as 12 effect coefficients derived from EPA's expert elicitation on PM mortality. The results shown are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies. These estimates do not include benefits from reducing HAP emissions, and they do not include the energy disbenefits described in the next section.

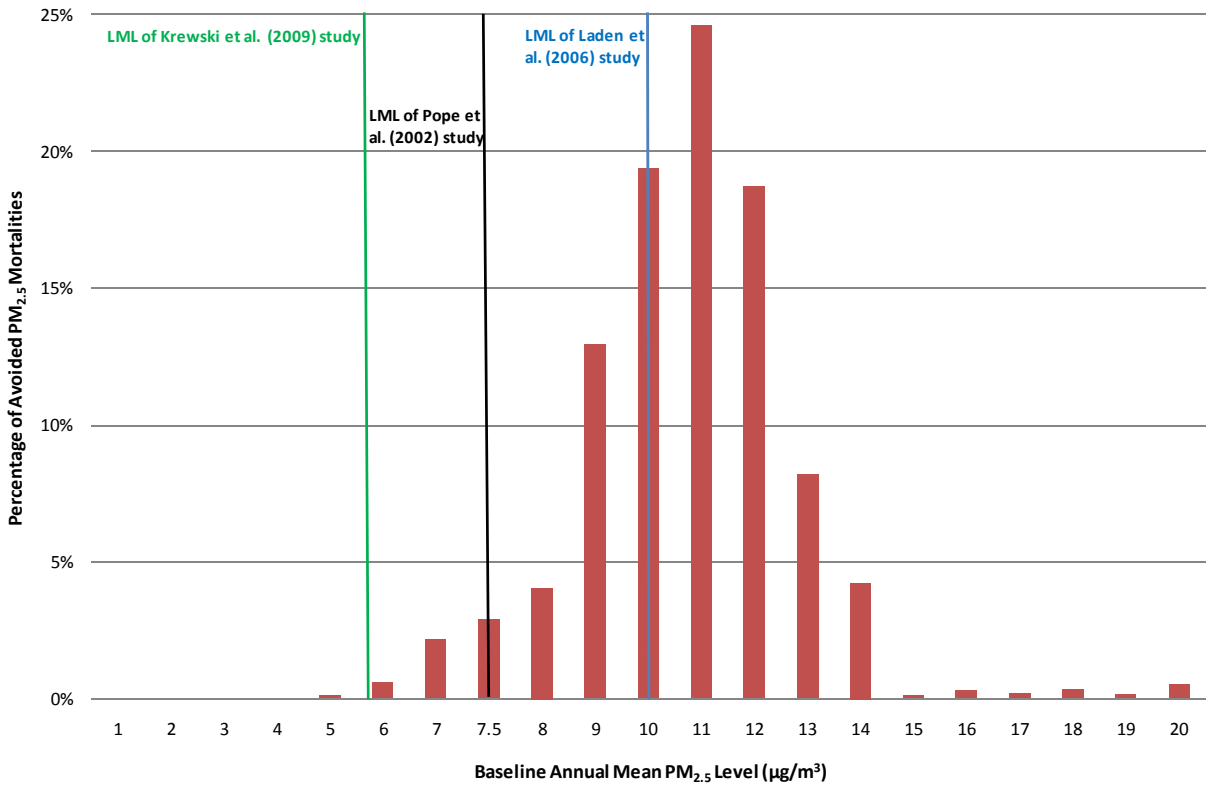

Figure 6-6. Percentage of Total PM-Related Mortalities Avoided by Baseline Air Quality Level for Final Portland Cement NESHAP and NSPS [a]

[a] Approximately 94% of the mortality impacts occur among populations with baseline exposure to annual mean $PM_{2.5}$ levels at or above 7.5 µg/m^3, which is the lowest air quality level considered in the ACS cohort study by Pope et al. (2002).

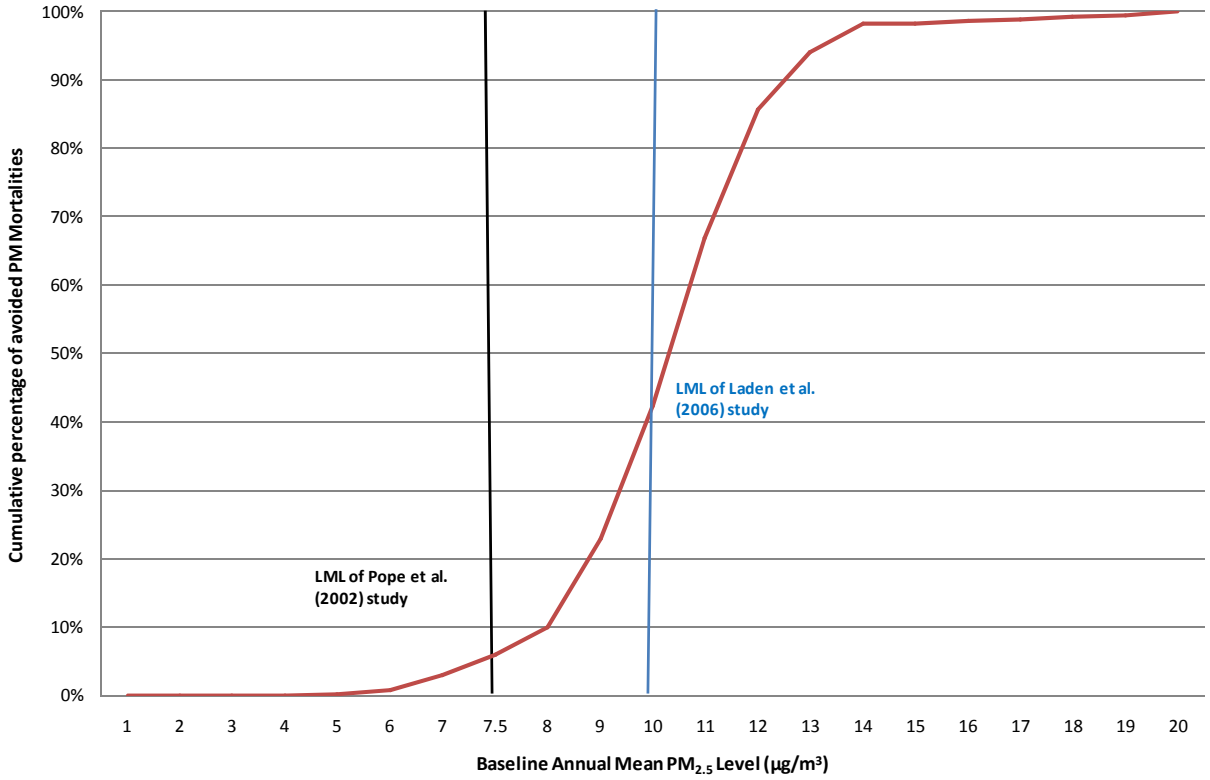

Figure 6-7. Cumulative Percentage of Total PM-related Mortalities Avoided by Baseline Air Quality Level for Final Portland Cement NESHAP and NSPS [a]

[a] Approximately 94% of the mortality impacts occur among populations with baseline exposure to annual mean $PM_{2.5}$ levels at or above 7.5 µg/m^3, which is the lowest air quality level considered in the ACS cohort study by Pope et al. (2002).

6.4 Energy Disbenefits

Electricity usage associated with the operation of control devices is anticipated to increase emissions of criteria pollutants from utility boilers that supply electricity to the Portland cement facilities. We estimate increased energy demand associated with the installation of scrubbers, ACI systems, and RTO. The increases for kilns subject to existing source standards are estimated to be 2,000 tpy of NO_x, 1,000 tpy of CO, 3,500 tpy of SO_2 and about 100 tpy of PM. For kilns subject to new source standards increases in secondary air pollutants are estimated to be 200 tpy of NO_X, 100 tpy of CO, 400 tpy of SO_2 and 10 tpy of PM. We also estimated increases of CO_2 to be 1.1 million tpy for kilns subject to existing source standards and 4,000 tpy for kilns subject to new source standards. The increase in electricity usage for the pumps used in the SNCR system to deliver reagent to the kiln is negligible.

6.4.1 $PM_{2.5}$ Disbenefits

The additional energy usage required for the emission control devices would increase emissions of PM, NOx, SO_2. Because NOx and SO_2 are also precursors to $PM_{2.5,}$ increasing these emissions would also increase $PM_{2.5}$ formation, human exposure, and the incidence of $PM_{2.5}$-related health effects. Due to time and resource limitations, it was not possible to provide a comprehensive estimate of the $PM_{2.5}$-related disbenefits using air quality modeling. Instead, we used the "benefit-per-ton" approach to estimate these disbenefits based on the methodology described in Fann, Fulcher, and Hubbell (2009). These $PM_{2.5}$ benefit-per-ton estimates provide the total monetized human health benefits (the sum of premature mortality and premature morbidity) of reducing one ton of $PM_{2.5}$ from a specified source. EPA has used the benefit per-ton technique in several previous RIAs, including the proposal for this rule (U.S. EPA, 2009a). For this analysis, we use the benefit-per-ton estimates associated with the EGU sector. It is important to note that the disbenefits associated with directly emitted PM are overestimated in this analysis because we assume that all of the increased PM tons are in the $PM_{2.5}$ fraction. Table 6-5 summarizes the benefit-per-ton estimates and the monetized $PM_{2.5}$ disbenefits at discount rates of 3% and 7%.

Table 6-5. Summary of Monetized $PM_{2.5}$ Energy Disbenefits for the Final Portland Cement NSPS and NESHAP in 2013 (2005$)

Pollutant	Emissions Reductions (tons)	Benefit per ton (Pope, 3%)	Benefit per ton (Laden, 3%)	Benefit per ton (Pope, 7%)	Benefit per ton (Laden, 7%)	Monetized $PM_{2.5}$ Disbenefits (millions, 3%)	Monetized $PM_{2.5}$ Disbenefits (millions, 7%)
Direct $PM_{2.5}$	110	$210,000	$510,000	$190,000	$460,000	$23 to $56	$21 to $50
$PM_{2.5}$ Precursors							
SO_2	3,900	$37,000	$91,000	$34,000	$82,000	$150 to $360	$130 to $320
NO_X	2,200	$6,800	$17,000	$6,100	$15,000	$15 to $36	$13 to $33
					Total	**$180 to $450**	**$170 to $400**

[a] All estimates are for the implementation year (2013), and are rounded to two significant figures so numbers may not sum across columns. All fine particles are assumed to have equivalent health effects, but the benefit per ton estimates vary because each ton of precursor reduced has a different propensity to become $PM_{2.5}$. The monetized disbenefits incorporate the conversion from precursor emissions to ambient fine particles. Confidence intervals are unavailable for this analysis because of the benefit-per-ton methodology. The disbenefits associated with directly emitted PM are overestimated in this analysis because we assume that all of the increased PM tons are in the $PM_{2.5}$ fraction.

6.4.2 Social Cost of Carbon and Greenhouse Gas Disbenefits

EPA has assigned a dollar value to reductions in carbon dioxide (CO_2) emissions using recent estimates of the "social cost of carbon" (SCC). The SCC is an estimate of the monetized damages associated with an incremental increase in carbon emissions in a given year. It is intended to include (but is not limited to) changes in net agricultural productivity, human health, property damages from increased flood risk, and the value of ecosystem services due to climate change. The SCC estimates used in this analysis were developed through an interagency process that included EPA and other executive branch entities, and concluded in February 2010. EPA first used these SCC estimates in the benefits analysis for the final joint EPA/DOT Rulemaking to establish Light-Duty Vehicle Greenhouse Gas Emission Standards and Corporate Average Fuel Economy Standards; see the rule's preamble for discussion about application of SCC (75 FR 25324; 5/7/10). The SCC Technical Support Document (SCC TSD) provides a complete discussion of the methods used to develop these SCC estimates.[11]

The interagency group selected four SCC values for use in regulatory analyses, which we have applied in this analysis: $5, $21, $35, and $65 per metric ton of CO_2 emissions[12] in 2010, in 2007 dollars. The first three values are based on the average SCC from three integrated assessment models, at discount rates of 2.5, 3, and 5 percent, respectively. SCCs at several discount rates are included because the literature shows that the SCC is quite sensitive to assumptions about the discount rate, and because no consensus exists on the appropriate rate to use in an intergenerational context. The fourth value is the 95th percentile of the SCC from all three models at a 3 percent discount rate. It is included to represent higher-than-expected impacts from temperature change further out in the tails of the SCC distribution. Low probability, high impact events are incorporated into all of the SCC values through explicit consideration of their effects in two of the three models as well as the use of a probability density function for equilibrium climate sensitivity. Treating climate sensitivity probabilistically results in more high temperature outcomes, which in turn lead to higher projections of damages.

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[11] Docket ID EPA-HQ-OAR-2009-0472-114577, *Technical Support Document: Social Cost of Carbon for Regulatory Impact Analysis Under Executive Order 12866,* Interagency Working Group on Social Cost of Carbon, with participation by Council of Economic Advisers, Council on Environmental Quality, Department of Agriculture, Department of Commerce, Department of Energy, Department of Transportation, Environmental Protection Agency, National Economic Council, Office of Energy and Climate Change, Office of Management and Budget, Office of Science and Technology Policy, and Department of Treasury (February 2010). Also available at http://www.epa.gov/otaq/climate/regulations.htm

[12] The interagency group decided that these estimates apply only to CO_2 emissions. Given that warming profiles and impacts other than temperature change (e.g. ocean acidification) vary across GHGs, the group concluded "transforming gases into CO_2-equivalents using GWP, and then multiplying the carbon-equivalents by the SCC, would not result in accurate estimates of the social costs of non-CO_2 gases" (SCC TSD, pg 13).

The SCC increases over time because future emissions are expected to produce larger incremental damages as physical and economic systems become more stressed in response to greater climatic change. Note that the interagency group estimated the growth rate of the SCC directly using the three integrated assessment models rather than assuming a constant annual growth rate. This helps to ensure that the estimates are internally consistent with other modeling assumptions. The SCC estimates for the analysis years of 2013, in 2005 dollars are provided in Table 6-6.

When attempting to assess the incremental economic impacts of carbon dioxide emissions, the analyst faces a number of serious challenges. A recent report from the National Academies of Science (NRC, 2008) points out that any assessment will suffer from uncertainty, speculation, and lack of information about (1) future emissions of greenhouse gases, (2) the effects of past and future emissions on the climate system, (3) the impact of changes in climate on the physical and biological environment, and (4) the translation of these environmental impacts into economic damages. As a result, any effort to quantify and monetize the harms associated with climate change will raise serious questions of science, economics, and ethics and should be viewed as provisional.

The interagency group noted a number of limitations to the SCC analysis, including the incomplete way in which the integrated assessment models capture catastrophic and non-catastrophic impacts, their incomplete treatment of adaptation and technological change, uncertainty in the extrapolation of damages to high temperatures, and assumptions regarding risk aversion. The limited amount of research linking climate impacts to economic damages makes the interagency modeling exercise even more difficult. The interagency group hopes that over time researchers and modelers will work to fill these gaps and that the SCC estimates used for regulatory analysis by the Federal government will continue to evolve with improvements in modeling. Additional details on these limitations are discussed in the SCC TSD.

In light of these limitations, the interagency group has committed to updating the current estimates as the science and economic understanding of climate change and its impacts on society improves over time. Specifically, the interagency group has set a preliminary goal of revisiting the SCC values within two years or at such time as substantially updated models become available, and to continue to support research in this area.

Applying the global SCC estimates to the estimated increases in CO_2 emissions for the range of policy scenarios, we estimate the dollar value of the climate-related disbenefits captured by the models for each analysis year. For internal consistency, the annual disbenefits are

discounted back to NPV terms using the same discount rate as each SCC estimate (i.e. 5%, 3%, and 2.5%) rather than 3% and 7%.[13] These estimates are provided in Table 6-7.

Table 6-6. Social Cost of Carbon (SCC) Estimates (per tonne of CO_2) for 2013 [a]

Discount Rate and Statistic	SCC estimate (2005$)
5% Average	$5.0
3% Average	$21.5
2.5% Average	$34.9
3% 95%ile	$65.6

[a] The SCC values are dollar-year and emissions-year specific. SCC values represent only a partial accounting of climate impacts.

Table 6-7. Monetized Disbenefits of CO_2 Emission Increases in 2013 [a]

Discount Rate and Statistic	SCC-derived disbenefits (millions of 2005$)
5% Average	$5.1
3% Average	$22
2.5% Average	$36
3% 95%ile	$67

[a] The SCC values are dollar-year and emissions-year specific. SCC values represent only a partial accounting of climate impacts.

6.4.3 Total Monetized Disbenefits

The additional energy usage required for the emission control devices would increase emissions of several pollutants. In this analysis, we were able to monetize the disbenefits associated with the increased emissions of PM, NO_X, SO_2, and CO_2, but we were unable to monetize the disbenefits associated with the increased emissions of CO. We estimate that the total monetized disbenefits at a 3% discount rate are $210 to $470 million. Therefore, these disbenefits reduce the total monetized benefits to $7.4 billion to $18 billion and $6.7 billion to $17 billion, at discount rates of 3% and 7% respectively.

In addition, we were unable to quantify the emission increases or monetize the disbenefits associated with "leakage" of emissions to other counties. This benefits analysis only incorporates the domestic emission changes, but this regulation could lead to increased imports

[13] It is possible that other benefits or costs of proposed regulations unrelated to CO_2 emissions will be discounted at rates that differ from those used to develop the SCC estimates.

and production in other countries. For this analysis, because we do not have sufficient information on origin of these imports, the specific location of the additional emissions, or the level of control on those facilities, we are unable to estimate the potential disbenefits associated with increased emissions in other countries that might occur as a result of this regulation. However, the monetized benefits estimates do not account for the decrease in domestic emissions associated with the decrease in domestic production and transportation. The economic analysis estimates that domestic production would decrease by 10 million tons, but imports would increase by only 3 million tons. The net effect on global pollutants like CO_2 and mercury is difficult to determine because it depends on many factors, and quantifying the benefits associated with either omission is beyond the scope of this analysis.

6.5 Unquantified or Nonmonetized Benefits

The monetized benefits estimated in this RIA only reflect the portion of benefits attributable to the health impacts associated with exposure to ambient fine particles. Data, resource, and methodological limitations prevented EPA from quantifying or monetizing the benefits from several important benefit categories, including benefits from reducing toxic emissions, ecosystem effects, and visibility impairment. The health benefits from reducing hazardous air pollutants (HAPs) have not been monetized in this analysis. In addition to being a $PM_{2.5}$ precursor, SO_2 emissions also contribute to adverse effects from acidic deposition in aquatic and terrestrial ecosystems, increase mercury methylation, as well as visibility impairment.

6.5.1 Other SO_2 and PM Benefits

In addition to being a precursor to $PM_{2.5}$, SO_2 emissions are also associated with a variety of respiratory health effects. Unfortunately, we were unable to estimate the health benefits associated with reduced SO_2 exposure in this analysis because we do not have air quality modeling data available. Without knowing the location of the emission reductions and the resulting ambient concentrations, we were unable to estimate the exposure to SO_2 for nearby populations. Therefore, this analysis only quantifies and monetizes the $PM_{2.5}$ benefits associated with the reductions in SO_2 emissions.

Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the *Integrated Science Assessment (ISA) for Sulfur Dioxide* concluded that there is a causal relationship between respiratory health effects and short-term exposure to SO_2 (U.S. EPA, 2008b). According to summary of the ISA in EPA's risk and exposure assessment (REA) for the SO_2 NAAQS"the immediate effect of SO_2 on the respiratory system in humans is

bronchoconstriction" (U.S. EPA, 2009c). In addition, the REA summarized from the ISA that "asthmatics are more sensitive to the effects of SO_2 likely resulting from preexisting inflammation associated with this disease." A clear concentration-response relationship has been demonstrated in laboratory studies following exposures to SO_2 at concentrations between 20 and 100 ppb, both in terms of increasing severity of effect and percentage of asthmatics adversely affected (U.S. EPA, 2009c). Based on our review of this information, we identified four short-term morbidity endpoints that the SO_2 ISA identified as a "causal relationship": asthma exacerbation, respiratory-related emergency department visits, and respiratory-related hospitalizations. The differing evidence and associated strength of the evidence for these different effects is described in detail in the SO_2 ISA. The SO_2 ISA also concluded that the relationship between short-term SO_2 exposure and premature mortality was "suggestive of a causal relationship" because it is difficult to attribute the mortality risk effects to SO_2 alone. Although the SO_2 ISA stated that studies are generally consistent in reporting a relationship between SO_2 exposure and mortality, there was a lack of robustness of the observed associations to adjustment for pollutants.

SO_2 emissions also contribute to adverse welfare effects from acidic deposition, visibility impairment, and enhanced mercury methylation. Deposition of sulfur causes acidification, which can cause a loss of biodiversity of fishes, zooplankton, and macro invertebrates in aquatic ecosystems, as well as a decline in sensitive tree species, such as red spruce (*Picea rubens*) and sugar maple (*Acer saccharum*) in terrestrial ecosystems. In the northeastern United States, the surface waters affected by acidification are a source of food for some recreational and subsistence fishermen and for other consumers and support several cultural services, including aesthetic and educational services and recreational fishing. Biological effects of acidification in terrestrial ecosystems are generally linked to aluminum toxicity, which can cause reduced root growth, which restricts the ability of the plant to take up water and nutrients. These direct effects can, in turn, increase the sensitivity of these plants to stresses, such as droughts, cold temperatures, insect pests, and disease leading to increased mortality of canopy trees. Terrestrial acidification affects several important ecological services, including declines in habitat for threatened and endangered species (cultural), declines in forest aesthetics (cultural), declines in forest productivity (provisioning), and increases in forest soil erosion and reductions in water retention (cultural and regulating). (U.S. EPA, 2008c)

Reducing SO_2 and PM emissions would improve the level of visibility throughout the United States. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon, and soil (Sisler, 1996). These suspended particles and

gases degrade visibility by scattering and absorbing light. Higher visibility impairment levels in the East are due to generally higher concentrations of fine particles, particularly sulfates, and higher average relative humidity levels. In fact, particulate sulfate is the largest contributor to regional haze in the eastern U.S. (i.e., 40% or more annually and 75% during summer). In the western U.S., particulate sulfate contributes to 20-50% of regional haze (U.S. EPA, 2009b). Visibility has direct significance to people's enjoyment of daily activities and their overall sense of wellbeing. Good visibility increases the quality of life where individuals live and work, and where they engage in recreational activities. Due to time and resource limitations, we were unable to estimate the monetized benefits associated with visibility improvements. Previous analyses (U.S. EPA, 2006; U.S. EPA, 2010c) show that visibility benefits are a significant welfare benefit category.

6.5.2 HAP Benefits

Americans are exposed to ambient concentrations of air toxics at levels which have the potential to cause adverse health effects.[14] The levels of air toxics to which people are exposed vary depending on where people live and work and the kinds of activities in which they engage. In order to identify and prioritize air toxics, emission source types and locations which are of greatest potential concern, U.S. EPA conducts the National-Scale Air Toxics Assessment (NATA). The most recent NATA was conducted for calendar year 2002, and was released in June 2009.[15] NATA for 2002 includes four steps:

1) Compiling a national emissions inventory of air toxics emissions from outdoor sources
2) Estimating ambient concentrations of air toxics across the United States
3) Estimating population exposures across the United States
4) Characterizing potential public health risk due to inhalation of air toxics including both cancer and noncancer effects

Noncancer health effects can result from chronic,[16] subchronic,[17] or acute[18] inhalation exposures to air toxics, and include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems. According to the 2002

[14] U.S. EPA. (2009) 2002 National-Scale Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata2002/
[15] U.S. EPA. (2009) 2002 National-Scale Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata2002/
[16] Chronic exposure is defined in the glossary of the Integrated Risk Information (IRIS) database (http://www.epa.gov/iris) as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).
[17] Defined in the IRIS database as exposure to a substance spanning approximately 10% of the lifetime of an organism.
[18] Defined in the IRIS database as exposure by the oral, dermal, or inhalation route for 24 hours or less.

NATA, nearly the entire U.S. population was exposed to an average concentration of air toxics that has the potential for adverse noncancer respiratory health effects.[19] Figures 6-8 and 6-9 depict estimated county-level carcinogenic risk and noncancer respiratory hazard from the assessment. The respiratory hazard is dominated by a single pollutant, acrolein.

This rule is anticipated to reduce 16,400 pounds of mercury, 5,800 tons of HCl, and 5,200 tons of organic HAPs each year. Due to data, resource, and methodology limitations, we were unable to estimate the benefits associated with the thousands tons of hazardous air pollutants that would be reduced as a result of this rule. Available emissions data show that several different HAPs are emitted from Portland cement manufacturing plants, either released from kilns systems, raw material dryers, clinker coolers, raw mills, finish mills, storage bins, conveying system transfer points, bagging systems, or bulk loading and unloading systems.

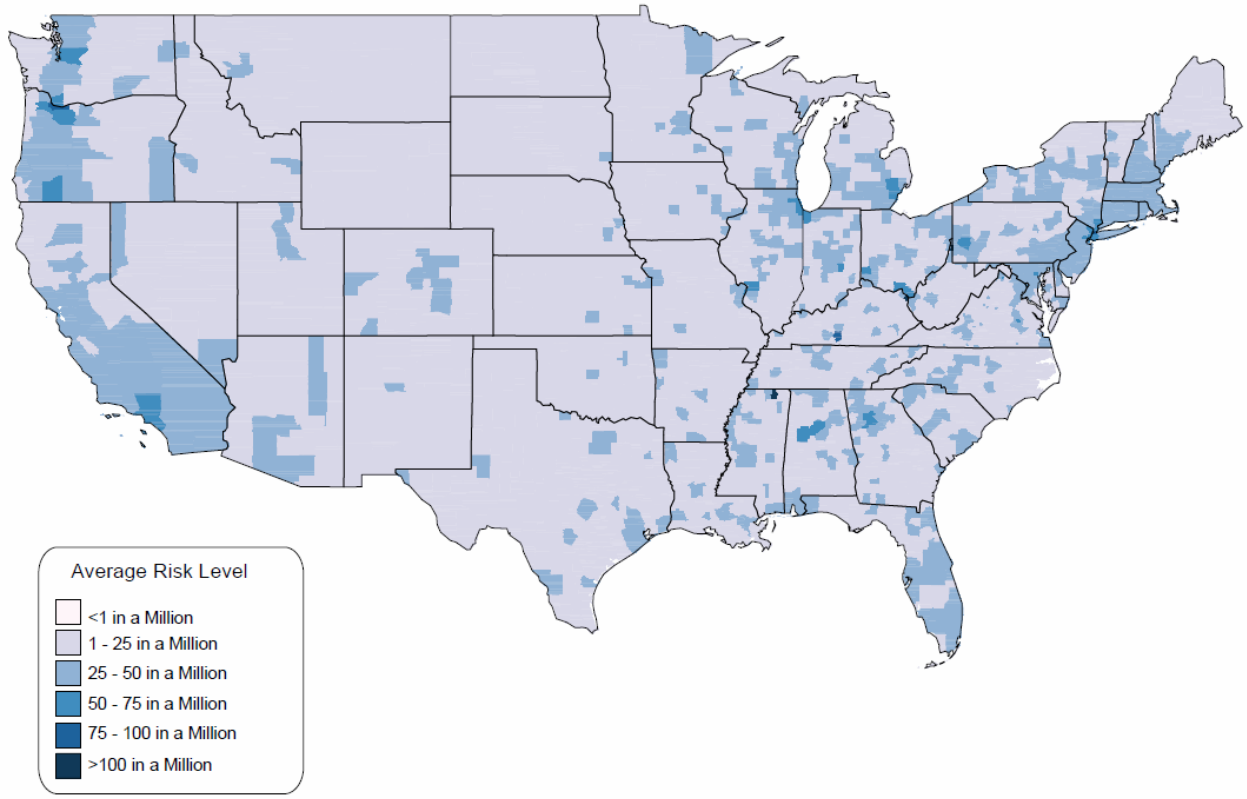

Figure 6-8. Estimated County Level Carcinogenic Risk from HAP exposure from outdoor sources (NATA, 2002)

[19] The NATA modeling framework has a number of limitations which prevent its use as the sole basis for setting regulatory standards. These limitations and uncertainties are discussed on the 2002 NATA website. Even so, this modeling framework is very useful in identifying air toxic pollutants and sources of greatest concern, setting regulatory priorities, and informing the decision making process. U.S. EPA. (2009) 2002 National-Scale Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata2002/

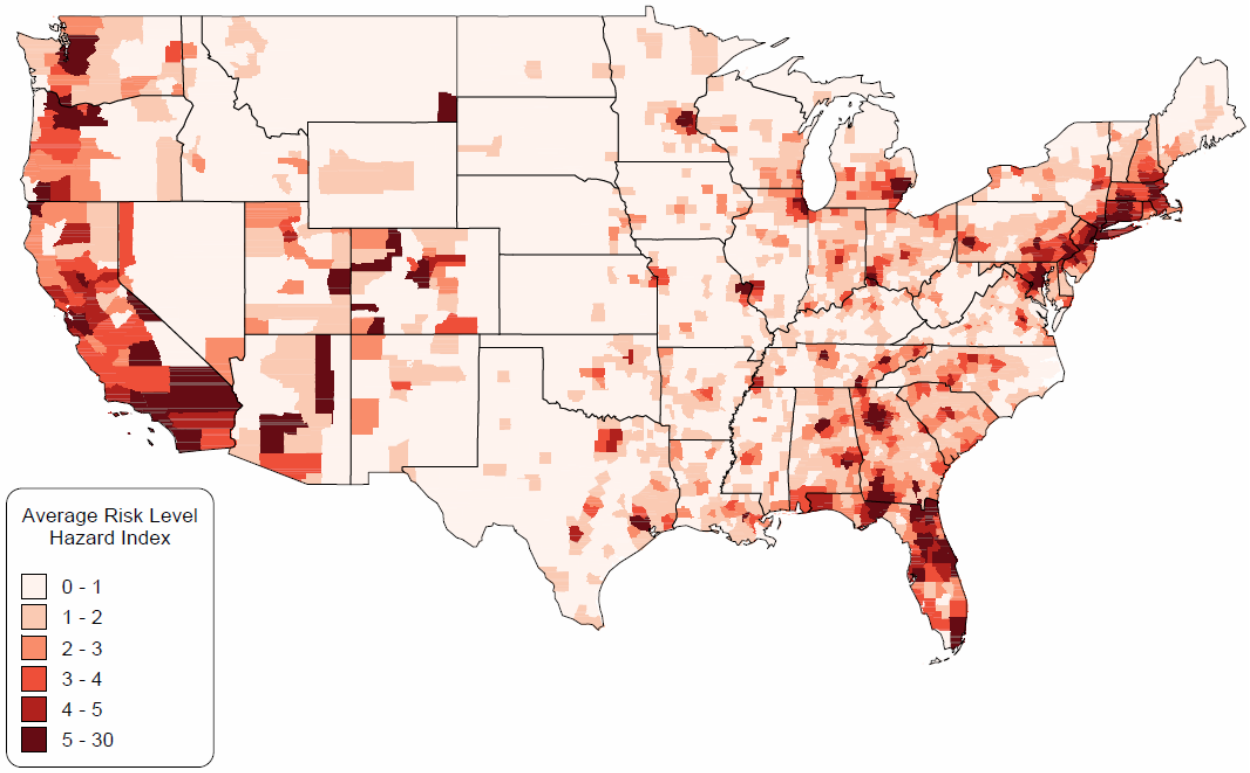

Figure 6-9. Estimated County Level Noncancer (Respiratory) Risk from HAP exposure from outdoor sources (NATA, 2002)

6.5.2.1 Mercury

Mercury is a highly neurotoxic contaminant that enters the food web as a methylated compound, methylmercury (U.S. EPA, 2008c). The contaminant is concentrated in higher trophic levels, including fish eaten by humans. Experimental evidence has established that only inconsequential amounts of methylmercury can be produced in the absence of sulfate (U.S. EPA, 2008c). Current evidence indicates that in watersheds where mercury is present, increased sulfate deposition very likely results in methylmercury accumulation in fish (Drevnick et al., 2007; Munthe et al, 2007). The SO_2 ISA concluded that evidence is sufficient to infer a casual relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments (U.S. EPA, 2008c).

In addition to the role of sulfate deposition on methylation, these rules would also reduce mercury emissions. Mercury is emitted to the air from various man-made and natural sources. These emissions transport through the atmosphere and eventually deposit to land or water bodies. This deposition can occur locally, regionally, or globally, depending on the form of mercury emitted and other factors such as the weather. The form of mercury emitted varies depending on

the source type and other factors. Available data indicate that the mercury emissions from these sources are a mixture of gaseous elemental mercury (35%), inorganic divalent mercury (reactive gas phase mercury) (58%), and particulate bound mercury (7%) (U.S. EPA, 2010c). Gaseous elemental mercury can be transported very long distances, even globally, to regions far from the emissions source (becoming part of the global "pool") before deposition occurs. Inorganic divalent and particulate bound mercury have a shorter atmospheric lifetime and can deposit to land or water bodies closer to the emissions source. Furthermore, elemental mercury in the atmosphere can undergo transformation into divalent mercury, providing a significant pathway for deposition of emitted elemental mercury.

Potential exposure routes to mercury emissions include both direct inhalation and consumption of fish containing methylmercury. The primary route of human exposure to mercury emissions from industrial sources is generally indirectly through the consumption of fish containing methylmercury. As described above, mercury that has been emitted to the air eventually settles into water bodies or onto land where it can either move directly or be leached into waterbodies. Once deposited, certain microorganisms can change it into methylmercury, a highly toxic form that builds up in fish, shellfish and animals that eat fish. Consumption of fish and shellfish are the main sources of methylmercury exposure to humans. Methylmercury builds up more in some types of fish and shellfish than in others. The levels of methylmercury in fish and shellfish vary widely depending on what they eat, how long they live, and how high they are in the food chain. Most fish, including ocean species and local freshwater fish, contain some methylmercury. For example, in recent studies by EPA and the U.S. Geological Survey (USGS) of fish tissues, every fish sampled contained some methylmercury (Scudder, 2009).

The majority of fish consumed in the U.S. are ocean species. The methylmercury concentrations in ocean fish species are primarily influenced by the global mercury pool. However, the methylmercury found in local fish can be due, at least partly, to mercury emissions from local sources. Research shows that most people's fish consumption does not cause a mercury-related health concern. However, certain people may be at higher risk because of their routinely high consumption of fish (e.g., tribal and other subsistence fishers and their families who rely heavily on fish for a substantial part of their diet). It has been demonstrated that high levels of methylmercury in the bloodstream of unborn babies and young children may harm the developing nervous system, making the child less able to think and learn. Moreover, mercury exposure at high levels can harm the brain, heart, kidneys, lungs, and immune system of people of all ages.

Several studies suggest that the methylmercury content of fish may reduce these cardio-protective effects of fish consumption. Some of these studies also suggest that methylmercury may cause adverse effects to the cardiovascular system. For example, the NRC (2000) review of the literature concerning methylmercury health effects took note of two epidemiological studies that found an association between dietary exposure to methylmercury and adverse cardiovascular effects.[20] Moreover, in a study of 1,833 males in Finland aged 42 to 60 years, Solonen et al. (1995) observed a relationship between methylmercury exposure via fish consumption and acute myocardial infarction (AMI or heart attacks), coronary heart disease, cardiovascular disease, and all-cause mortality.[21] The NRC also noted a study of 917 seven year old children in the Faroe Islands, whose initial exposure to methylmercury was *in utero* although post natal exposures may have occurred as well. At seven years of age, these children exhibited an increase in blood pressure and a decrease in heart rate variability.[22] Based on these and other studies, NRC concluded in 2000 that, while "the data base is not as extensive for cardiovascular effects as it is for other end points (i.e. neurologic effects) the cardiovascular system appears to be a target for methylmercury toxicity."[23]

Since publication of the NRC report there have been some 30 published papers presenting the findings of studies that have examined the possible cardiovascular effects of methylmercury exposure. These studies include epidemiological, toxicological, and toxicokinetic investigations. Over a dozen review papers have also been published. If there is a causal relationship between methylmercury exposure and adverse cardiovascular effects, then reducing exposure to methylmercury would result in public health benefits from reduced cardiovascular effects.

In early 2010, EPA sponsored a workshop in which a group of experts were asked to assess the plausibility of a causal relationship between methylmercury exposure and cardiovascular health effects and to advise EPA on methodologies for estimating population

[20] National Research Council (NRC). 2000. Toxicological Effects of Methylmercury. Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology. National Academies Press. Washington, DC. pp.168-173.

[21] Salonen, J.T., Seppanen, K. Nyyssonen et al. 1995. "Intake of mercury from fish lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular and any death in Eastern Finnish men." Circulation, 91 (3):645-655.

[22] Sorensen, N, K. Murata, E. Budtz-Jorgensen, P. Weihe, and Grandjean, P., 1999. "Prenatal Methylmercury Exposure As A Cardiovascular Risk Factor At Seven Years of Age", Epidemiology, pp370-375.

[23] National Research Council (NRC). 2000. Toxicological Effects of Methylmercury. Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology. National Academies Press. Washington, DC. p. 229.

level cardiovascular health impacts of reduced methylmercury exposure. The report from that workshop is in preparation.

Portland cement manufacturing plants emitted about 16 tons of mercury in the air in 2006 in the U.S. Based on the EPA's National Emission Inventory, and about 103 tons of mercury were emitted from all anthropogenic sources in the U.S. in 2005. Moreover, the United Nations has estimated that about 2,100 tons of mercury were emitted worldwide by anthropogenic sources in 2005. We believe that total mercury emissions in the U.S. and globally in 2006 were about the same magnitude in 2005. Therefore, we estimate that in 2006, these sources emitted about 16% of the total anthropogenic mercury emissions in the U.S. and about 0.8% of the global emissions in 2005.

Using 2008 inventory estimates, the mercury emissions from Portland cement kilns only were approximately 9.1 tons. Overall, the NESHAP and NSPS would reduce mercury emissions by about 8.2 tons (90%) per year from current levels, and therefore, contribute to reductions in mercury exposures and health effects. Due to time and resource limitations, we were unable to model mercury methylation, bioaccumulation in fish tissue, and human consumption of mercury-contaminated fish that would be needed in order to estimate the human health benefits from reducing mercury emissions. However, we were able to model the change in mercury deposition using CAMx for the final Portland Cement NESHAP and NSPS.[24] These modeling results indicate significantly reduced total mercury deposition (wet and dry forms), including reducing deposition by up to 30% in the West and up to 17% in the East in 2013. This modeling indicates that mercury deposition reductions tend to be greatest nearest the sources. Figure 6-10 shows the change in mercury deposition as a result of the final Portland Cement NESHAP and NSPS in the Eastern U.S., and Figure 6.11 shows the change in mercury deposition in the Western U.S.

[24] See Section 5 of this RIA for more information on the air quality modeling.

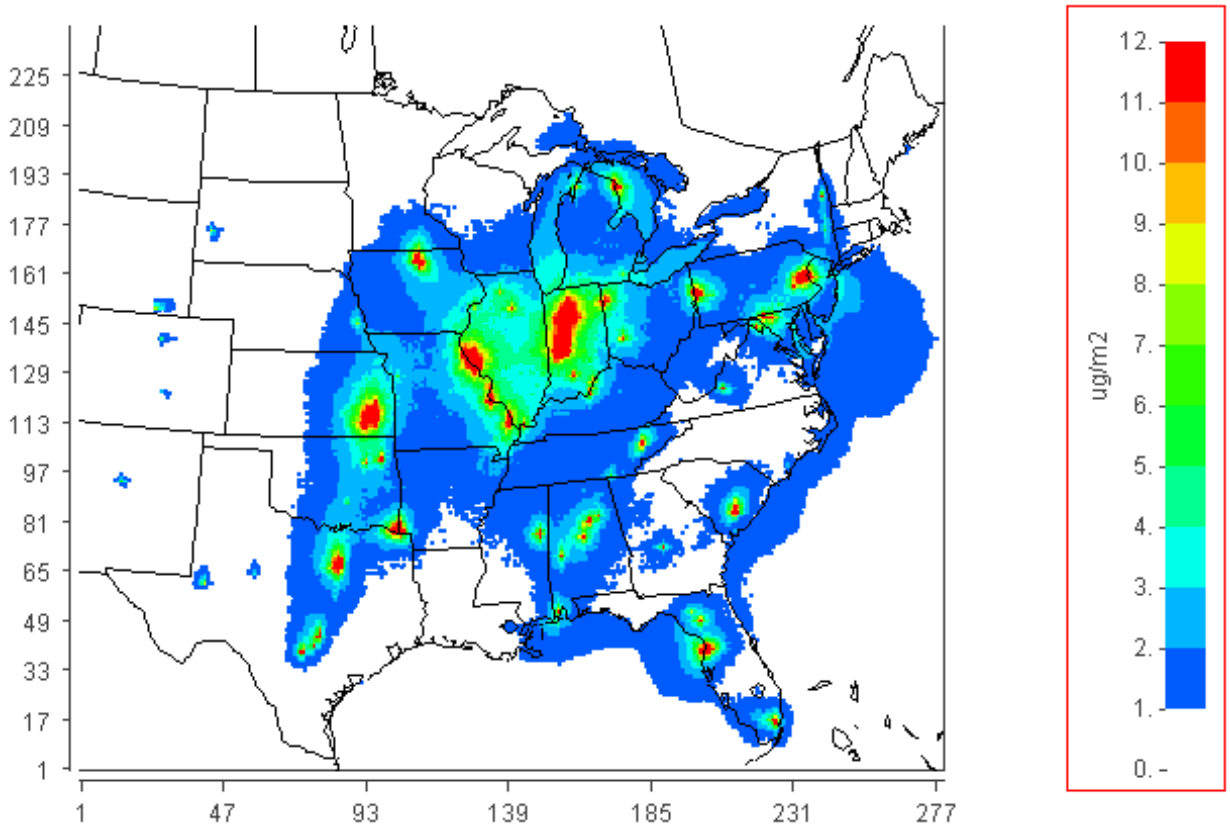

Figure 6-10. Reductions in Total Mercury Deposition (µg/m^2) in the Eastern U.S.

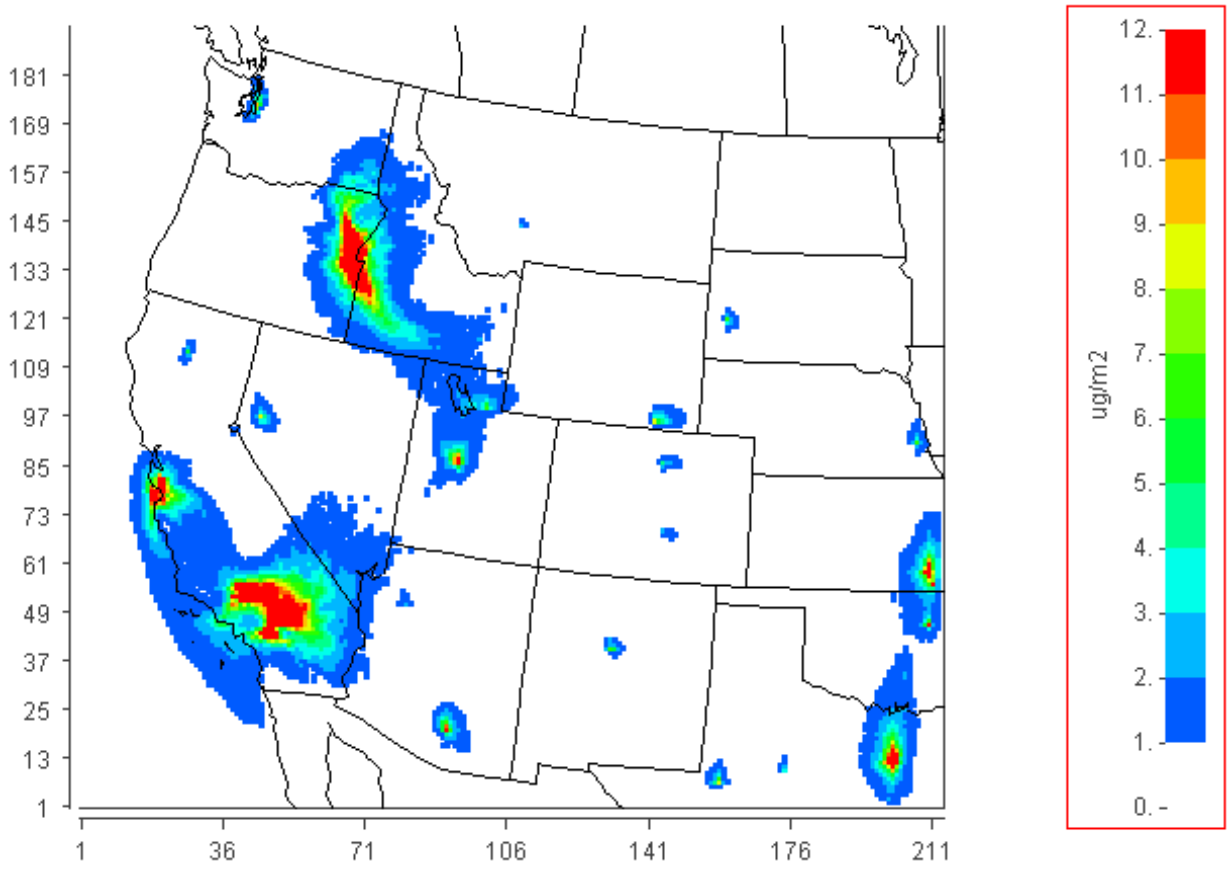

Figure 6-11. Reductions in Total Mercury Deposition (µg/m^2) in the Western U.S.

6.5.2.2 Hydrogen Chloride (HCl)[25]

Hydrogen chloride gas is intensely irritating to the mucous membranes of the nose, throat, and respiratory tract. Brief exposure to 35 ppm causes throat irritation, and levels of 50 to 100 ppm are barely tolerable for 1 hour. The greatest impact is on the upper respiratory tract; exposure to high concentrations can rapidly lead to swelling and spasm of the throat and suffocation. Most seriously exposed persons have immediate onset of rapid breathing, blue coloring of the skin, and narrowing of the bronchioles. Patients who have massive exposures may develop an accumulation of fluid in the lungs. Exposure to hydrogen chloride can lead to Reactive Airway Dysfunction Syndrome (RADS), a chemically- or irritant-induced type of asthma. Children may be more vulnerable to corrosive agents than adults because of the relatively smaller diameter of their airways. Children may also be more vulnerable to gas

[25] All health effects language for this section came from: Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Hydrogen Chloride (HCl). CAS#: 7647-01-0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at <http://www.atsdr.cdc.gov/Mhmi/mmg173.html>.

exposure because of increased minute ventilation per kg and failure to evacuate an area promptly when exposed. Hydrogen chloride has not been classified for carcinogenic effects.

6.5.2.3 Toluene[26]

Toluene is found in evaporative as well as exhaust emissions from motor vehicles. Under the 2005 Guidelines for Carcinogen Risk Assessment, there is inadequate information to assess the carcinogenic potential of toluene because studies of humans chronically exposed to toluene are inconclusive, toluene was not carcinogenic in adequate inhalation cancer bioassays of rats and mice exposed for life, and increased incidences of mammary cancer and leukemia were reported in a lifetime rat oral bioassay.

The central nervous system (CNS) is the primary target for toluene toxicity in both humans and animals for acute and chronic exposures. CNS dysfunction (which is often reversible) and narcosis have been frequently observed in humans acutely exposed to low or moderate levels of toluene by inhalation; symptoms include fatigue, sleepiness, headaches, and nausea. Central nervous system depression has been reported to occur in chronic abusers exposed to high levels of toluene. Symptoms include ataxia, tremors, cerebral atrophy, nystagmus (involuntary eye movements), and impaired speech, hearing, and vision. Chronic inhalation exposure of humans to toluene also causes irritation of the upper respiratory tract, eye irritation, dizziness, headaches, and difficulty with sleep.

Human studies have also reported developmental effects, such as CNS dysfunction, attention deficits, and minor craniofacial and limb anomalies, in the children of women who abused toluene during pregnancy. A substantial database examining the effects of toluene in subchronic and chronic occupationally exposed humans exists. The weight of evidence from these studies indicates neurological effects (i.e., impaired color vision, impaired hearing, decreased performance in neurobehavioral analysis, changes in motor and sensory nerve conduction velocity, headache, dizziness) as the most sensitive endpoint.

6.5.2.4 Formaldehyde

Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[27] EPA is currently reviewing

[26] All health effects language for this section came from: U.S. EPA. 2005. "Full IRIS Summary for Toluene (CASRN 108-88-3)" Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Available on the Internet at <http://www.epa.gov/iris/subst/0118.htm>.

[27] U.S. EPA. 1987. Assessment of Health Risks to Garment Workers and Certain Home Residents from Exposure to

recently published epidemiological data. For instance, research conducted by the National Cancer Institute (NCI) found an increased risk of nasopharyngeal cancer and lymphohematopoietic malignancies such as leukemia among workers exposed to formaldehyde.[28,29] In an analysis of the lymphohematopoietic cancer mortality from an extended follow-up of these workers, NCI confirmed an association between lymphohematopoietic cancer risk and peak exposures.[30] A recent National Institute of Occupational Safety and Health (NIOSH) study of garment workers also found increased risk of death due to leukemia among workers exposed to formaldehyde.[31] Extended follow-up of a cohort of British chemical workers did not find evidence of an increase in nasopharyngeal or lymphohematopoietic cancers, but a continuing statistically significant excess in lung cancers was reported.[32]

In the past 15 years there has been substantial research on the inhalation dosimetry for formaldehyde in rodents and primates by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology), with a focus on use of rodent data for refinement of the quantitative cancer dose-response assessment.[33,34,35] CIIT's risk assessment of formaldehyde incorporated mechanistic and dosimetric information on formaldehyde. However, it should be noted that recent research published by EPA indicates that when two-stage modeling assumptions are varied, resulting dose-response estimates can vary by several orders of magnitude.[36,37,38,39] These findings are not supportive of interpreting the CIIT model results as

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Formaldehyde, Office of Pesticides and Toxic Substances, April 1987.

[28] Hauptmann, M..; Lubin, J. H.; Stewart, P. A.; Hayes, R. B.; Blair, A. 2003. Mortality from lymphohematopoetic malignancies among workers in formaldehyde industries. Journal of the National Cancer Institute 95: 1615-1623.

[29] Hauptmann, M..; Lubin, J. H.; Stewart, P. A.; Hayes, R. B.; Blair, A. 2004. Mortality from solid cancers among workers in formaldehyde industries. American Journal of Epidemiology 159: 1117-1130.

[30] Beane Freeman, L. E.; Blair, A.; Lubin, J. H.; Stewart, P. A.; Hayes, R. B.; Hoover, R. N.; Hauptmann, M. 2009. Mortality from lymphohematopoietic malignancies among workers in formaldehyde industries: The National Cancer Institute cohort. J. National Cancer Inst. 101: 751-761.

[31] Pinkerton, L. E. 2004. Mortality among a cohort of garment workers exposed to formaldehyde: an update. Occup. Environ. Med. 61: 193-200.

[32] Coggon, D, EC Harris, J Poole, KT Palmer. 2003. Extended follow-up of a cohort of British chemical workers exposed to formaldehyde. J National Cancer Inst. 95:1608-1615.

[33] Conolly, RB, JS Kimbell, D Janszen, PM Schlosser, D Kalisak, J Preston, and FJ Miller. 2003. Biologically motivated computational modeling of formaldehyde carcinogenicity in the F344 rat. Tox Sci 75: 432-447.

[34] Conolly, RB, JS Kimbell, D Janszen, PM Schlosser, D Kalisak, J Preston, and FJ Miller. 2004. Human respiratory tract cancer risks of inhaled formaldehyde: Dose-response predictions derived from biologically-motivated computational modeling of a combined rodent and human dataset. Tox Sci 82: 279-296.

[35] Chemical Industry Institute of Toxicology (CIIT).1999. Formaldehyde: Hazard characterization and dose-response assessment for carcinogenicity by the route of inhalation. CIIT, September 28, 1999. Research Triangle Park, NC.

[36] U.S. EPA. Analysis of the Sensitivity and Uncertainty in 2-Stage Clonal Growth Models for Formaldehyde with Relevance to Other Biologically-Based Dose Response (BBDR) Models. U.S. Environmental Protection Agency, Washington, D.C., EPA/600/R-08/103, 2006

[37] Subramaniam, R; Chen, C; Crump, K; .et .al. (2006) Uncertainties in biologically-based modeling of formaldehyde-induced cancer risk: identification of key issues. Risk Anal 28(4):907-923.

providing a conservative (health protective) estimate of human risk.[40] EPA research also examined the contribution of the two-stage modeling for formaldehyde towards characterizing the relative weights of key events in the mode-of-action of a carcinogen. For example, the model-based inference in the published CIIT study that formaldehyde's direct mutagenic action is not relevant to the compound's tumorigenicity was found not to hold under variations of modeling assumptions.[41]

Based on the developments of the last decade, in 2004, the working group of the IARC concluded that formaldehyde is carcinogenic to humans (Group 1), on the basis of sufficient evidence in humans and sufficient evidence in experimental animals - a higher classification than previous IARC evaluations. After reviewing the currently available epidemiological evidence, the IARC (2006) characterized the human evidence for formaldehyde carcinogenicity as "sufficient," based upon the data on nasopharyngeal cancers; the epidemiologic evidence on leukemia was characterized as "strong."[42] EPA is reviewing the recent work cited above from the NCI and NIOSH, as well as the analysis by the CIIT Centers for Health Research and other studies, as part of a reassessment of the human hazard and dose-response associated with formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (burning and watering of the eyes), nose and throat. Effects from repeated exposure in humans include respiratory tract irritation, chronic bronchitis and nasal epithelial lesions such as metaplasia and loss of cilia. Animal studies suggest that formaldehyde may also cause airway inflammation – including eosinophil infiltration into the airways. There are several studies that suggest that formaldehyde may increase the risk of asthma – particularly in the young.[43,44]

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[38] Subramaniam, R; Chen, C; Crump, K; .et .al. (2007). Uncertainties in the CIIT 2-stage model for formaldehyde-induced nasal cancer in the F344 rat: a limited sensitivity analysis-I. Risk Anal 27:1237

[39] Crump, K; Chen, C; Fox, J; .et .al. (2006) Sensitivity analysis of biologically motivated model for formaldehyde-induced respiratory cancer in humans. Ann Occup Hyg 52:481-495.

[40] Crump, K; Chen, C; Fox, J; .et .al. (2006) Sensitivity analysis of biologically motivated model for formaldehyde-induced respiratory cancer in humans. Ann Occup Hyg 52:481-495.

[41] Subramaniam, R; Chen, C; Crump, K; .et .al. (2007). Uncertainties in the CIIT 2-stage model for formaldehyde-induced nasal cancer in the F344 rat: a limited sensitivity analysis-I. Risk Anal 27:1237

[42] International Agency for Research on Cancer (2006) Formaldehyde, 2-Butoxyethanol and 1-tert-Butoxypropan-2-ol. Monographs Volume 88. World Health Organization, Lyon, France.

[43] Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for Formaldehyde. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp111.html

[44] WHO (2002) Concise International Chemical Assessment Document 40: Formaldehyde. Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organization, and the

6.5.2.5 Dioxins (Chlorinated dibenzodioxins (CDDs)[45]

A number of effects have been observed in people exposed to 2,3,7,8-TCDD levels that are at least 10 times higher than background levels. The most obvious health effect in people exposure to relatively large amounts of 2,3,7,8-TCDD is chloracne. Chloracne is a severe skin disease with acne-like lesions that occur mainly on the face and upper body. Other skin effects noted in people exposed to high doses of 2,3,7,8-TCDD include skin rashes, discoloration, and excessive body hair. Changes in blood and urine that may indicate liver damage also are seen in people. Alterations in the ability of the liver to metabolize (or breakdown) hemoglobin, lipids, sugar, and protein have been reported in people exposed to relatively high concentrations of 2,3,7,8-TCDD. Most of the effects are considered mild and were reversible. However, in some people these effects may last for many years. Slight increases in the risk of diabetes and abnormal glucose tolerance have been observed in some studies of people exposed to 2,3,7,8-TCDD. We do not have enough information to know if exposure to 2,3,7,8-TCDD would result in reproductive or developmental effects in people, but animal studies suggest that this is a potential health concern.

In certain animal species, 2,3,7,8-TCDD is especially harmful and can cause death after a single exposure. Exposure to lower levels can cause a variety of effects in animals, such as weight loss, liver damage, and disruption of the endocrine system. In many species of animals, 2,3,7,8-TCDD weakens the immune system and causes a decrease in the system's ability to fight bacteria and viruses at relatively low levels (approximately 10 times higher than human background body burdens). In other animal studies, exposure to 2,3,7,8-TCDD has caused reproductive damage and birth defects. Some animal species exposed to CDDs during pregnancy had miscarriages and the offspring of animals exposed to 2,3,7,8-TCDD during pregnancy often had severe birth defects including skeletal deformities, kidney defects, and weakened immune responses. In some studies, effects were observed at body burdens 10 times higher than human background levels.

World Health Organization, and produced within the framework of the Inter-Organization Programme for the Sound Management of Chemicals. Geneva.

[45] All health effects language for this section came from: Agency for Toxic Substances and Disease Registry (ATSDR). 1999. ToxFAQs for Chlorinated Dibenzo-p-dioxins (CDDs) (CAS#: 2,3,7,8-TCDD 1746-01-6). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at http://www.atsdr.cdc.gov/tfacts104.html.

6.5.2.6 Furans (Chlorinated dibenzofurans (CDFs))[46]

Most of the information on the adverse health effects comes from studies in people who were accidentally exposed to food contaminated with CDFs. The amounts that these people were exposed to were much higher than are likely from environmental exposures or from a normal diet. Skin and eye irritations, especially severe acne, darkened skin color, and swollen eyelids with discharge, were the most obvious health effects of the CDF poisoning. CDF poisoning also caused vomiting and diarrhea, anemia, more frequent lung infections, numbness, effects on the nervous system, and mild changes in the liver. Children born to exposed mothers had skin irritation and more difficulty learning, but it is unknown if this effect was permanent or caused by CDFs alone or CDFs and polychlorinated biphenyls in combination.

Many of the same effects that occurred in people accidentally exposed also occurred in laboratory animals that ate CDFs. Animals also had severe weight loss, and their stomachs, livers, kidneys, and immune systems were seriously injured. Some animals had birth defects and testicular damage, and in severe cases, some animals died. These effects in animals were seen when they were fed large amounts of CDFs over a short time, or small amounts over several weeks or months. Nothing is known about the possible health effects in animals from eating CDFs over a lifetime.

6.5.2.7 Benzene

According to NATA for 2002, benzene is the largest contributor to cancer risk of all 124 pollutants quantitatively assessed in the 2002 NATA.[47] The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure, and concludes that exposure is associated with additional health effects, including genetic changes in both humans and animals and increased proliferation of bone marrow cells in mice.[48] EPA states in its IRIS database that data indicate a causal relationship between benzene exposure and acute

[46] All health effects language for this section came from: Agency for Toxic Substances and Disease Registry (ATSDR). 1995. ToxFAQs™ for Chlorodibenzofurans (CDFs). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at <http://www.atsdr.cdc.gov/tfacts32.html>.

[47] U.S. EPA. (2009) 2002 National-Scale Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata2002/

[48] U.S. EPA. 2000. Integrated Risk Information System File for Benzene. This material is available electronically at: http://www.epa.gov/iris/subst/0276.htm.

International Agency for Research on Cancer, IARC monographs on the evaluation of carcinogenic risk of chemicals to humans, Volume 29, Some industrial chemicals and dyestuffs, International Agency for Research on Cancer, World Health Organization, Lyon, France, p. 345-389, 1982.

Irons, R.D.; Stillman, W.S.; Colagiovanni, D.B.; Henry, V.A. (1992) Synergistic action of the benzene metabolite hydroquinone on myelopoietic stimulating activity of granulocyte/macrophage colony-stimulating factor in vitro, Proc. Natl. Acad. Sci. 89:3691-3695.

lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. The International Agency for Research on Carcinogens (IARC) has determined that benzene is a human carcinogen and the U.S. Department of Health and Human Services (DHHS) has characterized benzene as a known human carcinogen.[49] A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[50] The most sensitive noncancer effect observed in humans, based on current data, is the depression of the absolute lymphocyte count in blood.[51] In addition, recent work, including studies sponsored by the Health Effects Institute (HEI), provides evidence that biochemical responses are occurring at lower levels of benzene exposure than previously known.[52] EPA's IRIS program has not yet evaluated these new data.

6.5.2.8 Other Air Toxics

In addition to the compounds described above, other compounds in gaseous hydrocarbon and PM emissions would be affected by this rule, including metal and organic HAPs. Information regarding the health effects of these compounds can be found in EPA's IRIS database.[53]

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[49] International Agency for Research on Cancer (IARC). 1987. Monographs on the evaluation of carcinogenic risk of chemicals to humans, Volume 29, Supplement 7, Some industrial chemicals and dyestuffs, World Health Organization, Lyon, France.

U.S. Department of Health and Human Services National Toxicology Program 11th Report on Carcinogens available at: http://ntp.niehs.nih.gov/go/16183.

[50] Aksoy, M. (1989). Hematotoxicity and carcinogenicity of benzene. Environ. Health Perspect. 82: 193-197.

Goldstein, B.D. (1988). Benzene toxicity. Occupational medicine. State of the Art Reviews. 3: 541-554.

[51] Rothman, N., G.L. Li, M. Dosemeci, W.E. Bechtold, G.E. Marti, Y.Z. Wang, M. Linet, L.Q. Xi, W. Lu, M.T. Smith, N. Titenko-Holland, L.P. Zhang, W. Blot, S.N. Yin, and R.B. Hayes (1996) Hematotoxicity among Chinese workers heavily exposed to benzene. Am. J. Ind. Med. 29: 236-246.

U.S. EPA 2002 Toxicological Review of Benzene (Noncancer Effects). Environmental Protection Agency, Integrated Risk Information System (IRIS), Research and Development, National Center for Environmental Assessment, Washington DC. This material is available electronically at http://www.epa.gov/iris/subst/0276.htm.

[52] Qu, O.; Shore, R.; Li, G.; Jin, X.; Chen, C.L.; Cohen, B.; Melikian, A.; Eastmond, D.; Rappaport, S.; Li, H.; Rupa, D.; Suramaya, R.; Songnian, W.; Huifant, Y.; Meng, M.; Winnik, M.; Kwok, E.; Li, Y.; Mu, R.; Xu, B.; Zhang, X.; Li, K. (2003). HEI Report 115, Validation & Evaluation of Biomarkers in Workers Exposed to Benzene in China.

Qu, Q., R. Shore, G. Li, X. Jin, L.C. Chen, B. Cohen, et al. (2002). Hematological changes among Chinese workers with a broad range of benzene exposures. Am. J. Industr. Med. 42: 275- 285.

Lan, Qing, Zhang, L., Li, G., Vermeulen, R., et al. (2004). Hematotoxically in Workers Exposed to Low Levels of Benzene. Science 306: 1774-1776. Turtletaub, K.W. and Mani, C. (2003). Benzene metabolism in rodents at doses relevant to human exposure from Urban Air. Research Reports Health Effect Inst. Report No.113.

[53] U.S. EPA Integrated Risk Information System (IRIS) database is available at: www.epa.gov/iris

6.6 Limitations and Uncertainties

The National Research Council (NRC) (2002) concluded that EPA's general methodology for calculating the benefits of reducing air pollution is reasonable and informative in spite of inherent uncertainties. To address these inherent uncertainties, NRC highlighted the need to conduct rigorous quantitative analysis of uncertainty and to present benefits estimates to decisionmakers in ways that foster an appropriate appreciation of their inherent uncertainty. In response to these comments, EPA's Office of Air and Radiation (OAR) is developing a comprehensive strategy for characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates. Components of that strategy include emissions modeling, air quality modeling, health effects incidence estimation, and valuation.

In this analysis, we use three methods to assess uncertainty quantitatively: Monte Carlo analysis, alternate concentration-response functions for PM mortality, and LML assessment. We also provide a qualitative assessment for those aspects that we are unable to address quantitatively in this analysis. Each of these analyses is described in detail in the following sections.

This analysis includes many data sources as inputs, including emission inventories, air quality data from models (with their associated parameters and inputs), population data, health effect estimates from epidemiology studies, and economic data for monetizing benefits. Each of these inputs may be uncertain and would affect the benefits estimate. When the uncertainties from each stage of the analysis are compounded, small uncertainties can have large effects on the total quantified benefits. In this analysis, we are unable to quantify the cumulative effect of all of these uncertainties, but we provide the following analyses to characterize many of the largest sources of uncertainty.

6.6.1 Monte Carlo analysis

Similar to other recent RIAs, we used Monte Carlo methods for characterizing random sampling error associated with the concentration response functions and economic valuation functions. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of morbidity. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. The reported standard errors in the epidemiological studies determined the distributions for individual effect estimates, as shown in Tables 6-2 and 6-3.

6.6.2 Alternate concentration-response functions for PM mortality

$PM_{2.5}$ mortality benefits are the largest benefit category that we monetized in this analysis. To better understand the concentration-response relationship between $PM_{2.5}$ exposure and premature mortality, EPA conducted an expert elicitation in 2006 (Roman et al., 2008; IEc, 2006). In general, the results of the expert elicitation support the conclusion that the benefits of $PM_{2.5}$ control are very likely to be substantial. In previous RIAs, EPA presented benefits estimates using concentration response functions derived from the $PM_{2.5}$ Expert Elicitation as a range from the lowest expert value (Expert K) to the highest expert value (Expert E). However, this approach did not indicate the agency's judgment on what the best estimate of PM benefits may be, and EPA's Science Advisory Board described this presentation as misleading. Therefore, we began to present the cohort-based studies (Pope et al, 2002; and Laden et al., 2006) as our core estimates in the proposal RIA for this rule (U.S. EPA, 2009a). Using alternate relationships between $PM_{2.5}$ and premature mortality supplied by experts, higher and lower benefits estimates are plausible, but most of the expert-based estimates fall between the two epidemiology-based estimates (Roman et al., 2008).

In this analysis, we present the results derived from the expert elicitation as indicative of the uncertainty associated with a major component of the health impact functions, and we provide the independent estimates derived from each of the twelve experts to better characterize the degree of variability in the expert responses. In this section, we provide the results using the concentration-response functions derived from the expert elicitation in both tabular (Tables 6-2 and 6-3) and graphical form (Figure 6-5). Please note that these results are not the direct results from the studies or expert elicitation; rather, the estimates are based in part on the concentration-response function provided in those studies.

6.6.3 LML assessment

$PM_{2.5}$ mortality benefits are the largest benefit category that we monetized in this analysis. To better characterize the uncertainty associated with mortality impacts that are estimated to occur in areas with low baseline levels of $PM_{2.5}$, we included the LML assessment. We have more confidence in the mortality impacts among populations exposed to levels of $PM_{2.5}$ above the lowest LML of the large cohort studies, and our confidence in the results diminish as we model that are lower than the LML. While an LML assessment provides some insight into the level of uncertainty in the estimated PM mortality benefits, EPA does not view the LML as a threshold and continues to quantify PM-related mortality impacts using a full range of modeled air quality concentrations. It is important to emphasize that just because we have greater confidence in the benefits above the LML, this does not mean that we have no confidence that

benefits occur below the LML. In section 6.3, we provide the results of the LML assessment in Figures 6-6 and 6-7.

6.6.4 Qualitative assessment of uncertainty and other analysis limitations

Although we strive to incorporate as many quantitative assessments of uncertainty, there are several aspects for which we are only able to address qualitatively. These aspects are important factors to consider when evaluating the relative benefits of the attainment strategies for each of the alternative standards:

Above we present the estimates of the total monetized benefits, based on our interpretation of the best available scientific literature and methods and supported by the SAB-HES and the NAS (NRC, 2002). The benefits estimates are subject to a number of assumptions and uncertainties. For example, the key assumptions underlying the estimates for premature mortality, which typically account for at least 90% of the total monetized benefits, include the following:

1. We assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because $PM_{2.5}$ produced via transported precursors emitted from EGUs may differ significantly from direct $PM_{2.5}$ released from diesel engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

2. We assume that the health impact function for fine particles is linear down to the lowest air quality levels modeled in this analysis. Thus, the estimates include health benefits from reducing fine particles in areas with varied concentrations of $PM_{2.5,}$ including both regions that are in attainment with fine particle standard and those that do not meet the standard down to the lowest modeled concentrations.

3. To characterize the uncertainty in the relationship between $PM_{2.5}$ and premature mortality (which typically accounts for 85% to 95% of total monetized benefits), we include a set of twelve estimates based on results of the expert elicitation study in addition to our core estimates. Even these multiple characterizations omit the uncertainty in air quality estimates, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. As a result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the $PM_{2.5}$ estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis. For more information on the uncertainties associated with $PM_{2.5}$ benefits, please consult the $PM_{2.5}$ NAAQS RIA (Table 5.5).

In addition, there is some uncertainty associated with the specificity of the air quality inputs to benefits model for this particular regulatory scenario. By assuming that each kiln

proportionately reduces their emissions by the same percentage as the national percentage reduction, we may be slightly under or overestimating the air quality impacts at specific locations and the associated monetized benefits. By including the hazardous waste kilns in the emissions inventory, we may be slightly overestimating the air quality impacts and monetized benefits. By omitting the decrease in domestic cement production and transportation, we are underestimating the air quality impacts and monetized benefits. By omitting the increase in cement imports, we may be overestimating the monetized benefits by not accounting for additional global pollutants. By using national average benefit-per-ton estimates to calculate the energy disbenefits, we may be under or overestimating these monetized disbenefits. Despite our inability to fully characterize and quantify these relatively small effects, we believe that, on net, the air quality impacts and associated monetized benefits are representative of the magnitude of benefits anticipated from this regulation.

As previously described, we strive to monetize as many of the benefits anticipated from this rule as possible, but the monetized benefits estimated in this RIA inevitably only reflect the portion of benefits. Specifically, only the benefits attributable to the health impacts associated with exposure to ambient fine particles have been monetized in this analysis. Data, resource, and methodological limitations prevented EPA from quantifying or monetizing the benefits from several important benefit categories, including benefits from reducing toxic emissions, ecosystem effects, and visibility impairment. Data limitations include limited monitoring for HAPs, incomplete emissions inventories for HAPs, and limited photochemical air quality modeling for non-mercury HAPs. Resource limitations include limited staff and extramural funding in conjunction with a heavy regulatory workload. Methodological limitations include an absence of concentration-response functions for many HAP health effects, with issues such as exposure misclassification, small number of cases, confounding, and extrapolation of toxicological effects down to ambient levels (IEc, 2008). Despite our inability to monetize all of the benefit categories, the monetized benefits still exceed the costs by a substantial margin.

This RIA does not include the type of detailed uncertainty assessment found in the PM NAAQS RIA. However, the results of the Monte Carlo analyses of the health and welfare benefits presented in Chapter 5 of the PM RIA can provide some evidence of the uncertainty surrounding the benefits results presented in this analysis.

6.7 Comparison of Benefits and Costs

Using a 3% discount rate, we estimate the total monetized benefits of the final Portland Cement NESHAP and NSPS to be $7.4 billion to $18 billion in the implementation year (2013).

Using a 7% discount rate, we estimate the total monetized benefits of the final Portland Cement NESHAP and NSPS to be $6.7 billion to $16 billion. These estimates include the energy disbenefits associated with increased electricity usage by the control devices. The annualized social costs of the final NESHAP and NSPS are $926 to $950 million.[54] Thus, net benefits are $6.5 billion to $17 billion at a 3% discount rate for the benefits and $5.8 billion to $16 billion at a 7% discount rate. In addition, the benefits from reducing 16,400 pounds of mercury, 4,400 tons of NOx, 5,800 tons of HCl, and 5,200 tons of organic HAPs each year have not been included in these estimates. All estimates are in 2005$.

Table 6-5 shows a summary of the monetized benefits, social costs, and net benefits for the final Portland Cement NESHAP and NSPS, the final NSPS only, the final NESHAP only, and the more stringent NSPS and final NESHAP. Figures 6-12 and 6-13 show the full range of net benefits estimates (i.e., annual benefits minus annualized costs) utilizing the 14 different $PM_{2.5}$ mortality functions at discount rates of 3% and 7%. Data, resource, and methodological limitations prevented EPA from monetizing the benefits from several important benefit categories, including benefits from reducing hazardous air pollutants, ecosystem effects, and visibility impairment. EPA believes that the benefits are likely to exceed the costs under this rulemaking even when taking into account uncertainties in the cost and benefit estimates.

[54] For more information on the annualized costs, please refer to Section 4 of this RIA.

Table 6-8. Summary of the Monetized Benefits, Social Costs, and Net Benefits for the final Portland Cement NESHAP in 2013 (millions of 2005$)[1]

Final NESHAP and NSPS						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[2]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[3]	$926	to	$950	$926	to	$950
Net Benefits	$6,500	to	$17,000	$5,800	to	$16,000
Non-monetized Benefits[d]	4,400 tons of NO_x (includes energy disbenefits) 5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					
Final NSPS only						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[2]	$510	to	$1,300	$460	to	$1,100
Total Social Costs[3]		$72			$40	
Net Benefits	$470	to	$1,300	$420	to	$1,100
Non-monetized Benefits[d]	6,600 tons of NO_x 520 tons of HCl Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					
Final NESHAP only						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[2]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[3]	$904	to	$930	$904	to	$930
Net Benefits	$6,500	to	$17,000	$5,800	to	$16,000
Non-monetized Benefits[d]	5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, SO_2 exposure Ecosystem effects Visibility impairment					
Alternative: More Stringent NSPS and Final NESHAP						
	3% Discount Rate			7% Discount Rate		
Total Monetized Benefits[2]	$7,400	to	$18,000	$6,700	to	$16,000
Total Social Costs[3]	$955	to	$979	$955	to	$979
Net Benefits	$6,500	to	$17,000	$5,700	to	$15,000
Non-monetized Benefits[4]	7,800 tons of NO_x (includes energy disbenefits) 5,200 tons of organic HAPs 5,900 tons of HCl 16,400 pounds of mercury Health effects from HAPs, NO_2, and SO_2 exposure Ecosystem effects Visibility impairment					

[1] All estimates are for the implementation year (2013), and are rounded to two significant figures.

[2] The total monetized benefits reflect the human health benefits associated with reducing exposure to $PM_{2.5}$ through reductions of directly emitted $PM_{2.5}$ and $PM_{2.5}$ precursors such as SO_2. It is important to note that the monetized benefits include many but not all health effects associated with $PM_{2.5}$ exposure. Benefits are shown as a range from Pope et al. (2002) to Laden et al. (2006). These models assume that all fine particles, regardless of their chemical composition, are equally potent in causing premature mortality because there is no clear scientific evidence that would support the development of differential effects estimates by particle type. The total monetized benefits include the energy disbenefits.

[3] The methodology used to estimate social costs for one year in the multimarket model using surplus changes results in the same social costs for both discount rates.

[4] Due to data, methodology, and resource limitations, we were unable to monetize the benefits associated with these categories of benefits.

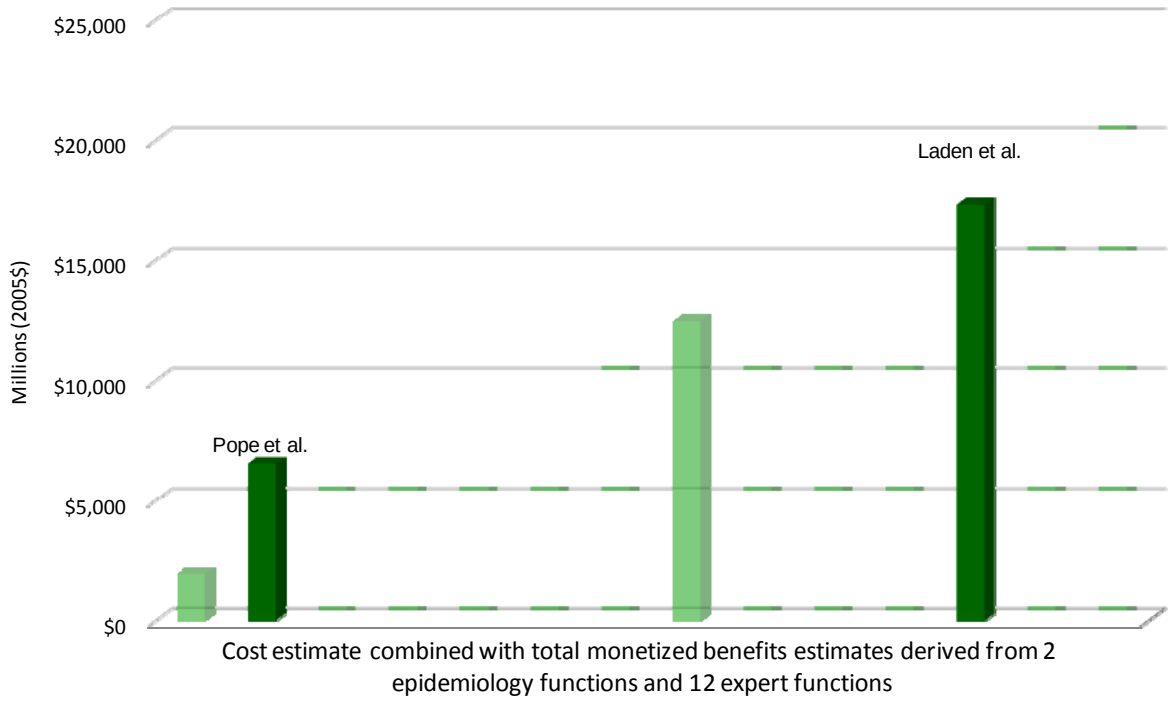

Figure 6-12. Net Benefits for the Final Portland Cement NESHAP and NSPS at 3% Discount Rate [a]

[a] Net Benefits are quantified in terms of $PM_{2.5}$ benefits for implementation year (2013). This graph shows 14 benefits estimates combined with the cost estimate. All combinations are treated as independent and equally probable. All fine particles are assumed to have equivalent health effects, but the benefit per ton estimates vary because each ton of precursor reduced has a different propensity to become $PM_{2.5}$. The monetized benefits incorporate the conversion from precursor emissions to ambient fine particles. These net benefits include the energy disbenefits. Due to data, methodology, and resource limitations, we were unable to monetize the benefits associated with several categories of benefits, including exposure to HAPs, NO_2, and SO_2, ecosystem effects, and visibility effects.

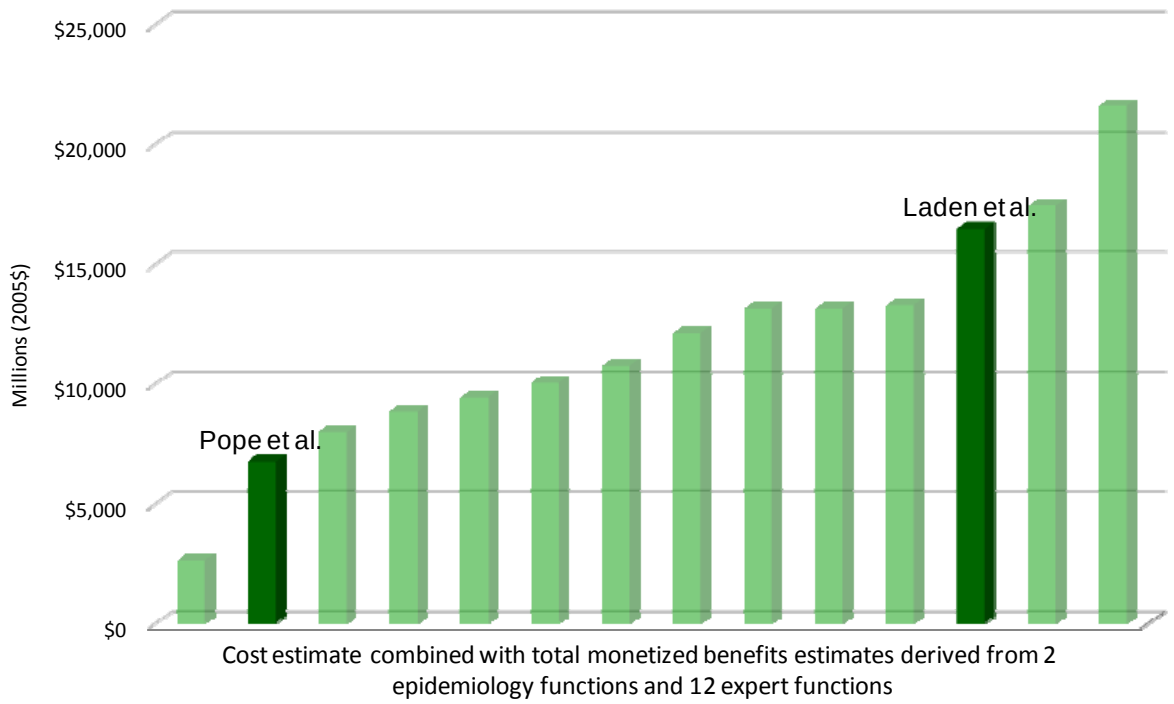

Figure 6-13. Net Benefits for the Final Portland Cement NESHAP and NSPS at 7% Discount Rate [a]

[a] Net Benefits are quantified in terms of $PM_{2.5}$ benefits for implementation year (2013). This graph shows 14 benefits estimates combined with the cost estimate. All combinations are treated as independent and equally probable. All fine particles are assumed to have equivalent health effects, but the benefit per ton estimates vary because each ton of precursor reduced has a different propensity to become $PM_{2.5}$. The monetized benefits incorporate the conversion from precursor emissions to ambient fine particles. These net benefits include the energy disbenefits. Due to data, methodology, and resource limitations, we were unable to monetize the benefits associated with several categories of benefits, including exposure to HAPs, NO_2, and SO_2, ecosystem effects, and visibility effects.

SECTION 7
REFERENCES

Abt Associates, Inc. 2008. Environmental Benefits and Mapping Program (Version 3.0). Bethesda, MD. Prepared for U.S. Environmental Protection Agency Office of Air Quality Planning and Standards. Research Triangle Park, NC. Available on the Internet at <http://www.epa.gov/air/benmap>.

Baker, K. and P. Scheff. 2007. "Photochemical model performance for $PM_{2.5}$, sulfate, nitrate, ammonium, and precursor species SO_2, HNO3, and NH_3 at background monitor locations in the central and eastern United States." *Atmospheric Environment* 41: 6185-6195.

Berman, E. and L. T. M. Bui. 2001. Environmental regulation and labor demand: Evidence from the south coast air basin. *Journal of Public Economics* 79(2):265-295.

Broda, C., N. Limao, and D.E. Weinstein. 2008b. "Optimal Tariffs and Market Power: The Evidence." *American Economic Review* 98(5):2032-2065.

Chang, J. S., R. A. Brost, Et Al. 1987. "A 3-Dimensional Eulerian Acid Deposition Model – Physical Concepts And Formulation." *Journal Of Geophysical Research-Atmospheres* 92(D12): 14681-14700.

Cole, M. A. and R. J. Elliott. 2007. Do environmental regulations cost jobs? An industry-level analysis of the UK. *B E Journal of Economic Analysis & Policy* 7(1) (Topics), Article 28.

Davidson K, Hallberg A, McCubbin D, Hubbell BJ. 2007. "Analysis of $PM_{2.5}$ Using the Environmental Benefits Mapping and Analysis Program (BenMAP)". *J Toxicol Environ Health* 70: 332—346.

Drevnick, P.E., D.E. Canfield, P.R. Gorski, A.L.C. Shinneman, D.R. Engstrom, D.C.G. Muir, G.R. Smith, P.J. Garrison, L.B. Cleckner, J.P. Hurley, R.B. Noble, R.R. Otter, and J.T. Oris. 2007. Deposition and cycling of sulfur controls mercury accumulation in Isle Royale fish. Environmental Science and Technology 41(21):7266-7272.

Dun & Bradstreet, Inc. 2007. *D&B Million Dollar Directory*. Bethlehem, PA: Dun & Bradstreet, Inc.

ENVIRON. 2008. *User's Guide Comprehensive Air Quality Model With Extensions*. Novato, ENVIRON International Corporation.

Fann, N., C.M. Fulcher, B.J. Hubbell. 2009. The influence of location, source, and emission type in estimates of the human health benefits of reducing a ton of air pollution. *Air Qual Atmos Health* 2:169-176.

Gery, M. W., G. Z. Whitten, Et Al. 1989. "A Photochemical Kinetics Mechanism For Urban And Regional Scale Computer Modeling." *Journal Of Geophysical Research-Atmospheres* 94(D10): 12925-12956.

Greenstone, M. 2002. "The Impacts of Environmental Regulations on Industrial Activity: Evidence from the 1970 and 1977 Clean Air Act Amendments and the Census of Manufactures." *Journal of Political Economy* 110(6):1175-1219.

Henderson, J. V. 1996. "Effects of Air Quality Regulation." *American Economic Review* 86(4):789-813.

Hubbell BJ, Fann N, Levy JI. 2009. "Methodological Considerations in Developing Local-Scale Health Impact Assessments: Balancing National, Regional and Local Data." *Air Qual Atmos Health* 2:99–110.

Industrial Economics, Inc. 2006. *Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality*. Prepared for the U.S. EPA, Office of Air Quality Planning and Standards, September. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf>.

Industrial Economics, Inc. 2008. *Section 812 Prospective Study of the Benefits and Costs of the Clean Air Act: Air Toxics Case Study – Health Benefits of Benzene Reductions in Houston, 1990-2020.* Prepared for the U.S. EPA, Office of Policy Analysis and Review, March. Available on the Internet at <http://www.epa.gov/oar/sect812/mar08/812CAA_Benzene_Houston_Draft_Report_March2008.pdf>.

Jans, I. and D. I. Rosenbaum. 1997. "Multimarket Contact and Pricing: Evidence from the U.S. Cement Industry." *International Journal of Industrial Organization* 15:391-412.

Kelly, T. and G. Matos. 2007a. "Historical Statistics for Mineral and Material Commodities in the United States: Cement End Use Statistics." U.S. Geological Survey Data Series 140, Version 1.2. Available at <http://minerals.usgs.gov/ds/2005/140/>.

Kelly, T. and G. Matos. 2007b. "Historical Statistics for Mineral and Material Commodities in the United States: Cement Supply and Demand Statistics." U.S. Geological Survey Data Series 140, Version 1.2. Available at <http://minerals.usgs.gov/ds/2005/140/>. Last modified April 11, 2006.

Kochi, I., B. Hubbell, and R. Kramer. 2006. "An Empirical Bayes Approach to Combining Estimates of the Value of Statistical Life for Environmental Policy Analysis." *Environmental and Resource Economics* 34:385-406.

Kunzli, N., S. Medina, R. Kaiser, P. Quenel, F. Horak Jr., and M. Studnicka. 2001. "Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates Based on Time Series or on Cohort Studies?" *American Journal of Epidemiology* 153(11):1050-55.

Laden, F., J. Schwartz, F.E. Speizer, and D.W. Dockery. 2006. "Reduction in Fine Particulate Air Pollution and Mortality." *American Journal of Respiratory and Critical Care Medicine* 173:667-672.

Levy JI, Baxter LK, Schwartz J. 2009. "Uncertainty and variability in health-related damages from coal-fired power plants in the United States." *Risk Anal.* 29 (7): 1000-1013.

LexisNexis. LexisNexis Academic [electronic resource]. Dayton, OH: LexisNexis.

Morgenstern, R. D., W. A. Pizer, and J. S. Shih. 2002. "Jobs versus the Environment: An Industry-Level Perspective." *Journal of Environmental Economics and Management* 43(3):412-436.

Mrozek, J.R., and L.O. Taylor. 2002. "What Determines the Value of Life? A Meta-Analysis." *Journal of Policy Analysis and Management* 21(2):253-270.

Munthe, J., R.A. Bodaly, B.A. Branfireun, C.T. Driscoll, C.C. Gilmour, R. Harris, M. Horvat, M. Lucotte, and O. Malm. 2007. "Recovery of Mercury-Contaminated Fisheries." *AMBIO: A Journal of the Human Environment* 36:33-44.

National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

National Research Council (NRC). 2008. *Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution.* National Academies Press. Washington, DC.

Nenes, A., S. N. Pandis, et al. 1999. "Continued development and testing of a new thermodynamic aerosol module for urban and regional air quality models." *Atmospheric Environment* 33(10): 1553-1560.

Nobel, C. E., E. C. McDonald-Buller, et al. 2001. "Accounting for spatial variation of ozone productivity in NO_x emission trading." *Environmental Science & Technology* 35(22): 4397-4407.

Office of Management and Budget (OMB). 2003. *Circular A-4: Regulatory Analysis*. Washington, DC. Available on the internet at http://www.whitehouse.gov/omb/circulars/a004/a-4.html.

Pope, C.A., III, 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." *Journal of the American Medical Association* 287:1132-1141.

Portland Cement Association (PCA). 2008a. "Cement and Concrete Basics: History & Manufacture of Portland Cement." Available at <http://www.cement.org/basics/concretebasics_history.asp>.

Portland Cement Association (PCA). 2008b. "Market Research: Producer Price Indices—Competitive Building Materials." Available at <http://www.cement.org/market/>.

Portland Cement Association (PCA). 2008c. "2007 North American Cement Industry Annual Yearbook." Available at <http://testinter.cement.org/bookstore/profile.asp?printpage=true&store=&id=15743>.

Portland Cement Association (PCA). December 2004. *U.S. and Canadian Portland Cement Industry: Plant Information Summary*. Skokie, IL: Portland Cement Association Economic Research Department.

Portland Cement Association (PCA). December 2005. *U.S. and Canadian Labor-Energy Input Survey 2005*. Skokie, IL: Portland Cement Association Economic Research Department.

Portland Cement Association (PCA). September 28, 2007. "Flash Report: Capacity Expansion Update." Available at < http://www.cement.org/econ/pdf/NONMEMBERCapacity Expansion.pdf>. As obtained on March 31, 2008.

Roman, Henry A., Katherine D. Walker, Tyra L. Walsh, Lisa Conner, Harvey M. Richmond, Bryan J. Hubbell, and Patrick L. Kinney. 2008. *Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S. Environ. Sci. Technol*., 42(7):2268-2274.

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* 54(3):158-164.

Russell, A. G. (2008). "EPA Supersites Program-related emissions-based particulate matter modeling: Initial applications and advances." *Journal of the Air & Waste Management Association* 58(2): 289-302.

Ryan, S. 2006. "The Cost of Environmental Regulation in a Concentrated Industry." Available at <http://econ-www.mit.edu/files/1166>.

Scudder, B.C., Chasar, L.C., Wentz, D.A., Bauch, N.J., Brigham, M.E., Moran, P.W., and Krabbenhoft, D.P. 2009. Mercury in fish, bed sediment, and water from streams across the United States, 1998–2005: U.S. Geological Survey Scientific Investigations Report 2009–5109, 74 p.

Sisler, J.F. 1996. Spatial and seasonal patterns and long-term variability of the composition of the haze in the United States: an analysis of data from the IMPROVE network. CIRA Report, ISSN 0737-5352-32, Colorado State University.

Tagaris E, Liao KJ, Delucia AJ, et al. 2009. "Potential impact of climate change on air-pollution related human health effects." *Environ. Sci. Technol*. 43: 4979—4988.

U.S. Department of Commerce, Bureau of the Census. 2003. *2001 Annual Survey of Manufactures*. M05(AS)-1. Washington, DC: Government Printing Office. Available at <http://www.census.gov/prod/2003pubs/m01as-1.pdf>. As obtained on March 14, 2008.

U.S. Department of Commerce, Bureau of the Census. 2006. *2005 Annual Survey of Manufactures*. M05(AS)-1. Washington, DC: Government Printing Office. Available at <http://www.census.gov/prod/2003pubs/m01as-1.pdf>. As obtained on March 14, 2008.

U.S. Department of Commerce, Bureau of the Census. 2010. *Sector 31: EC0731I1: Manufacturing: Industry Series: Detailed Statistics by Industry for the United States: 2007*. Available at < http://factfinder.census.gov/servlet/IBQTable?_bm=y&-ds_name=EC0731I1&-ib_type=NAICS2007&-NAICS2007=327310>.

U.S. Department of Energy, Energy Information Administration. 2007. *Annual Energy Outlook 2007*. Supplemental Washington, DC: U.S. Energy Information Administration.

U.S. Department of Energy, Energy Information Administration. 2010. *Annual Energy Outlook 2010*. Washington, DC: U.S. Energy Information Administration.

U.S. Department of Energy, Energy Information Administration. 2010. Supplemental Tables to the Annual Energy Outlook 2010. Available at <://www.eia.doe.gov/oiaf/aeo/supplement/supref.html >

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2007a. "Current Employment Statistics (National): Customizable Data Tables." Available at <http://www.bls.gov/ces/>. As obtained on March 14, 2008.

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2007b. "State and Area Employment, Hours and Earnings 2005." Washington, DC: U.S. Department of Labor.

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2008. "Consumer Price Index All Items – U.S. City Average Data: Customizable Data Tables." Available at <http://www.bls.gov/cpi/>. As obtained on March 14, 2008.

U.S. Department of Labor, Bureau of Labor Statistics (BLS). 2008. "Consumer Price Index All Items – U.S. City Average Data: Customizable Data Tables." Available at <http://www.bls.gov/cpi/>. As obtained on March 14, 2008.

U.S. Department of the Interior, U.S. Geological Survey. 2000. *Minerals Yearbook, Cement.* . Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/>. As obtained on March 14, 2008.

U.S. Department of the Interior, U.S. Geological Survey. 2001. *2000 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at ,<http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2002. *2001 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2003. *2002 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2004. *2003 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2005. *2004 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2007a. *2005 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Tables 11 and 15. http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/.

U.S. Department of the Interior, U.S. Geological Survey. 2007b. *2005 Minerals Yearbook, Crushed Stone*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2008a. *2006 Minerals Yearbook, Cement*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.er.usgs.gov/minerals/pubs/commodity/cement/>.

U.S. Department of the Interior, U.S. Geological Survey. 2008b. *Minerals Commodity Summaries, Cement 2008*. Washington, DC: U.S. Department of the Interior. Available at <http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2008-cemen.pdf>.

U.S. Department of the Interior, U.S. Geological Survey. 2010. *Minerals Commodity Summaries, Cement 2010*. Washington, DC: U.S. Department of the Interior. Available at < http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2010-cemen.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 1998. *Regulatory Impact Analysis of Cement Kiln Dust Rulemaking*. Appendix C, p. 12. June Washington, DC: U.S. Environmental Protection Agency. Available at <http://www.epa.gov/epaoswer/other/ckd/ckd/ckdriafn.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 1999. Office of Air Quality Planning and Standards. *OAQPS Economic Analysis Resource Document.* Available at <http://www.epa.gov/ttn/ecas/analguid.html>.

U.S. Environmental Protection Agency (U.S. EPA). 2000. *Guidelines for Preparing Economic Analyses*. EPA 240-R-00-003. September. National Center for Environmental Economics, Office of Policy Economics and Innovation. Washington, DC: EPA. Available at <http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/Guidelines.html>.

U.S. Environmental Protection Agency (U.S. EPA). 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines. EPA420-R-04-007.* Prepared by Office of Air and Radiation. Available at <http://www.epa.gov/nonroad-diesel/2004fr/420r04007.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2006. *Regulatory Impact Analysis, 2006 National Ambient Air Quality Standards for Particulate Matter, Chapter 5.* Available at <http://www.epa.gov/ttn/ecas/regdata/RIAs/Chapter%205--Benefits.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2007. *Guidance on the Use of Models and Other Analyses for Demonstrating Attainment of Air Quality Goals for Ozone, $PM_{2.5}$, and Regional Haze*. April. Office of Air Quality Planning and Standards RTP, NC. Available on the Internet at <http://www.epa.gov/ttn/scram/guidance/guide/final-03-pm-rh-guidance.pdf>

U.S. Environmental Protection Agency (U.S. EPA). 2008a. *Regulatory Impact Analysis, 2008 National Ambient Air Quality Standards for Ground-level Ozone, Chapter 6.* Available at <http://www.epa.gov/ttn/ecas/regdata/RIAs/6-ozoneriachapter6.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2008c. *Guidelines for Preparing Economic Analyses: External Review Draft.* National Center for Environmental Economics, Office of Policy Economics and Innovation. Washington, DC. Available at <http://yosemite.epa.gov/ee/epa/eermfile.nsf/vwAN/EE-0516-01.pdf/$File/EE-0516-01.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2008b. *Integrated Science Assessment for Sulfur Oxides—Health Criteria* (Final Report). National Center for Environmental Assessment, Research Triangle Park, NC. September. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=198843>.

U.S. Environmental Protection Agency (U.S. EPA). 2008c. *Integrated Science Assessment for Oxides of Nitrogen and Sulfur–Ecological Criteria National* (Final Report). National Center for Environmental Assessment, Research Triangle Park, NC. EPA/600/R-08/139. December. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=201485>.

U.S. Environmental Protection Agency (U.S. EPA). 2009a. *Regulatory Impact Analysis: National Emission Standards for Hazardous Air Pollutants from the Portland Cement Manufacturing Industry*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. April. Available at <http://www.epa.gov/ttn/ecas/regdata/RIAs/portlandcementria_4-20-09.pdf >.

U.S. Environmental Protection Agency (U.S. EPA). 2009b. *Integrated Science Assessment for Particulate Matter* (Final Report). EPA-600-R-08-139F. National Center for Environmental Assessment—RTP Division. December. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546>.

U.S. Environmental Protection Agency (U.S. EPA). 2009c. Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards: Final Report. Office of Air Quality Planning and Standards, Research Triangle Park, NC. August.

Available on the Internet at <http://www.epa.gov/ttn/naaqs/standards/so2/data/Risk%20and%20Exposure%20Assessment%20to%20Support%20the%20Review%20of%20the%20SO2%20Primary%20National%20Ambient%20Air%20Quality%20Standards-%20Final%20Report.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2010a. *Regulatory Impact Analysis for the Transport Rule.* Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available at <http://www.epa.gov/ttn/ecas/ria.html>.

U.S. Environmental Protection Agency (U.S. EPA). 2010b. Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality: Technical Support Document. Compiled by Office of Air Quality Planning and Standards, Research Triangle Park, NC. July. Available at <http://www.epa.gov/ttn/ecas/benefits.html>.

U.S. Environmental Protection Agency (U.S. EPA). 2010c. Air Quality Modeling Technical Support Document: National Emission Standards for Hazardous Air Pollutants from the Portland Cement Manufacturing Industry. Compiled by Office of Air Quality Planning and Standards, Research Triangle Park, NC. July. Available at <http://www.epa.gov/ttn/scram/reports/EPA-454_R-10-004.pdf>.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2004. *Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990–2020.* Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis. EPA-SAB-COUNCIL-ADV-04-002. March. Available on the Internet at <http://yosemite.epa.gov/sab%5CSABPRODUCT.NSF/08E1155AD24F871C85256E5400433D5D/$File/council_adv_04002.pdf>.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2005. *EPA's Review of the National Ambient Air Quality Standards for Particulate Matter (Second Draft PM Staff Paper, January 2005)*. EPA-SAB-CASAC-05-007. June.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2007. *SAB Advisory on EPA's Issues in Valuing Mortality Risk Reductio*n. EPA-SAB-08-001. October. Available at <http://yosemite.epa.gov/sab/sabproduct.nsf/4128007E7876B8F0852573760058A978/$File/sab-08-001.pdf >.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2009a. *Review of EPA's Integrated Science Assessment for Particulate Matter* (First External Review Draft, December 2008). EPA-COUNCIL-09-008. May. Available at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf>.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2009b. *Consultation on EPA's Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment*. EPA-COUNCIL-09-009. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/723FE644C5D758DF852575BD00763A32/$File/EPA-CASAC-09-009-unsigned.pdf>.

U.S. Environmental Protection Agency – Science Advisory Board (U.S. EPA-SAB). 2010. *Review of EPA's DRAFT Health Benefits of the Second Section 812 Prospective Study of the Clean Air Act.* EPA-COUNCIL-10-001. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/0/72D4EFA39E48CDB28525774500738776/$File/EPA-COUNCIL-10-001-unsigned.pdf>.

U.S. International Trade Commission (USITC). 2006. Gray Portland Cement and Cement Clinker from Japan Investigation No. 731-TA-461 (Second Review). Publication 3856. Available at <http://hotdocs.usitc.gov/docs/pubs/701_731/pub3856.pdf>.

Van Oss, H.G., and A.C. Padovani. 2002. "Cement Manufacture and the Environment Part I: Chemistry and Technology." *Journal of Industrial Ecology* 6(1):89-105.

Varian, H. 1992. *Microeconomic Analysis.* 3rd Ed. New York: W.W. Norton & Company.

Viscusi, V.K., and J.E. Aldy. 2003. "The Value of a Statistical Life: A Critical Review of Market Estimates throughout the World." *Journal of Risk and Uncertainty* 27(1):5-76.

APPENDIX A
SHORT-RUN REGIONAL PORTLAND CEMENT ECONOMIC MODEL

The Office of Air Quality Planning and Standards (OAQPS) has adopted the standard-industry level analysis described in the Office's resource manual (EPA, 1999a). This approach is consistent with previous EPA analyses of the Portland cement industry (EPA, 1998; EPA, 1999b) and uses a single-period static partial-equilibrium model to compare prepolicy cement market baselines with expected postpolicy outcomes in these markets. The benchmark time horizon for the analysis is the intermediate run where producers have some constraints on their flexibility to adjust factors of production. This time horizon allows us to capture important transitory impacts of the program on existing producers. Key measures in this analysis include

- market-level effects (market prices, changes in domestic production and consumption, and international trade),
- industry-level effects (changes in operating profits and employment),
- facility-level effects (plant utilization changes), and
- social costs (changes in producer and consumer surplus).

In this appendix, we provide additional details about economic model updates, model equations and parameters.

A.1 Economic Impact Model Updates Since Proposal

The need for a complete set of statistics makes the use of a 2005 baseline the best choice for a typical year. At the time of proposal model development, it was the latest year for which the PCA had published their plant information summary and complete statistics for updating variable cost functions were available. Details of model development are provided in EPA (2009), Appendix A. Since proposal, EPA identified several plants where operations had changed (see Table A-1). As a result, EPA modified the baseline U.S. production quantities to approximate these changes and maintain consistency with 2005 market conditions (Table A-2).

EPA also recognizes that the demand for cement is a derived demand because it depends on demand for sectors such as housing and construction. As a result, business cycles also significantly influence the cement industry (see Table A-3). If 2013 is more or less favorable for the cement industry than 2005, then impacts would be expected to change accordingly.

Table A-1. Economic Model Population Updates: 2005

Market	Approximate Clinker Capacity Removed (thousand metric tons)	Description	Approximate Clinker Capacity Added (thousand metric tons)	Net Change in Market Plant Population
Atlanta	300	Closure	0	−1
Baltimore/Philadelphia	500	Closure	400	−1
Chicago	600	Replacement	1,100	0
Dallas	800	Replacement	800	0
Detroit	900	Closure	0	-1
Kansas City	300	Closure	0	−1
Los Angeles	1,100	Replacement	2,200	0
Phoenix	600	Replacement	700	+1
San Antonio	300	Closure	0	0
St. Louis	500	Replacement	1,200	0

Table A-2. Revised Portland Cement Markets (10^6 metric tons): 2005

Market	U.S. Production Proposal	U.S. Production Revised	Difference
Atlanta	6.1	5.8	−0.3
Baltimore/Philadelphia	8.0	7.8	−0.2
Birmingham	5.9	5.9	0.0
Chicago	4.3	4.7	0.4
Cincinnati	3.7	3.7	0.0
Dallas	8.2	8.1	−0.1
Denver	3.4	3.4	0.0
Detroit	4.8	3.8	−1.0
Florida	5.6	5.5	−0.1
Kansas City	5.3	5.0	−0.3
Los Angeles	9.6	10.6	1.0
Minneapolis	1.7	1.7	0.0
New York/Boston	3.2	3.2	0.0
Phoenix	4.1	4.3	0.2
Pittsburgh	1.5	1.5	0.0
St. Louis	5.4	6.0	0.6
Salt Lake City	2.4	2.4	0.0
San Antonio	5.7	5.5	−0.2
San Francisco	3.4	3.4	0.0
Seattle	1.1	1.1	0.0
Total, Grey	93.6	93.6	0.0

Source: EPA calculations.

Table A-3. Recent Market Trends

Economic Variable	2005	2006	2007	2008	2009[a]
Clinker production (million metric tons)	87	89	86	78	58
Price, average mill value ($/metric ton)	$91	$102	$104	$103	$100
Employment (thousand)	16	16	16	15	14
Share of consumption provided by imports (percent)	25	27	19	11	8

[a]estimated.

Source: USGS, 2010. Mineral Commodity Survey 2010.
http://minerals.usgs.gov/minerals/pubs/commodity/cement/mcs-2010-cemen.pdf

A.2 Partial Equilibrium Model

The partial equilibrium analysis performed for this rule uses the cement market model developed during proposal (U.S. EPA, 2009). The model simulates how stakeholders (consumers and firms) may respond to the additional regulatory program costs. In the near term, the regional cement markets are assumed to have few sellers that offer similar/identical products. As a result, EPA used an oligopoly market structure[1]. As described in Section 3, this market structure assumption suggests that the observed baseline market price will be higher than marginal production costs (i.e., there may be a preexisting market distortion prior to regulation). To provide some intuition about factors that influence the size of the existing distortion, we express a seller's "best" supply decision as a function of the market price, the seller's market share, the market demand elasticity, and the seller's marginal costs (see Varian [1992], pp. 289–290):

$$\text{Price} \times (1 + \text{Market Share}_i/\text{Demand Elasticity}) = \text{Marginal Cost}_i.$$

This equation shows the relationship between the oligopoly model and perfect competition. The market distortion will typically be higher when market share_i is high (there are few sellers) and in markets where the quantity demanded is less sensitive to price (i.e., the demand elasticity is inelastic).

A.2.1 Model Equations

To estimate the economic impacts of the regulation, EPA used four linear equations to calculate the following unknown variables:

- change in domestic plant production (dq_i),
- change in imports ($dq^{imports}$),
- change in cement market quantity (dQ), and
- change in cement price (dP).

Equation 1: Domestic Supply. For each plant, we describe its response to the regulatory program as follows. The total compliance cost per ton (c_i) is applied to each kiln, and the difference in the highest cost kiln with-regulation and the highest cost kiln in the baseline approximates the plant's change in the marginal cost of production (dMC_i). In with-regulation

[1] There are different commonly used models of oligopoly in the economics literature. They differ with respect to the assumption about how a company believes competing companies will react to its own production decision. EPA selected the Cournot model where the company assumes competing companies' output is fixed in its own production decision.

equilibrium, the change in marginal revenue (dMRi) must equal the change in the marginal cost (dMCi) for each plant.[1]

$$\text{dmarginal Revenue}_i = \text{dmarginal Cost}_i$$

or

$$\text{dprice} \times \left(1 + \frac{\text{mkt share}_i}{\text{dem elasticity}}\right) + \frac{\text{dplant q}}{\text{market Q}} \times \frac{\text{price}}{\text{dem elasticity}} - \frac{\text{dmarket Q}}{(\text{market Q})^2} \times \text{plant q} \times \frac{\text{price}}{\text{dem elasticity}} = \text{dmarginal cost}$$

Equation 2: Supply of Imports. If applicable to the market, an equation describing the supply of cement from other countries was included:

$$\text{dimports} = \text{import supply elasticity} \times (\text{dprice/baseline price}) \times \text{baseline imports}.$$

For import supply, EPA used the latest empirical work on how other countries who export (i.e., supply imports) to the United States respond to price changes. Broda et al. (2008) report that the export supply elasticity for commodities imported by the United States was approximately two. This implies that a 1% increase in prices results in a 2% increase in the volume of exports for a typical good.

Equation 3: Market Supply. Market supply of Portland cement equals the change in domestic production and imports:

$$\text{dmarket Q} = \text{dtotal domestic production} + \text{dimports}.$$

This condition ensures that the market quantity is consistent with the individual supply decisions of domestic plants and imports in the new with-regulation equilibrium for each regional market.

Equation 4: Market Demand. The demand for Portland cement is derived from the demand for concrete products, which, in turn, is derived largely from the demand for construction. Based on a linear demand equation, the market demand condition for Portland cement must hold based on the projected change in market price, that is,

$$\text{dMarketQ} = \text{demand elasticity (dprice/baseline price)} \times \text{baseline consumption}.$$

The use of published estimates from previous rulemakings is appropriate in cases when the cost of preparing original estimates is high (EPA, 2000). In previous analyses, EPA econometrically

[1] To highlight and make transparent the assumptions regarding seller behavior, this equation is formally derived in Appendix B.

estimated the demand elasticity for cement and reported a point estimate of −0.88 (EPA, 1998). This value suggests that a 1% increase in the cement price would lead to a 0.88% reduction in cement consumption.

APPENDIX B
THE CEMENT PLANT'S PRODUCTION DECISION: A MATHEMATICAL REPRESENTATION

This appendix provides additional detail about the cement 's production decision used in the economic model (see Equation 1 in Section 3 of the RIA). Table B-1 identifies and describes the key variables of the cement plant's profit function.

Table B-1. Variable Descriptions

P	Market price
$Q = \sum q_i$	Market output
q_i	Domestic plant i's output
FC_i	Plant fixed costs
VC_i	Plant variable costs

Step 1: First, we assume the plant's goal is to maximize profits:

$$\max_{q_i} \pi_i(Q) = P(Q)q_i - VC_i(q_i) - FC_i \ .$$

Step 2: The first-order conditions for a profit maximum are:

$$\frac{\partial \pi_i}{q_i} = P + \frac{\partial P(Q)}{\partial q_i} q_i - \frac{\partial VC_i(q_i)}{q_i} q_i = 0 \ .$$

Step 3: Apply two key assumptions in the Cournot price model:

- Plant's (i) recognizes its own production decisions influence the market price:

$$\frac{\partial P}{\partial q_i} \neq 0$$

- Plant (i) output decisions do not affect those of any other plant (j) (e.g., there is no strategic action among cement plants):

$$\frac{\partial q_j}{\partial q_i} = 0$$

Step 4: Next, multiply second term by

$$1 = \frac{Q}{P}\frac{P}{Q}$$

$$P + \frac{\partial P(Q)}{\partial q_i} q_i \left(\frac{Q}{P} \frac{P}{Q} \right) - \frac{\partial VC_i(q_i)}{q_i} q_i = 0 \ .$$

Step 5: Rearranging terms:

$$P + \left(\frac{\partial P(Q)}{P} \frac{Q}{\partial q_i} \right) \left(\frac{q_i}{Q} \right) P - \frac{\partial VC_i(q_i)}{q_i} q_i = 0 \ .$$

Step 6: Use and apply the following definitions:

$$\left(\frac{\partial P(Q)}{P} \frac{Q}{\partial q_i} \right) = \frac{1}{\eta} = \text{inverse demand elasticity}$$

$$\left(\frac{q_i}{Q} = q_i Q^{-1} \right) = \text{plant's market share} \ .$$

We derive the following expression:

$$P \left[1 + \left(\frac{\frac{q_i}{Q}}{\eta} \right) \right] = \frac{\partial VC_i(q_i)}{q_i} \ .$$

Step 7: The total differential of this equation is determined and gives us the optimal decision rule for the plant:

$$dP \left[1 + \left(\frac{\frac{q_i}{Q}}{\eta} \right) \right] + dq_i \left[\frac{P}{\eta} \frac{1}{Q} \right] - dQ \left[\frac{P}{\eta} \frac{q_i}{Q^2} \right] = \text{dMC} \ .$$

APPENDIX C
SOCIAL COST METHODOLOGY

The Office of Air Quality Planning and Standards (OAQPS) has adopted the standard industry-level analysis described in the Office's resource manual (EPA, 1999a). This approach is consistent with previous EPA analyses of the Portland cement industry (EPA, 1998; EPA, 1999b) and uses a single-period static partial-equilibrium model to compare prepolicy cement market baselines with expected postpolicy outcomes in these markets. The benchmark time horizon for the analysis is the intermediate run where producers have some constraints on their flexibility to adjust factors of production. This time horizon allows us to capture important transitory impacts of the program on existing producers. The model provides an estimate of the social costs (changes in producer and consumer surplus) associated with controls applying to existing kilns (see Section 4). Since the social cost methodology is identical to the approach used in previous cement analysis (EPA, 1998, Appendix C), we have included elements of the previous report's Appendix C in this RIA.

Figure C-1 illustrates the conceptual framework for evaluating the social cost and distributive impacts under the imperfectly competitive structure of U.S. cement markets. The baseline equilibrium is given by the price, P_0, and the quantity, Q_0. Without the regulation, the total benefits of consuming cement are given by the area under the demand curve up to the market output, Q_0. This equals the area filled by the letters ABCDEFGHIJ. The total variable cost to society of producing Q_0 equals the area under the MC function, given by the area IJ. Thus, the total surplus value to society from the production and consumption of output level Q_0 equals the total benefits minus the total costs, or the area filled by the letters ABCDEFGH.

This total surplus value to society can be further divided into producer surplus and consumer surplus. Producer surplus accrues to the suppliers of cement and reflects the value they receive in the market for producing Q_0 units of cement less their costs of production, i.e., their profits. As shown in Figure C-1, producer surplus is given by the area DEFGH, which is the difference between cement revenues (i.e., area DEFGHIJ) and production costs (area IJ). Consumer surplus accrues to the consumers of cement and reflects the value they place on consumption (total benefits of consumption) less what they must pay on the market, i.e., P_0. Consumer surplus is thereby given by the area ABC.

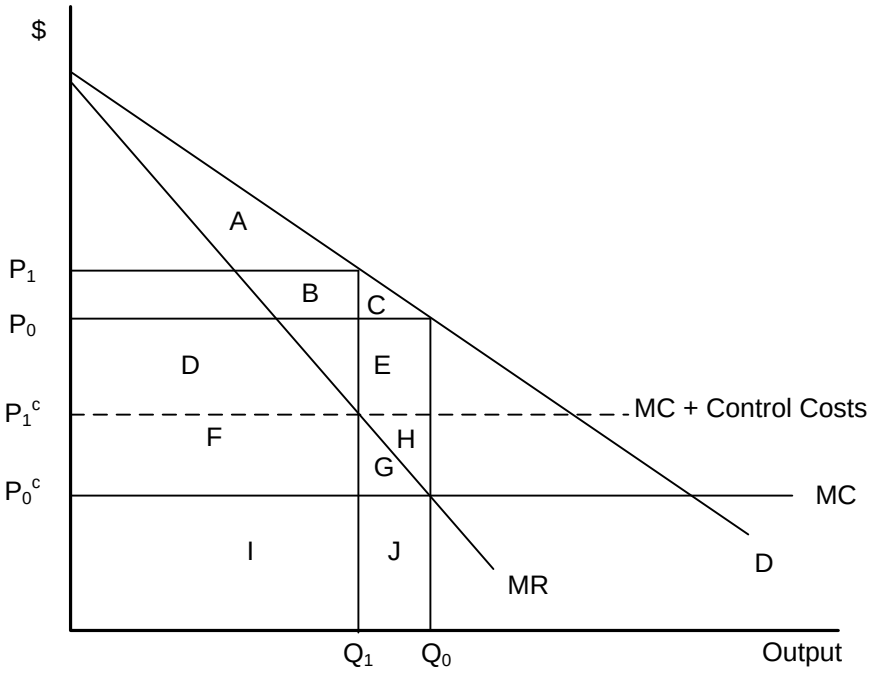

Figure C-1. Social Cost of Regulation Under Imperfect Competition

The final rule will increase the marginal cost of producing cement and thereby shift this curve upward by the amount of the incremental compliance costs. As shown in Figure C-1, this results in a new market equilibrium that occurs at a higher market price for cement, P_1, and a lower level of output, Q_1. In this scenario, the total benefits of consumption are equal to area ABDFI and the total variable costs of production are equal to area FI. This yields a with-regulation social surplus equal to area ABD with area BD representing the new producer surplus and area A being the new consumer surplus. The social cost of the regulation equals the total change in social surplus caused by the regulation. Therefore, the social cost of the regulation is represented by the area FGHEC in Figure C-1.

The distributive effects are estimated by separating the social cost into producer surplus and consumer surplus losses. First, the change in producer surplus is given by

$$\Delta PS = B - F - (G+H+E) \qquad (C.1)$$

Producers gain B from the increase in price (a transfer from consumers to producers), but lose F from the increase in production costs due to the incremental compliance costs. Furthermore, the reduction of cement production leads to foregone baseline profits of G+H+E.

The change in consumer surplus is given by

$$\Delta CS = -(B + C) \tag{C.2}$$

This change results from the reduction in consumer surplus from the baseline value of ABC to the with-regulation value of A. In this case, consumers lose area B as a transfer to producers through the increase in the price they pay for the with-regulation level of cement consumption, while the reduction in cement consumption due to regulation leads to foregone baseline value of consumption equal to area C.

The social cost or total change in social surplus can then be derived simply by adding the changes in producer and consumer surplus, i.e.,

$$\text{Social Cost} = \Delta PS + \Delta CS = -(F + G + H + E + C) \tag{C.3}$$

This estimate can be compared to the engineering estimate of incremental compliance cost to demonstrate the difference between these two estimates of social cost. The incremental compliance cost estimate is given by the area FGH, which is simply the constant cost per unit times the baseline output level of cement. The social cost estimate from Equation (C.3) above, however, exceeds the engineering estimate by the area EC. In other words, the incremental compliance cost estimate understates the social costs of the regulation. The reason for this follows directly from the imperfectly competitive structure of the markets for cement. A comparison with the outcome under perfect competition will assist in illustrating this point.

Suppose that the MR curve in Figure C-1 was the demand function for a competitive market, rather than the marginal revenue function for an imperfectly competitive producer. Similarly, let the MC function be the aggregate supply function for all producers in the market. The market equilibrium is still determined at the intersection of MC and MR, but given the revised interpretation of MR as the competitive demand function, the baseline (competitive) market price, P_0^C, is now equal to MC and Q_0 is now interpreted as the competitive level of cement demand. In this case, all social surplus goes to the consumer. This is because producers receive a price that just covers their costs of production.

In the with-regulation perfectly competitive equilibrium, the market price would rise by the per unit control cost amount to P_1^c. The social cost of the regulation is given entirely by the loss in consumer surplus as given by area FG. As shown in Figure C-1, this estimate of social cost is less than the incremental compliance cost estimate (i.e., area FGH) so that the engineering

estimate overstates the social cost of the regulation under perfect competition. The overstatement results from the fact that the incremental compliance costs are estimated based on the baseline market level of cement output. With regulation, output is projected to decline to Q_1, so that the actual incremental compliance costs incurred by the industry are given by area F. Area G represents the foregone value of cement consumption to consumers, also referred to as the deadweight loss (analogous to area C under the imperfect competition scenario).

In addition, the estimate of social cost under perfect competition is less than the estimate under imperfect competition by the area HEC, i.e.,

$$SC^{imp} - SC^{perf} = -[(F+G+H+E+C) - (F+G)] = -(H + E + C) \quad (C.4)$$

The difference between these two measures results from the fact that the price paid by consumers (i.e., marginal value to society for cement) exceeds the cost of producing cement (i.e., the marginal cost to society of producing cement). As shown in Figure C-1, this difference in social cost is equal to the area between the demand curve (D) and the marginal revenue curve (MR) that exist under imperfectly competitive market structure. This area does not exist under perfect competition because the MR curve is interpreted as the demand curve so that the price paid by consumers equals the marginal cost of producing cement. The pre-existing social inefficiency of imperfect competition is exacerbated as the regulation moves society further away from the socially optimal level of cement production, which results in social costs greater than the incremental compliance cost imposed on the cement industry.

APPENDIX D

SUMMARY OF EXPERT OPINIONS ON THE EXISTENCE OF A THRESHOLD IN THE CONCENTRATION-RESPONSE FUNCTION FOR $PM_{2.5}$-RELATED MORTALITY

Summary of Expert Opinions on the Existence of a Threshold in the Concentration-Response Function for $PM_{2.5}$-related Mortality

Technical Support Document (TSD)

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Compiled by:
U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impact Division
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A. HES Comments on 812 Analysis (2010)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2010. Review of EPA's DRAFT Health Benefits of the Second Section 812 Prospective Study of the Clean Air Act. EPA-COUNCIL-10-001. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/0/72D4EFA39E48CDB28525774500738776/$File/EPA-COUNCIL-10-001-unsigned.pdf>.

Pg 2: "The HES generally agrees with other decisions made by the EPA project team with respect to PM, in particular, the PM mortality effect threshold model, the cessation lag model, the inclusion of infant mortality estimation, and differential toxicity of PM."

Pg 2: "Further, the HES fully supports EPA's use of a no-threshold model to estimate the mortality reductions associated with reduced PM exposure."

Pg 6: "The HES also supports the Agency's choice of a no-threshold model for PM-related effects."

Pg 13: "The HES fully supports EPA's decision to use a no-threshold model to estimate mortality reductions. This decision is supported by the data, which are quite consistent in showing effects down to the lowest measured levels. Analyses of cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. Therefore, there is no evidence to support a truncation of the CRF."

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B. Scientific Statement from American Heart Association (2010)

Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC Jr, Whitsel L, Kaufman JD; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism. (2010). "Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association." ***Circulation*****. 121: 2331-2378.**

Pg 2338: "Finally, there appeared to be no lower-limit threshold below which PM_{10} was not associated with excess mortality across all regions."

Pg 2350: "There also appears to be a monotonic (eg, linear or log-linear) concentration-response relationship between $PM_{2.5}$ and mortality risk observed in cohort studies that extends below present-day regulations of 15 $\mu g/m^3$ for mean annual levels, without a discernable "safe" threshold." (cites Pope 2004, Krewski 2009, and Schwartz 2008)

Pg 2364: "The $PM_{2.5}$ concentration– cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 $\mu g/m^3$ (the 2006 annual NAAQS level) without a discernable "safe" threshold."

Pg 2365: "This updated review by the AHA writing group corroborates and strengthens the conclusions of the initial scientific statement. In this context, we agree with the concept and continue to support measures based on scientific evidence, such as the US EPA NAAQS, that seek to control PM levels to protect the public health. Because the evidence reviewed supports that there is no safe threshold, it appears that public health benefits would accrue from lowering $PM_{2.5}$ concentrations even below present-day annual (15 $\mu g/m^3$) and 24-hour (35 $\mu g/m^3$) NAAQS, if feasible, to optimally protect the most susceptible populations."

Pg 2366: "Although numerous insights have greatly enhanced our understanding of the PM-cardiovascular relationship since the first AHA statement was published, the following list represents broad strategic avenues for future investigation: ... Determine whether any "safe" PM threshold concentration exists that eliminates both acute and chronic cardiovascular effects in healthy and susceptible individuals and at a population level."

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C. Integrated Science Assessment for Particulate Matter (2009)

U.S. Environmental Protection Agency (U.S. EPA). 2009. Integrated Science Assessment for Particulate Matter (Final Report). EPA-600-R-08-139F. National Center for Environmental Assessment – RTP Division. December. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546>.

Pg 1-22: "An important consideration in characterizing the public health impacts associated with exposure to a pollutant is whether the concentration-response relationship is linear across the full concentration range encountered, or if nonlinear relationships exist along any part of this range. Of particular interest is the shape of the concentration-response curve at and below the level of the current standards. The shape of the concentration-response curve varies, depending on the type of health outcome, underlying biological mechanisms and dose. At the human population level, however, various sources of variability and uncertainty tend to smooth and "linearize" the concentration-response function (such as the low data density in the lower concentration range, possible influence of measurement error, and individual differences in susceptibility to air pollution health effects). In addition, many chemicals and agents may act by perturbing naturally occurring background processes that lead to disease, which also linearizes population concentration-response relationships (Clewell and Crump, 2005, 156359; Crump et al., 1976, 003192; Hoel, 1980, 156555). These attributes of population dose-response may explain why the available human data at ambient concentrations for some environmental pollutants (e.g., PM, O_3, lead [Pb], ETS, radiation) do not exhibit evident thresholds for health effects, even though likely mechanisms include nonlinear processes for some key events. These attributes of human population dose-response relationships have been extensively discussed in the broader epidemiologic literature (Rothman and Greenland, 1998, 086599)."

Pg 2-16: "In addition, cardiovascular hospital admission and mortality studies that examined the PM_{10} concentration-response relationship found evidence of a log-linear no-threshold relationship between PM exposure and cardiovascular-related morbidity (Section 6.2) and mortality (Section 6.5)."

Pg 2-25: "2.4.3. PM Concentration-Response Relationship
An important consideration in characterizing the PM-morbidity and mortality association is whether the concentration-response relationship is linear across the full concentration range that is encountered or if there are concentration ranges where there are departures from linearity (i.e., nonlinearity). In this ISA studies have been identified that attempt to characterize the shape of the concentration-response curve along with possible PM "thresholds" (i.e., levels which PM concentrations must exceed in order to elicit a health response). The epidemiologic studies evaluated that examined the shape of the concentration-response curve and the potential presence of a threshold have focused on cardiovascular hospital admissions and ED visits and mortality associated with short-term exposure to PM_{10} and mortality associated with long-term exposure to $PM_{2.5}$.

"A limited number of studies have been identified that examined the shape of the PM cardiovascular hospital admission and ED visit concentration-response relationship. Of these

studies, some conducted an exploratory analysis during model selection to determine if a linear curve most adequately represented the concentration-response relationship; whereas, only one study conducted an extensive analysis to examine the shape of the concentration-response curve at different concentrations (Section 6.2.10.10). Overall, the limited evidence from the studies evaluated supports the use of a no-threshold, log-linear model, which is consistent with the observations made in studies that examined the PM-mortality relationship.

"Although multiple studies have previously examined the PM-mortality concentration-response relationship and whether a threshold exists, more complex statistical analyses continue to be developed to analyze this association. Using a variety of methods and models, most of the studies evaluated support the use of a no-threshold, log-linear model; however, one study did observe heterogeneity in the shape of the concentration-response curve across cities (Section 6.5). Overall, the studies evaluated further support the use of a no-threshold log-linear model, but additional issues such as the influence of heterogeneity in estimates between cities, and the effect of seasonal and regional differences in PM on the concentration-response relationship still require further investigation.

"In addition to examining the concentration-response relationship between short-term exposure to PM and mortality, Schwartz et al. (2008, 156963) conducted an analysis of the shape of the concentration-response relationship associated with long-term exposure to PM. Using a variety of statistical methods, the concentration-response curve was found to be indistinguishable from linear, and, therefore, little evidence was observed to suggest that a threshold exists in the association between long-term exposure to $PM_{2.5}$ and the risk of death (Section 7.6)."

Pg 6-75: "6.2.10.10. Concentration Response
The concentration-response relationship has been extensively analyzed primarily through studies that examined the relationship between PM and mortality. These studies, which have focused on short- and long-term exposures to PM have consistently found no evidence for deviations from linearity or a safe threshold (Daniels et al., 2004, 087343; Samoli et al., 2005, 087436; Schwartz, 2004, 078998; Schwartz et al., 2008, 156963) (Sections 6.5.2.7 and 7.1.4). Although on a more limited basis, studies that have examined PM effects on cardiovascular hospital admissions and ED visits have also analyzed the PM concentration-response relationship, and contributed to the overall body of evidence which suggests a log-linear, no-threshold PM concentration-response relationship.

"The results from the three multicity studies discussed above support no-threshold log-linear models, but issues such as the possible influence of exposure error and heterogeneity of shapes across cities remain to be resolved. Also, given the pattern of seasonal and regional differences in PM risk estimates depicted in recent multicity study results (e.g., Peng et al., 2005, 087463), the very concept of a concentration-response relationship estimated across cities and for all-year data may not be very informative."

Pg 6-197: "6.5.2.7. Investigation of Concentration-Response Relationship
The results from large multicity studies reviewed in the 2004 PM AQCD (U.S. EPA, 2004, 056905) suggested that strong evidence did not exist for a clear threshold for PM mortality effects. However, as discussed in the 2004 PM AQCD (U.S. EPA, 2004, 056905), there are

several challenges in determining and interpreting the shape of PM-mortality concentration-response functions and the presence of a threshold, including: (1) limited range of available concentration levels (i.e., sparse data at the low and high end); (2) heterogeneity of susceptible populations; and (3) investigate the PM-mortality concentration-response relationship.

"Daniels et al. (2004, 087343) evaluated three concentration-response models: (1) log-linear models (i.e., the most commonly used approach, from which the majority of risk estimates are derived); (2) spline models that allow data to fit possibly non-linear relationship; and (3) threshold models, using PM_{10} data in 20 cities from the 1987-1994 NMMAPS data. They reported that the spline model, combined across the cities, showed a linear relation without indicating a threshold for the relative risks of death for all-causes and for cardiovascular-respiratory causes in relation to PM_{10}, but "the other cause" deaths (i.e., all cause minus cardiovascular-respiratory) showed an apparent threshold at around 50 $\mu g/m^3$ PM^{10}, as shown in Figure 6-35. For all-cause and cardio-respiratory deaths, based on the Akaike's Information Criterion (AIC), a log-linear model without threshold was preferred to the threshold model and to the spline model.

"The HEI review committee commented that interpretation of these results required caution, because (1) the measurement error could obscure any threshold; (2) the city-specific concentration-response curves exhibited a variety of shapes; and (3) the use of AIC to choose among the models might not be appropriate due to the fact it was not designed to assess scientific theories of etiology. Note, however, that there has been no etiologically credible reason suggested thus far to choose one model over others for aggregate outcomes. Thus, at least statistically, the result of Daniels et al. (2004, 087343) suggests that the log-linear model is appropriate in describing the relationship between PM10 and mortality.

"The Schwartz (2004, 078998) analysis of PM_{10} and mortality in 14 U.S. cities, described in Section 6.5.2.1, also examined the shape of the concentration-response relationship by including indicator variables for days when concentrations were between 15 and 25 $\mu g/m^3$, between 25 and 34 $\mu g/m^3$, between 35 and 44 $\mu g/m^3$, and 45 $\mu g/m^3$ and above. In the model, days with concentrations below 15 $\mu g/m^3$ served as the reference level. This model was fit using the single stage method, combining strata across all cities in the case-crossover design. Figure 6-36 shows the resulting relationship, which does not provide sufficient evidence to suggest that a threshold exists. The authors did not examine city-to-city variation in the concentration-response relationship in this study.

"PM_{10} and mortality in 22 European cities (and BS in 15 of the cities) participating in the APHEA project. In nine of the 22 cities, PM10 levels were estimated using a regression model relating co-located PM10 to BS or TSP. They used regression spline models with two knots (30 and 50 $\mu g/m^3$) and then combined the individual city estimates of the splines across cities. The investigators concluded that the association between PM and mortality in these cities could be adequately estimated using the log-linear model. However, in an ancillary analysis of the concentration-response curves for the largest cities in each of the three distinct geographic areas (western, southern, and eastern European cities): London, England; Athens, Greece; and Cracow, Poland, Samoli et al. (2005, 087436) observed a difference in the shape of the concentration-response curve across cities. Thus, while the combined curves (Figure 6-37) appear to support

no-threshold relationships between PM_{10} and mortality, the heterogeneity of the shapes across cities makes it difficult to interpret the biological relevance of the shape of the combined curves.

"The results from the three multicity studies discussed above support no-threshold log-linear models, but issues such as the possible influence of exposure error and heterogeneity of shapes across cities remain to be resolved. Also, given the pattern of seasonal and regional differences in PM risk estimates depicted in recent multicity study results (e.g., Peng et al., 2005, 087463), the very concept of a concentration-response relationship estimated across cities and for all-year data may not be very informative."

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D. CASAC comments on PM ISA and REA (2009)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009. Review of EPA's Integrated Science Assessment for Particulate Matter (First External Review Draft, December 2008). EPA-COUNCIL-09-008. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf>.

Pg 9: "There is an appropriate discussion of the time-series studies, but this section needs to have an explicit finding that the evidence supports a relationship between PM and mortality that is seen in these studies. This conclusion should be followed by the discussion of statistical methodology and the identification of any threshold that may exist."

U.S. Environmental Protection Agency Science Advisory Board (U.S. EPA-SAB). 2009. Consultation on EPA's Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment. EPA-COUNCIL-09-009. May. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/723FE644C5D758DF852575BD00763A32/$File/EPA-CASAC-09-009-unsigned.pdf>.

Pg 6: "On the issue of cut-points raised on 3-18, the authors should be prepared to offer a scientifically cogent reason for selection of a specific cut-point, and not simply try different cut-points to see what effect this has on the analysis. The draft ISA was clear that there is little evidence for a population threshold in the C-R function."

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2009. Review of *Integrated Science Assessment for Particulate Matter (Second External Review Draft, July 2009)*. EPA-CASAC-10-001. November. Available on the Internet at <http://yosemite.epa.gov/sab/SABPRODUCT.NSF/81e39f4c09954fcb85256ead006be86e/151B1F83B0231455852576780068 36B9/$File/EPA-CASAC-10-001-unsigned.pdf>.

Pg 2: "The paragraph on lines 22-30 of page 2-37 is not clearly written. Twice in succession it states that the use of a no-threshold log-linear model is supported, but then cites other studies that suggest otherwise. It would be good to revise this paragraph to more clearly state – well, I'm not sure what. Probably that more research is needed."

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Dr. Donna Kenski, Data Analysis Director, Lake Michigan Air Directors Consortium, Rosemont, IL

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E. Krewski et al. (2009)

Krewski, Daniel, Michael Jerrett, Richard T. Burnett, Renjun Ma, Edward Hughes, Yuanli Shi, Michelle C. Turner, C. Arden Pope III, George Thurston, Eugenia E. Calle, and Michael J. Thun with Bernie Beckerman, Pat DeLuca, Norm Finkelstein, Kaz Ito, D.K. Moore, K. Bruce Newbold, Tim Ramsay, Zev Ross, Hwashin Shin, and Barbara Tempalski. (2009). Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. *HEI Research Repor*t, 140, Health Effects Institute, Boston, MA.

Pg 119: [About Pope et al. (2002)] "Each 10-μg/m^3 increase in long-term average ambient $PM_{2.5}$ concentrations was associated with approximately a 4%, 6%, or 8% increase in risk of death from all causes, cardiopulmonary disease, and lung cancer, respectively. There was no evidence of a threshold exposure level within the range of observed $PM_{2.5}$ concentrations. "

Krewski (2009). Letter from Dr. Daniel Krewski to HEI's Dr. Kate Adams (dated July 7, 2009) regarding "EPA queries regarding HEI Report 140". Dr. Adams then forwarded the letter on July 10, 2009 to EPA's Beth Hassett-Sipple. (letter placed in docket #EPA-HQ-OAR-2007-0492).

Pg 4: "6. The Health Review Committee commented that the Updated Analysis completed by Pope et al. 2002 reported "no evidence of a threshold exposure level within the range of observed $PM_{2.5}$ concentrations" (p. 119). In the Extended Follow-Up study, did the analyses provide continued support for a no-threshold response or was there evidence of a threshold?

"Response: As noted above, the HEI Health Review Committee commented on the lack of evidence for a threshold exposure level in Pope et al. (2002) with follow-up through the year 1998. The present report, which included follow-up through the year 2000, also does not appear to demonstrate the existence of a threshold in the exposure-response function within the range of observed $PM_{2.5}$ concentrations."

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Dr. Lianne Sheppard, PhD, Professor, Department of Biostatistics, University of Washington

F. Schwartz et al. (2008)

Schwartz J, Coull B, Laden F. (2008). The Effect of Dose and Timing of Dose on the Association between Airborne Particles and Survival. *Environmental Health Perspectives*. 116: 64-69.

Pg 67: "A key finding of this study is that there is little evidence for a threshold in the association between exposure to fine particles and the risk of death on follow-up, which continues well below the U.S. EPA standard of 15 $\mu g/m^3$."

Pg 68: "In conclusion, penalized spline smoothing and model averaging represent reasonable, feasible approaches to addressing questions of the shape of the exposure–response curve, and can provide valuable information to decisionmakers. In this example, both approaches are consistent, and suggest that the association of particles with mortality has no threshold down to close to background levels."

G. Expert Elicitation on PM-Mortality (2006, 2008)

Industrial Economics, Inc., 2006. *Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between $PM_{2.5}$ Exposure and Mortality*. Prepared for the U.S.EPA, Office of Air Quality Planning and Standards, September. Available on the Internet at <http://www.epa.gov/ttn/ecas/regdata/Uncertainty/pm_ee_report.pdf>.

Pg v: "Each expert was given the option to integrate their judgments about the likelihood of a causal relationship and/or threshold in the C-R function into his distribution or to provide a distribution "conditional on" one or both of these factors."

Pg vii: "Only one of 12 experts explicitly incorporated a threshold into his C-R function.[3] The rest believed there was a lack of empirical and/or theoretical support for a population threshold. However, three other experts gave differing effect estimate distributions above and below some cut-off concentration. The adjustments these experts made to median estimates and/or uncertainty at lower $PM^{2.5}$ concentrations were modest."

> "[3] Expert K indicated that he was 50% sure that a threshold existed. If there were a threshold, he thought that there was an 80% chance that it would be less than or equal to 5 $\mu g/m^3$, and a 20% chance that it would fall between 5 and 10 $\mu g/m^3$."

Pg ix: "Compared to the pilot study, experts in this study were in general more confident in a causal relationship, less likely to incorporate thresholds, and reported higher mortality effect estimates. The differences in results compared with the pilot appear to reflect the influence of new research on the interpretation of the key epidemiological studies that were the focus of both elicitation studies, more than the influence of changes to the structure of the protocol."

Pg 3-25: "3.1.8 THRESHOLDS
The protocol asked experts for their judgments regarding whether a threshold exists in the $PM_{2.5}$ mortality C-R function. The protocol focused on assessing expert judgments regarding theory and evidential support for a population threshold (i.e., the concentration below which no member of the study population would experience an increased risk of death).[32] If an expert wished to incorporate a threshold in his characterization of the concentration-response relationship, the team then asked the expert to specify the threshold $PM_{2.5}$ concentration probabilistically, incorporating his uncertainty about the true threshold level.

"From a theoretical and conceptual standpoint, all experts generally believed that individuals exhibit thresholds for PM-related mortality. However, 11 of them discounted the idea of a population threshold in the C-R function on a theoretical and/or empirical basis. Seven of these experts noted that theoretically one would be unlikely to observe a population threshold due to the variation in susceptibility at any given time in the study population resulting from combinations of genetic, environmental, and socioeconomic factors.[33] All 11 thought that there was insufficient empirical support for a population threshold in the C-R function. In addition, two experts (E and L) cited analyses of the ACS cohort data in Pope et al. (2002) and another (J) cited Krewski et al. (2000a & b) as supportive of a linear relationship in the study range.

"Seven of the experts favored epidemiological studies as ideally the best means of addressing the population threshold issue, because they are best able to evaluate the full range of susceptible individuals at environmentally relevant exposure levels. However, those who favored epidemiologic studies generally acknowledged that definitive studies addressing thresholds would be difficult or impossible to conduct, because they would need to include a very large and diverse population with wide variation in exposure and a long follow-up period. Furthermore, two experts (B and I) cited studies documenting difficulties in detecting a threshold using epidemiological studies (Cakmak et al. 1999, and Brauer et al., 2002, respectively). The experts generally thought that clinical and toxicological studies are best suited for researching mechanisms and for addressing thresholds in very narrowly defined groups. One expert, B, thought that a better understanding of the detailed biological mechanism is critical to addressing the question of a threshold.

"One expert, K, believed it was possible to make a conceptual argument for a population threshold. He drew an analogy with smoking, indicating that among heavy smokers, only a proportion of them gets lung cancer or demonstrates an accelerated decline in lung function. He thought that the idea that there is no level that is biologically safe is fundamentally at odds with toxicological theory. He did not think that a population threshold was detectable in the currently available epidemiologic studies. He indicated that some of the cohort studies showed greater uncertainty in the shape of the C-R function at lower levels, which could be indicative of a threshold.

"Expert K chose to incorporate a threshold into his C-R function. He indicated that he was 50% sure that a threshold existed. If there were a threshold, he thought that there was an 80% chance that it would be less than or equal to 5 $\mu g/m^3$, and a 20% chance that it would fall between 5 and 10 $\mu g/m^3$."

Roman, Henry A., Katherine D. Walker, Tyra L. Walsh, Lisa Conner, Harvey M. Richmond, Bryan J. Hubbell, and Patrick L. Kinney. (2008). "Expert Judgment Assessment of the Mortality Impact of Changes in Ambient Fine Particulate Matter in the U.S." ***Environ. Sci. Technol.*****, 42(7):2268-2274.**

Pg 2271: "Eight experts thought the true C-R function relating mortality to changes in annual average $PM_{2.5}$ was log-linear across the entire study range (ln(mortality)) $\beta \times$ PM). Four experts (B, F, K, and L) specified a "piecewise" log-linear function, with different β coefficients for PM concentrations above and below an expert-specified break point. This approach allowed them to express increased uncertainty in mortality effects seen at lower concentrations in major epidemiological studies. Expert K thought the relationship would be log-linear above a threshold."

Pg 2271: "Expert K also applied a threshold, T, to his function, which he described probabilistically. He specified $P(T > 0) = 0.5$. Given $T > 0$, he indicated $P(T \leq 5\ \mu g/m^3) = 0.8$ and $P(5\ \mu g/m^3 < T \leq 10\ \mu g/m^3) = 0.2$. Figure 3 does not include the impact of applying expert K's threshold, as the size of the reduction in benefits will depend on the distribution of baseline PM levels in a benefits analysis."

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Dr. Dan Krewski, University of Ottawa

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Dr. Morton Lippmann, Professor, Nelson Institute of Environmental Medicine, New York University School of Medicine, Tuxedo, NY

Dr. Joe Mauderly, Lovelace Respiratory Research Institute

Dr. Bart Ostro, Chief, Air Pollution Epidemiology Unit, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland, CA

Dr. Arden Pope, Professor, Department of Economics, Brigham Young University, Provo, UT

Dr. Richard Schlesinger, Pace University

Dr. Joel Schwartz, Harvard School of Public Health

Dr. George Thurston—Department of Environmental Medicine, NYU, Tuxedo, NY

Dr. Mark Utell, University of Rochester School of Medicine and Dentistry

H. CASAC comments on PM Staff Paper (2005)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2005. EPA's Review of the National Ambient Air Quality Standards for Particulate Matter (Second Draft PM Staff Paper, January 2005). EPA-SAB-CASAC-05-007. June. Available on the Internet at <http://yosemite.epa.gov/sab/sabproduct.nsf/E523DD36175EB5AD8525701B007332AE/$File/SAB-CASAC-05-007_unsigned.pdf>.

Pg 6: "A second concern is with methodological issues. The issue of the selection of concentration-response (C-R) relationships based on locally-derived coefficients needs more discussion. The Panel did not agree with EPA staff in calculating the burden of associated incidence in their risk assessment using either the predicted background or the lowest measured level (LML) in the utilized epidemiological analysis. The available epidemiological database on daily mortality and morbidity does not establish either the presence or absence of threshold concentrations for adverse health effects. Thus, in order to avoid emphasizing an approach that assumes effects that extend to either predicted background concentrations or LML, and to standardize the approach across cities, for the purpose of estimating public health impacts, the Panel favored the primary use of an assumed threshold of 10 $\mu g/m^3$. The original approach of using background or LML, as well as the other postulated thresholds, could still be used in a sensitivity analysis of threshold assumptions.

"The analyses in this chapter highlight the impact of assumptions regarding thresholds, or lack of threshold, on the estimates of risk. The uncertainty associated with threshold or nonlinear models needs more thorough discussion. A major research need is for more work to determine the existence and level of any thresholds that may exist or the shape of nonlinear concentration-response curves at low levels of exposure that may exist, and to reduce uncertainty in estimated risks at the lowest PM concentrations."

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I. HES Comments on 812 Analysis (2004)

U.S. Environmental Protection Agency - Science Advisory Board (U.S. EPA-SAB). 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis – Benefits and Costs of the Clean Air Act, 1990-2020. Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis. EPA-SAB-COUNCIL-ADV-04-002. March. Available on the Internet at <http://yosemite.epa.gov/sab%5CSABPRODUCT.NSF/08E1155AD24F871C85256E5400433D5D/$File/council_adv_04002.pdf>.

Pg 20: "The Subcommittee agrees that the whole range of uncertainties, such as the questions of causality, shape of C-R functions and thresholds, relative toxicity, years of life lost, cessation lag structure, cause of death, biologic pathways, or susceptibilities may be viewed differently for acute effects versus long-term effects.

"For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between $PM_{2.5}$ and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3 and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

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J. NRC – Committee on Estimating the Health Risk Reduction Benefits of Proposed Air Pollution Regulations (2002)

National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

Pg 109: "**Linearity and Thresholds**

"The shape of the concentration-response functions may influence the overall estimate of benefits. The shape is particularly important for lower ambient air pollution concentrations to which a large portion of the population is exposed. For this reason, the impact of the existence of a threshold may be considerable.

"In epidemiological studies, air pollution concentrations are usually measured and modeled as continuous variables. Thus, it may be feasible to test linearity and the existence of thresholds, depending on the study design. In time-series studies with the large number of repeated measurements, linearity and thresholds have been formally addressed with reasonable statistical power. For pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold. For example, examination of the mortality effects of short-term exposure to PM_{10} in 88 cities indicates that the concentration-response functions are not due to the high concentrations and that the slopes of these functions do not appear to increase at higher concentrations (Samet et al. 2000). Many other mortality studies have examined the shape of the concentration-response function and indicated that a linear (nonthreshold) model fit the data well (Pope 2000). Furthermore, studies conducted in cities with very low ambient pollution concentrations have similar effects per unit change in concentration as those studies conducted in cities with higher concentrations. Again, this finding suggests a fairly linear concentration-response function over the observed range of exposures.

"Regarding the studies of long-term exposure, Krewski et al. (2000) found that the assumption of a linear concentration-response function for mortality outcomes was not unreasonable. However, the statistical power to assess the shape of these functions is weakest at the upper and lower end of the observed exposure ranges. Most of the studies examining the effects of long-term exposure on morbidity compare subjects living in a small number of communities (Dockery et al. 1996; Ackermmann-Liebrich 1997; Braun-Fahrländer et al. 1997). Because the number of long-term effects studies are few and the number of communities studied is relatively small (8 to 24), the ability to test formally the absence or existence of a no-effect threshold is not feasible. However, even if thresholds exist, they may not be at the same concentration for all health outcomes.

"A review of the time-series and cohort studies may lead to the conclusion that although a threshold is not apparent at commonly observed concentrations, one may exist at lower levels. An important point to acknowledge regarding thresholds is that for health benefits analysis a key threshold is the population threshold (the lowest of the individual thresholds). However, the population threshold would be very difficult to observe empirically through epidemiology, because epidemiology integrates information from very large groups of people (thousands). Air pollution regulations affect even larger groups of people (millions). It is reasonable to assume that among such large groups susceptibility to air pollution health

effects varies considerably across individuals and depends on a large set of underlying factors, including genetic makeup, age, exposure measurement error, preexisting disease, and simultaneous exposures from smoking and occupational hazards. This variation in individual susceptibilities and the resulting distribution of individual thresholds underlies the concentration-response function observed in epidemiology. Thus, until biologically based models of the distribution of individual thresholds are developed, it may be productive to assume that the population concentration-response function is continuous and to focus on finding evidence of changes in its slope as one approaches lower concentrations.

7.1.1.1 EPA's Use of Thresholds

"In EPA's benefits analyses, threshold issues were discussed and interpreted. For the PM and ozone National Ambient Air Quality Standards (NAAQS), EPA investigated the effects of a potential threshold or reference value below which health consequences were assumed to be zero (EPA 1997). Specifically, the high-end benefits estimate assumed a 12-microgram per cubic meter ($\mu g/m^3$) mean threshold for mortality associated with long-term exposure to $PM_{2.5}$. The low-end benefits estimate assumed a 15-$\mu g/m^3$ threshold for all PM-related health effects. The studies, however, included concentrations as low as 7.5 $\mu g/m^3$. For the Tier 2 rule and the HD engine and diesel-fuel rule, no threshold was assumed (EPA 1999, 2000). EPA in these analyses acknowledged that there was no evidence for a threshold for PM.

"Several points should be noted regarding the threshold assumptions. If a threshold is assumed where one was not apparent in the original study, then the data should be refit and a new curve generated with the assumption of a zero slope over a segment of the concentration-response function that was originally found to be positively sloped. The assumption of a zero slope over a portion of the curve will force the slope in the remaining segment of the positively sloped concentration-response function to be greater than was indicated in the original study. A new concentration-response function was not generated for EPA's benefits analysis for the PM and ozone NAAQS for which threshold assumptions were made. The generation of the steeper slope in the remaining portion of the concentration-response function may fully offset the effect of assuming a threshold. These aspects of assuming a threshold in a benefits analysis where one was not indicated in the original study should be conveyed to the reader. The committee notes that the treatment of thresholds should be evaluated in a consistent and transparent framework by using different explicit assumptions in the formal uncertainty analyses (see Chapter 5)."

Pg 117: "Although the assumption of no thresholds in the most recent EPA benefits analyses was appropriate, EPA should evaluate threshold assumptions in a consistent and transparent framework using several alternative assumptions in the formal uncertainty analysis."

Pg 136: "Two additional illustrative examples are thresholds for adverse effects and lag structures.[2] EPA considers implausible any threshold for mortality in the particulate matter (PM) exposure ranges under consideration (EPA 1999a, p. 3-8). Although the agency conducts sensitivity analyses incorporating thresholds, it provides no judgment as to their relative plausibility. In a probabilistic uncertainty analysis, EPA could assign appropriate weights to various threshold models. For PM-related mortality in the Tier 2 analysis, the committee expects that this approach would have resulted in only a slight widening of the probability distribution for avoided mortality and a slight reduction in the mean of that distribution, thus reflecting EPA's views about the implausibility of thresholds. The committee finds that such formal incorporation of EPA's expert judgments about the plausibility of thresholds into its primary analysis would have been an improvement.

"Uncertainty about thresholds is a special aspect of uncertainty about the shape of concentration-response functions. Typically, EPA and authors of epidemiological studies assume that these functions are linear

on some scale. Often, the scale is a logarithmic transformation of the risk or rate of the health outcome, but when a rate or risk is low, a linear function on the logarithmic scale is approximately linear on the scale of the rate or risk itself. Increasingly, epidemiological investigators are employing analytic methods that permit the estimation of nonlinear shapes for concentration-response functions (Greenland et al. 1999). As a consequence, EPA will need to be prepared to incorporate nonlinear concentration-response functions from epidemiological studies into the agency's health benefits analyses. Any source of error or bias that can distort an epidemiological association can also distort the shape of an estimated concentration -response function, as can variation in individual susceptibility (Hattis and Burmaster 1994; Hattis et al. 2001)."

Pg 137: "In principle, many components of the health benefits model need realistic probabilistic models (see Table 5-1 for a listing of such components), in addition to concentration-response thresholds and time lags between exposure and response. For example, additional features of the concentration-response function—such as projection of the results from the study population to the target populations (which may have etiologically relevant characteristics outside the range seen in the study population) and the projection of baseline frequencies of morbidity and mortality into the future—must be characterized probabilistically. Other uncertainties that might affect the probability distributions are the estimations of population exposure (or even concentration) from emissions, estimates of emissions themselves, and the relative toxicity of various classes of particles. Similarly, many aspects of the analysis of the impact of regulation on ambient concentrations and on population exposure involve considerable uncertainty and, therefore, may be beneficially modeled in this way. Depending on the analytic approach used, joint probability distributions will have to be specified to incorporate correlations between model components that are structurally dependent upon each other, or the analysis will have to be conducted in a sequential fashion that follows the model for the data-generating process.

"EPA should explore alternative options for incorporating expert judgment into its probabilistic uncertainty analyses. The agency possesses considerable internal expertise, which should be employed as fully as possible. Outside experts should also be consulted as needed, individually or in panels. In all cases, when expert judgment is used in the construction of a model component, the experts should be identified and the rationales and empirical bases for their judgments should be made available."

NRC members

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