In the Matter of:

THE APPLICATION OF LOUISVILLE GAS AND ELECTRIC COMPANY FOR CERTIFICATES OF PUBLIC CONVENIENCE AND NECESSITY AND APPROVAL OF ITS 2011 COMPLIANCE PLAN FOR RECOVERY BY ENVIRONMENTAL SURCHARGE	)	CASE NO. 2011-00162

Response to the Commission Staff's First Information Request dated July 12, 2011

One Paper Copy for Question No. 53(a)

Filed – September 23, 2011

LOUISVILLE GAS AND ELECTRIC COMPANY

Response to Commission Staff's First Information Request Dated July 12, 2011

Case No. 2011-00162

Question No. 53

Witness: John N. Voyles, Jr.

Q-53. Refer to LG&E Application, paragraph 13. A statement is made that "Building these Particulate Matter Control Systems is the most cost-effective means of complying with the HAPs Rule." Is this an industry-wide position or specific to the LG&E fleet?

a. If this is an industry position, provide the study/work papers which support this statement.

b. If LG&E specific, provide a summary of the support for this position.

A-53. The Companies' position is both an LG&E/KU position and an industry position. As discussed in Exhibit JNV-2, Pulse Jet Fabric Filters (PJFF) in combination with Powdered Activated Carbon (PAC) Injection systems are an effective way to meet particulate control regulations but also allows for compliance with the pending HAPs rule based on the coal specifications that the LG&E boilers are designed to utilize. To determine how cost-effective a particular compliance strategy will be first requires determining the appropriate technologies and then to assessing the constructability issues along with any balance-of-plant impacts associated with implementing those technologies. As Black and Veatch have engineering expertise in the suite of available technologies and familiarity with our unit design, they were selected to conduct studies throughout our fleet. Their recommendations to the Companies were based on which technologies would comply with EPA regulations and would be most cost effective based on their industry experiences as well as the results of their assessment of our fleet. Please reference the Black and Veatch reports for additional information.

The EPA's analyses on the Utility MACT regulation's impact on coal-fired generation states an expected 166 GW of coal fired units throughout the U.S. will be retrofitted with fabric filter technology. Please see the attached excerpt from the EPA's report titled "Regulatory Impact Analysis of the Proposed Toxics Rule" dated March 2011 that includes projection materials regarding the installation of PJFF technology throughout the industry. The full report is included on the CD in the folder titled Question 53.

March 2011

Regulatory Impact Analysis of the Proposed Toxics Rule:

Final Report

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Acronyms and Abbreviations

ACI	activated carbon injection
ACS	American Cancer Society
ADHD	attention deficit hyperactivity disorder
ADI	acceptable daily intake
ADP	adenosine diphosphate
AERMOD	American Meteorological Society/EPA Regulatory Model
AHRQ	Agency for Healthcare Research and Quality
AMI	acute myocardial infarction
ANL	Argonne National Laboratory
ASA	American Sportfishing Association
atm	atmosphere
ATSDR	Agency for Toxic Substance and Disease Registry
BAF	bioaccumulation factor
BASS	Bioaccumulation and Aquatic System Simulator
BC	boundary conditions
BEA	Bureau of Economic Analysis
BenMAP	Benefits Mapping and Analysis Program
BLS	Bureau of Labor Statistics
BMD	benchmark dose
BMDL	BMD lower statistical confidence limit
BMI	body mass index
BMR	benchmark response
BOC	Bureau of Census
C-R	concentration-response
CAAA	Clean Air Act Amendments
CAIR	Clean Air Interstate Rule
CAMR	Clean Air Mercury Rule
CDF	Cumulative Distribution Function
CEEPR	Center for Energy and Environmental Policy Research
cfs	cubic feet per second
CH3Hg	monomethyl mercury
CHD	coronary heart disease
CI	confidence interval
cm	centimeter
CMAQ	Community Multi-Scale Air Quality
CPS	Current Population Survey
CRDM	Climatological Regional Dispersion Model
CRF	capital recovery factor
CRST	Cheyenne River Sioux Tribal
CSFII	Continuing Survey of Food Intake by Individuals
CVD	cardiovascular disease
D-MCM	Dynamic Mercury Cycling Model
DEP	Department of Environmental Protection

DHHS	Department of Health and Human Services
DDT	dichloro-diphenyl-trichloroethane
DHA	docosahexaenoic acid
DOC	dissolved organic carbon
DOE	Department of Energy
DOI	Department of the Interior
DOM	dissolved organic matter
DPA	docosapentaenoic acid
E-MCM	Everglades Mercury Cycling Model
ECG	electrocardiogram
EFH	Exposure Factors Handbook
EGRID	Emissions & Generated Resource Integrated Database
EGU	electric generating unit
EIA	Economic Impact Analysis
EIA	Energy Information Administration
EKG	electrocardiogram
ELA	Experimental Lakes Area
EMF	emission modification factor
EMMA	Environmental Monitoring and Measurement Advisor
E.O.	Executive Order
EPA	Environmental Protection Agency
EPRI	Electric Power Research Institute
ESP	electrostatic precipitator
EU	European Union
EURAMIC	The European Multicenter Case Control Study on Antioxidants, Myocardial Infarction and Cancer of the Breast
EXAMS2	Exposure Analysis Modeling System, Version 2
F	Fahrenheit
FAO	Food and Agriculture Organization
FDA	Food and Drug Administration
FERC	Federal Energy Regulatory Commission
FF	fabric filters
FGD	flue gas desulfurization
FGETS	Food and Grill Exchange of Toxic Substances
FL	fork length
ft	feet
g	gram
GDP	gross domestic product
GIS	Geographic Information System
GRU	Gainesville Regional Utilities
GW	gigawatt
GWh	gigawatt hours
H-PAC	Hazard Prediction and Assessment Capability
HDL	high-density lipoprotein
Hg	mercury
Hg_0	elemental mercury

Hg(II)	inorganic divalent mercury
HgCl2	mercuric chloride
HgP	particulate mercury
HgT	total mercury
hrs	hours
HUC	hydrologic unit code
HYSPLIT	Hybrid Single Particle Lagrangian Integrated Trajectory
ICR	Information Collection Request
IEM-2M	Indirect Exposure Model, Version 2
IGCC	Integrated Gasification Combined Cycle
IMT	intima-media thickness
in	inch
IOM	Institute of Medicine
IOU	investor-owned utility
IQ	intelligence quotient
IPM	Integrated Planning Model
IRIS	Integrated Risk Information System
ISC3	Industrial Source Complex
K	Kelvin
kg	kilogram
KIHD	Kuopio Ischemic Heart Disease
km	kilometer
kWh	kilowatt hour
lb	pound
lbs	pounds
L	liter
LC50	lethal concentration for 50% percent of the population
LC	omega-3 PUFA long chain omega-3 polyunsaturated fatty acids
LDL	low-density lipoprotein
m	meter
M	molar mass
mm	millimeter
MACT	Maximum Achievable Control Technology
MAS/MILS	Mineral Availability System/Mineral Industry Location System
MCM	Mercury Cycling Model
MDN	Mercury Deposition Network
ME	Midwest/Northeast
MeHg	methylmercury
METAALICUS	Mercury Experiment to Assess Atmospheric Loading in Canada and the United States
mg	milligram
MI	myocardial infarction
MIT	Massachusetts Institute of Technology
MM5	Mesoscale Model
MMAPS	Mercury Maps
mmBtu	million British thermal units

mol	mole
MRFSS	Marine Recreational Fishing Statistical Survey
MRL	minimal risk level
MW	megawatt
MWC	municipal waste combustor
MWh	megawatt hour
MWI	medical waste incinerator
NAAQS	National Ambient Air Quality Standards
NAICS	North American Industry Classification System
NAS	National Academy of Sciences
NASS	National Agriculture Statistics Service
NDMMF	National Descriptive Model of Mercury in Fish
NEI	National Emissions Inventory
NERC	North American Electric Reliability Council
NES	Neurobehavioral Evaluation System
NFTS	National Fish Tissue Survey
ng	nanogram
NHD	National Hydrography Database
NHANES	National Health and Nutrition Examination Survey
NH3	ammonia
NLCD	National Land Cover Data
NLFA	National Listing of Fish and Wildlife Advisories
NLSY	National Longitudinal Study of Youth
NLFTS	National Lake Fish Tissue Survey
NMFS	National Marine Fisheries Service
NOAA	National Oceanic and Atmospheric Administration
NOAEC	No Observable Adverse Effects Concentration
NOAEL	No Observed Adverse Effect Level
NODA	Notice of Data Availability
NOx	nitrogen oxides
NPR	Notice of Proposed Rulemaking
NPV	net present value
NRC	National Research Council
NRS	National Recreation Survey
NSFHWR	National Survey of Fishing, Hunting and Wildlife-Associated Recreation
NSPS	New Source Performance Standards
NSR	New Source Review
NSRE	National Survey on Recreation and the Environment
NW	Northwest
O&M	operation and maintenance
OM	organic matter
OMB	Office of Management and Budget
OR	odds ratio
ORD	Office of Research and Development
P-PUFA	plasma polyunsaturated fatty acids
PAC	powder activated carbon

PCB	polychlorinated biphenyl
PCS	Permit Compliance System
pH	potential of hydrogen
PM	particulate matter
POTW	Publicly Owned Treatment Works
ppb	parts per billion
ppm	parts per million
RARE	Regional Applied Research Effort
RELMAP	Regional Lagrangian Model of Air Pollution
REMI	Regional Economic Models, Inc.
REMSAD	Regulatory Modeling System for Aerosols and Deposition
RFA	Regulatory Flexibility Act
RfD	Reference Dose
RFF	Resources for the Future
RGM	Reactive Gaseous Mercury
RIA	Regulatory Impact Analysis
RNA	ribonucleic acid
RQ	risk quotient
RR	relative risk
RtC	Report to Congress
RUSLE	Revised Universal Soil Loss Equation
SAB-HES	Science Advisory Board Health Effects Subgroup
SBA	Small Business Administration
SBREFA	Small Business Regulatory Enforcement Fairness Act
SCR	selective catalytic reduction
SD	standard deviation
SE	Southeast
sec	second
SIC	standard industrial classification
SIP	state implementation plan
SMR	standard mortality rate
SNCR	selective non-catalytic reduction
SO2	sulfur dioxide
sq km	square kilometer
SRB s	ulfate reducing bacteria
st dev	standard deviation
SW	Southwest
SWAT	Soil and Water Assessment Tool
TDI	tolerable daily intake
TL	total length
TMDL	Total Maximum Daily Load
TOLD-SL	Test of Language Development - Spoken Language
TRUM	Technology, Retrofit and Upgrading Model
TSD	Technical Support Document
UF	uncertainty factor
ug	microgram

uM	micromole
um	micrometer
UMRA	Unfunded Mandates Reform Act
U.S.	United States
USACE	United States Army Corps of Engineers
U.S.C.	United States Code
USDA	United States Department of Agriculture
USEPA	United States Environmental Protection Agency
USFWS	United States Fish and Wildlife Service
USGS	United States Geological Survey
UV-B	ultraviolet light, type B
VMI	Visual-Motor Integration
VSL	Value of Statistical Life
WASP	Water Quality Analysis Simulation Program
WCS	Watershed Characterization System
WHO	World Health Organization
WISC	Wechsler Intelligence Scales for Children
WISC-III	Wechsler Intelligence Scales for Children administered in the Seychelles Islands
WISC-R	Wechsler Intelligence Scales for Children administered in New Zealand and the Faroe Islands
WRAML	Wide-Range Assessment of Memory Learning
WTP	willingness to pay

Chapter 1
EXECUTIVE SUMMARY

This Regulatory Impact Analysis (RIA) presents the health and welfare benefits, costs, and other impacts of the proposed Toxics Rule (the Utility MACT and NSPS proposals) in 2016.

1.1 Key Findings

This proposed rule will reduce emissions of Hazardous Air Pollutants (HAP) including mercury from the electric power industry. As a co-benefit, the emissions of certain $PM_{2.5}$ precursors such as SO_2 will also decline. EPA estimates that this proposed rule will yield annual monetized benefits (in 2007$) of between $59 to $140 billion using a 3% discount rate and $53 and $130 billion using a 7% discount rate. The great majority of the estimates are attributable to co-benefits from reductions in $PM_{2.5}$-related mortality. The annual social costs are $10.9 billion (2007$) and the annual quantified net benefits are $48 to $130 billion using 3% discount rate or $42 to $120 billion using a 7% discount rate. The benefits outweigh costs by between 5 to 1 or 13 to 1 depending on the benefit estimate and discount rate used. The co-benefits are substantially attributable to the 6,800 to 17,000 fewer $PM_{2.5}$-related premature mortalities. There are some costs and important benefits that EPA could not monetize, such as those for the HAP being reduced by this proposed rule other than mercury. Upon considering these limitations and uncertainties, it remains clear that the benefits of the proposed Toxics Rule are substantial and far outweigh the costs. The annualized private compliance costs to the power industry in 2015 are $10.9 billion (2007$). Employment impacts associated with the proposed rule are estimated to be small. Effective policies to support end-use energy efficiency investments can reduce compliance costs and lessen impacts on electric rates and bills. In 2015, annualized private compliance costs to the industry are reduced by $0.3 billion (2007$) under an illustrative energy efficiency scenario.[1]

The benefits and costs in 2016 of the proposed rule are in Table 1-1.

[1] This is based on the illustrative energy efficiency sensitivity analysis discussed in Section 8.13 and Appendix D.

Table 1-1. Summary of EPA's Estimates of Benefits, Costs, and Net Benefits of the Proposed Toxics Rule in 2016[a] (billions of 2007$)

Description	Estimate (3% Discount Rate)	Estimate (7% Discount Rate)
Social costs[b]	$10.9	$10.9
Social benefits[c,d]	$59 to $140 + B	$53 to $130 + B
Net benefits (benefits-costs)	$48 to $130	$42 to $120

[a] All estimates are rounded to two significant digits and represent annualized benefits and costs anticipated for the year 2016. For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. A listing of health and welfare effects is provided in Table 1-5. Estimates here are subject to uncertainties discussed further in the body of the document.

[b] The reduction in premature mortalities account for over 90% of total monetized benefits. Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure described in Chapter 6. Results reflect 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (U.S. EPA, 2000; OMB, 2003).

[c] Social costs are estimated using the MultiMarket model, the model employed by EPA in this RIA to estimate economic impacts of the proposal to industries outside the electric power sector. This model does not estimate indirect impacts associated with a regulation such as this one. Details on the social cost estimates can be found in Chapter 9 and Appendix E of this RIA.

[d] Potential benefit categories that have not been quantified and monetized are listed in Table 1-5.

1.1.1 Health Benefits

The proposed Toxics Rule is expected to yield significant health benefits by reducing emissions not only of HAP such as mercury, but also significant co-benefits due to reductions in direct fine particles and in two key contributors to fine particle formation. Sulfur dioxide contributes to the formation of fine particle pollution ($PM_{2.5}$), and nitrogen oxide contributes to the formation of $PM_{2.5}$.

Our analyses suggest this rule would yield benefits in 2016 of $59 to $140 billion (based on a 3 percent discount rate) and $53 to $130 billion (based on a 7 percent discount rate). This estimate reflects the economic value of a range of avoided health outcomes, including 510 fewer mercury-related IQ points lost as well as a variety of avoided $PM_{2.5}$-related impacts, including 6,800 to 17,000 premature deaths, 11,000 nonfatal heart attacks, 5,300 hospitalizations for respiratory and cardiovascular diseases, 850,000 lost work days and 5.1 million days when adults restrict normal activities because of respiratory symptoms exacerbated by $PM_{2.5}$. This rule is also likely to produce significant ozone-related benefits, which we were unable to quantify in the RIA

due to the limitations of the scaling approach used to estimate benefits; further details may be found in the benefits chapter.

We also estimate substantial additional health improvements for children from reductions in upper and lower respiratory illnesses, acute bronchitis, and asthma attacks. See Table 1-2 for a list of the annual reduction in health effects expected in 2016 and Table 1-3 for the estimated value of those reductions.

We also include in our monetized benefits estimates the effect from the reduction in CO_2 emissions that is an outcome of this proposal. We calculate the benefits associated with these emission reductions using the social cost of carbon (SCC) approach, an approach that has been used to estimate such benefits in several recent rulemakings (e.g., proposed Transport Rule, final industrial boilers major and source area sources rules).

1.1.2 Welfare Benefits

The term *welfare benefits* covers both environmental and societal benefits of reducing pollution, such as reductions in damage to ecosystems, improved visibility and improvements in recreational and commercial fishing, agricultural yields, and forest productivity.

Table 1-2. Estimated Reduction in Incidence of Adverse Health Effects in 2016 for the Proposed Toxics Rule[a,b]

Health Effect	*Eastern U.S.*	*Western U.S.*	*Total*
Mercury-Related endpoints			
IQ Points Lost			510.8
PM-Related endpoints			
Premature death			
Pope et al. (2002) (age >30)	6,700 (1,900—12,000)	120 (33—200)	6,800 (1,900—12,000)
Laden et al. (2006) (age >25)	17,000 (7,900—26,000)	300 (140—470)	17,000 (8,100—27,000)
Infant (< 1 year)	29 (-32—90)	1 (-1—2)	30 (-33—92)
Chronic bronchitis	4,400 (150—8,600)	97 (3—190)	4,500 (150—8,800)
Non-fatal heart attacks (age > 18)	11,000 (2,700—18,000)	190 (48—330)	11,000 (2,700—19,000)
Hospital admissions—respiratory (all ages)	1,600 (650—2,600)	24 (10—39)	1,700 (660—2,600)
Hospital admissions—cardiovascular (age > 18)	3,500 (2,500—4,200)	50 (35—61)	3,600 (2,500—4,200)
Emergency room visits for asthma (age < 18)	6,900 (3,500—10,000)	52 (27—78)	6,900 (3,600—10,000)
Acute bronchitis (age 8-12)	10,000 (-2,300—23,000)	250 (-57—560)	11,000 (-2,400—23,000)
Lower respiratory symptoms (age 7-14)	120,000 (47,000—200,000)	3,000 (1,100—4,800)	130,000 (48,000—200,000)
Upper respiratory symptoms (asthmatics age 9-18)	93,000 (17,000—170,000)	2,300 (420—4,100)	95,000 (18,000—170,000)
Asthma exacerbation (asthmatics 6-18)	110,000 (4,000—380,000)	2,700 (96—9,300)	120,000 (4,100—390,000)
Lost work days (ages 18-65)	830,000 (710,000—960,000)	20,000 (17,000—22,000)	850,000 (720,000—980,000)
Minor restricted-activity days (ages 18-65)	5,000,000 (4,000,000—5,900,000)	110,000 (94,000—140,000)	5,100,000 (4,100,000—6,000,000)

[a] Estimates rounded to two significant figures; column values will not sum to total value.

[b] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

Table 1-3. Estimated Monetary Value of Reductions in Incidence of Health and Welfare for the Proposed Toxics Rule (in billions of 2007$)[a,b,c]

Health Effect		*Eastern U.S.*	*Western U.S.*	*Total*
Avoided IQ Loss Associated with Methylmercury Exposure from Self-Caught Fish Consumption among Recreational Anglers				
3% discount rate				$0.004 - $0.006
7% discount rate				$0.000005 - $0.000009
Adult premature death (Pope et al. 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$53 ($4.2—$160)	$0.9 ($0.1—$2.8)	$54 ($4.3—$160)
7% discount rate	$PM_{2.5}$	$48 ($3.8—$140)	$0.8 ($0. 1—$2.5)	$48 ($3.8—$150)
Adult premature death (Laden et al. 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$140 ($12—$390)	$2.4 ($0.2—$6.9)	$140 ($12—$400)
7% discount rate	$PM_{2.5}$	$120 ($11—$350)	$2.2 ($0.2—$6.3)	$120 ($11—$360)
Infant premature death	$PM_{2.5}$	$0.3 ($-0.3—$1.2)	<$0.01	$0.3 ($-0.3—$1.2)
Chronic Bronchitis	$PM_{2.5}$	$2.1 ($0.1—$9.6)	$0.05 (<$0.01—$0.2)	$2.1 ($0.1—$9.8)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$1.2 ($0.2—$2.9)	$0.02 (<$0.01—$0.05)	$1.2 ($0.2—$2.9)
7% discount rate	$PM_{2.5}$	$1.1 ($0.2—$2.8)	$0.02 (<$0.01—$0.03)	$1.2 ($0.2—$2.9)
Hospital admissions—respiratory	$PM_{2.5}$	<$0.01	<$0.01	$0.02 ($0. 01—$0.03)
Hospital admissions—cardiovascular	$PM_{2.5}$	<$0.01	<$0.01	$0.1 ($0.05—$0.14)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1—$0.1)	<$0.01	$0.1 ($0.1—$0.1)
Minor restricted-activity days	$PM_{2.5}$	$0.3 ($0.2—$0.5)	<$0.01	$0.3 ($0.2—$0.5)
Social cost of carbon (3% discount rate, 2016 value)	CO_2			$0.57

(continued)

Table 1-3. Estimated Monetary Value of Reductions in Incidence of Health and Welfare for the Proposed Toxics Rule (in billions of 2007$)[a,b,c] (continued)

Health Effect	*Eastern U.S.*	*Western U.S.*	*Total*
Monetized total Benefits			
(Pope et al. 2002 $PM_{2.5}$ mortality estimate)			
3% discount rate	$57 ($4.6—$170)	$1 ($0.1—$3.1)	$59 ($4.6—$180)
7% discount rate	$52 ($4.1—$160)	$0.9 ($0.1—$2.8)	$53 ($4.2—$160)
(Laden et al. 2006 $PM_{2.5}$ mortality estimate)			
3% discount rate	$140 ($12—$410)	$2.5 ($0.2—$7.2)	$140 ($12—$410)
7% discount rate	$130 ($11—$370)	$2.2 ($0.2—$6.6)	$130 ($11—$370)

[a] Estimates rounded to two significant figures. The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts. Confidence intervals reflect random sampling error and not the additional uncertainty associated with benefits scaling described above.

[1] The national scale assessment conducted for the RIA focuses on the exposures to methylmercury in populations who consume self-caught freshwater fish (recreational fishers and their families, especially women of child-bearing age). Benefits reflect estimated avoided IQ loss for children, as projected based on fertility rates applied to the women of child-bearing age, among all recreational freshwater anglers in the 48 contiguous U.S. states.

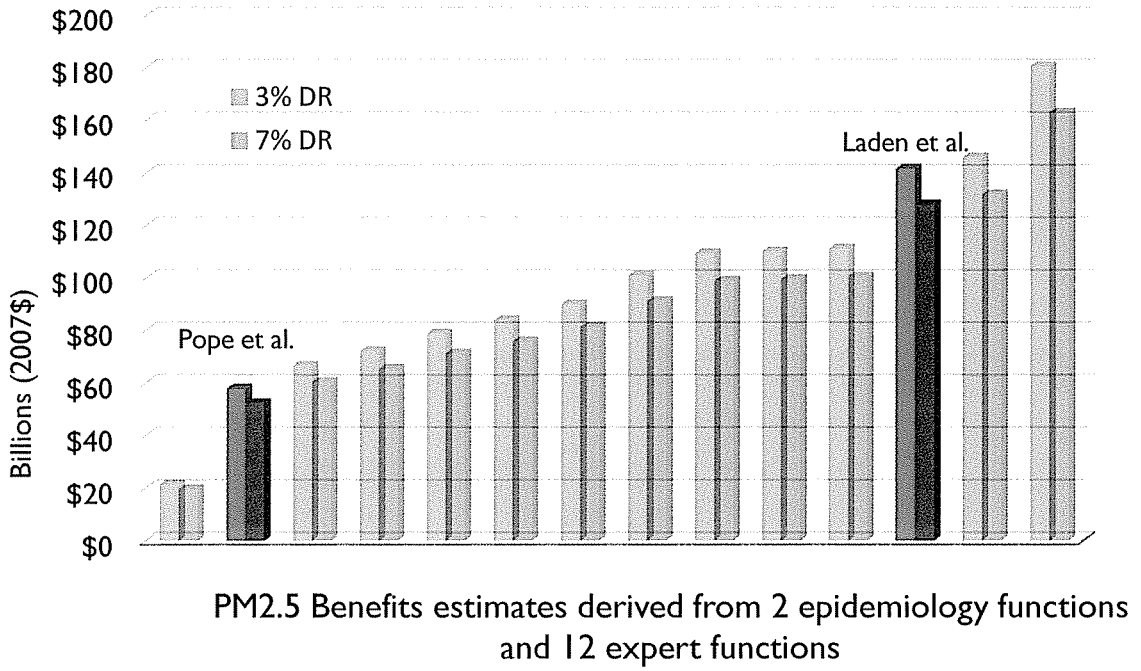
[2]As noted in chapter 5, monetized benefits estimates are for an immediate change in MeHg levels in fish (i.e., the potential lag period associated with fully realizing fish tissue MeHg levels was not reflected in benefits modeling). If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response.

[3]Monetized benefits estimates reported here are for the implementation year: 2016. As such, certain health endpoints that take years to manifest, such as avoided IQ loss from MeHg prenatal exposure, may not be fully quantified in the analysis year.

Figure 1-1 summarizes an array of $PM_{2.5}$-related monetized benefits estimates based on alternative epidemiology and expert-derived PM-mortality estimate.

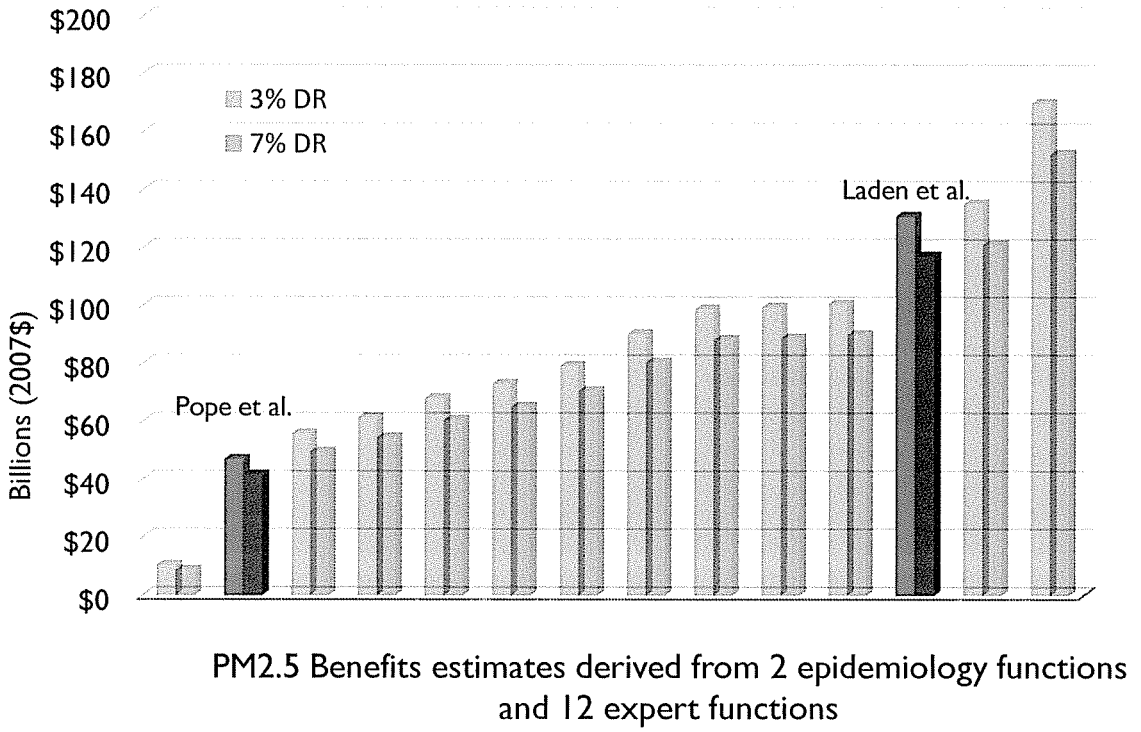
Figure 1-2 summarizes the estimated net benefits for the proposed rule by displaying all possible combinations of PM and ozone-related monetized benefits and costs. Each of the 14 bars in each graph represents a separate point estimate of net benefits under a certain combination of cost and benefit estimation methods. Because it is not a distribution, it is not possible to infer the likelihood of any single net benefit estimate.

Figure 1-1. Estimated Monetized Value of Estimated $PM_{2.5}$- Related Premature Mortalities Avoided According to Epidemiology or Expert-derived Derived PM Mortality Risk Estimate[a]



[A] Column total equals sum of $PM_{2.5}$-related mortality and morbidity benefits.

Figure 1-2. Net Benefits of the Toxics Rule According to $PM_{2.5}$ Epidemiology or Expert-derived Mortality Risk Estimate[a]



[A] Column total equals sum of $PM_{2.5}$-related mortality and morbidity.

1.2 Not All Benefits Quantified

EPA was unable to quantify or monetize all of the health and environmental benefits associated with the proposed Toxics Rule. EPA believes these unquantified benefits are substantial, including the overall value associated with HAP reductions, value of increased agricultural crop and commercial forest yields, visibility improvements, and reductions in nitrogen and acid deposition and the resulting changes in ecosystem functions. Table 1-4 provides a list of these benefits.

Table 1-4. Human Health and Welfare Effects of Pollutants Affected by the Toxics Rule

Pollutant/ Effect	*Quantified and monetized in base estimate*	*Unquantified*
PM: health[a]	Premature mortality based on cohort study estimates[b] and 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, pre-term birth and other reproductive outcomes 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[d] Household soiling 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]
Ozone: health		Premature mortality based on short-term study estimates Hospital admissions: respiratory Emergency room visits for asthma Minor restricted activity days School loss days Chronic respiratory damage Premature aging of the lungs Non-asthma respiratory emergency room visits UVb exposure (+/-)[c]
Ozone: welfare		Decreased outdoor worker productivity Yields for: --Commercial forests --Fruits and vegetables, and --Other commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-)[c] Climate impacts

Pollutant/ Effect	*Quantified and monetized in base estimate*	*Unquantified*
NO_2: health		Respiratory hospital admissions Respiratory emergency department visits Asthma exacerbation Acute respiratory symptoms Premature mortality Pulmonary function
NO_X: welfare		Commercial fishing and forestry from acidic deposition effects Commercial fishing, agriculture and forestry from nutrient deposition effects Recreation in terrestrial and estuarine ecosystems from nutrient deposition effects Other ecosystem services and existence values for currently healthy ecosystems Coastal eutrophication from nitrogen deposition effects
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		Impaired cognitive development Problems with language Abnormal social development Potential for fatal and non-fatal AMI (heart attacks) Association with genetic effects Possible autoimmunity effects in antibodies
Mercury: welfare		Neurological, behavioral, reproductive and survival effects in wildlife (birds, fish, and mammals)

[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.

[D] Visibility-related benefits quantified in air quality modeled scenario, but not the revised scenario. The total benefits reported in Table 1-1 do not reflect visibility benefits.

1.3 Costs, Economic, and Employment Impacts

The projected annual incremental private costs of the proposed Toxics Rule to the electric power industry are $10.9 billion in 2015. These costs represent the total cost to the electricity-generating industry of reducing HAP emissions to meet the emissions limits set out in the rule. Estimates are in 2007 dollars. These costs of the rule are estimated using the Integrated Planning Model (IPM).

There are several national changes in energy prices that result from the proposed Toxics Rule. Retail electricity prices are projected to increase nationally by an average of 3.7% in 2015 with the proposed Toxics Rule. On a weighted average basis, consumer natural gas price impacts are anticipated to range from 0.6% to 1.3% based on consumer class in response to the proposed Toxics Rule between 2015 and 2030.

There are several other types of energy impacts associated with the proposed Toxics Rule. A small amount of coal-fired capacity, about 9.9 GW (3 percent of all coal-fired capacity and 1 percent of all generating capacity in 2015), is projected to be uneconomic to maintain. These units are predominantly smaller and less frequently-used generating units dispersed throughout the area affected by the rule. If current forecasts of either natural gas prices or electricity demand were revised in the future to be higher, that would create a greater incentive to keep these units operational. Coal production for use in the power sector is projected to decrease by less than 2 percent by 2015, and we expect slightly reduced coal demand in Appalachia and the West with the proposed Toxics Rule.

Effective policies to support end-use energy efficiency investments can reduce compliance costs, lessen impacts on electric rates and bills, and reduce the need for new capacity. In 2015 and 2020, annualized private compliance costs to the industry are reduced by $0.3 billion (2007$) and $1.1 billion, respectively, under an energy efficiency scenario. Furthermore, the impacts of the Toxics Rule on retail electricity prices are reduced by 0.04 cents/kWh and 0.38 cents/kWh in 2015 and 2020, respectively, and the need for new capacity is reduced by 0.3 GW and 8.5 GW, respectively, in 2015 and 2020 under an energy efficiency scenario.

In addition to addressing the costs and benefits of the proposed Utility Air Toxics Rule (Toxics Rule), EPA has estimated a portion of the employment impacts of this rulemaking. We have estimated two types of impacts. One provides an estimate of the employment impacts on the regulated industry over time. The second covers the short-term employment impacts associated with the construction of needed pollution control equipment until the compliance date

of the regulation. We expect that the rule's impact on employment will be small, but will (on net) result in an increase in employment.

The approaches to estimate employment impacts use different analytical techniques and are applied to different industries during different time periods, and they use different units of analysis. No overlapping estimates are summed. Estimates from Morgenstern et al. (2002) are used to calculate the ongoing annual employment impacts for the regulated entities (the electric power sector). The short term estimates for employment needed to design, construct, and install the control equipment in the three or four year period before the compliance date are also provided using an approach that estimates employment impacts for the environmental protection sector. Finally some of the other types of employment impacts that will be ongoing are estimated but not summed because they omit some potentially important categories.

In Table 1-5, we show the employment impacts of the Toxics Rule as estimated by the environmental protection sector approach and by the Morgenstern approach.

Table 1-5. Estimated Employment Impact Table

	Annual (reoccurring)	**One time (construction during compliance period)**
Environmental Protection Sector approach*	Not Applicable	30,900
Net Effect on Electric Utility Sector Employment from Morgenstern et al. approach***	9,000** -17, 000 to +35,000****	Not Applicable

*These one-time impacts on employment are estimated in terms of job-years.

**This estimate is not statistically different from zero.

**These annual or reoccurring employment impacts are estimated in terms of production workers as defined by the US Census Bureau's Annual Survey of Manufacturers (ASM).

**** 95% confidence interval

1.4 Small Entity and Unfunded Mandates Impacts

After preparing an analysis of small entity impacts, EPA cannot certify that this proposal will not have a no SISNOSE (significant economic impacts on a substantial number of small entities). Of the 83 small entities affected, 59 are projected to have costs greater than 1 percent of their revenues. EPA's decision to exclude units smaller than 25 Megawatt capacity (MW) as per the requirements of the Clean Air Act has already significantly reduced the burden on small entities, and EPA participated in a Small Business Regulatory Enforcement Fairness Act

(SBREFA) to examine ways to mitigate the impact of the proposed Toxics Rule on affected small entities

EPA examined the potential economic impacts on state and municipality-owned entities associated with this rulemaking based on assumptions of how the affected states will implement control measures to meet their emissions. These impacts have been calculated to provide additional understanding of the nature of potential impacts and additional information.

According to EPA's analysis, of the 96 government entities considered in this, 55 may experience compliance costs in excess of 1 percent of revenues in 2015, based on our assumptions of how the affected states implement control measures to meet their emissions budgets as set forth in this rulemaking.

Government entities projected to experience compliance costs in excess of 1 percent of revenues may have some potential for significant impact resulting from implementation of the Toxics Rule.

1.5 Limitations and Uncertainties

Every analysis examining the potential benefits and costs of a change in environmental protection requirements is limited to some extent by data gaps, limitations in model capabilities (such as geographic coverage), and variability or uncertainties in the underlying scientific and economic studies used to configure the benefit and cost models. Despite the uncertainties, we believe this benefit-cost analysis provides a reasonable indication of the expected economic benefits and costs of the proposed Toxics Rule.

For this analysis, such uncertainties include possible errors in measurement and projection for variables such as population growth and baseline incidence rates; uncertainties associated with estimates of future-year emissions inventories and air quality; variability in the estimated relationships between changes in pollutant concentrations and the resulting changes in health and welfare effects; and uncertainties in exposure estimation.

Below is a summary of the key uncertainties of the analysis:

Costs

- Analysis does not capture employment shifts as workers are retrained at the same company or re-employed elsewhere in the economy.

- We do not include the costs of certain relatively small permitting costs associated with Title V that new program entrants face.

- Technological innovation is not incorporated into these cost estimates. Thus, these cost estimates may be potentially higher than what may occur in the future, all other things being the same.

Benefits

- The mercury concentration estimates for the analysis come from several different sources

- The mercury concentration estimates used in the model were based on simple temporal and spatial averages of reported fish tissue samples. This approach assumes that the mercury samples are representative of "local" conditions (i.e., within the same HUC 12) in similar waterbodies (i.e., rivers or lakes).

- State-level averages for fishing behavior of recreational anglers are applied to each modeled census tract in the state; which does not reflect within-state variation in these factors.

- Application of state-level fertility rates to specific census tracts (and specifically to women in angler households.

- Applying the state-level individual level fishing participation rates to approximate the household fishing rates conditions at a block level.

- Populations are only included in the model if they are within a reasonable distance of a waterbody with fish tissue MeHg samples. This approach undercounts the exposed population (by roughly 40 to 45%) and leads to underestimates of national aggregate baseline exposures and risks and underestimates of the risk reductions and benefits resulting from mercury emission reductions.

- Assumption of 8 g/day fish consumption rate for the general population in freshwater angler households.

- The dose-response model used to estimate neurological effects on children because of maternal mercury body burden has several important uncertainties, including selection of IQ as a primary endpoint when there may be other more sensitive

endpoints, selection of the blood-to-hair ratio for mercury, and the dose-response estimates from the epidemiological literature. Control for confounding from the potentially positive cognitive effects of fish consumption and, more specifically, omega-3 fatty acids.

- Valuation of IQ losses using a lost earning approach has several uncertainties, including (1) there is a linear relationship between IQ changes and net earnings losses, (2) the unit value applies to even very small changes in IQ, and (3) the unit value will remain constant (in real present value terms) for several years into the future. Each unit value for IQ losses has two main sources of uncertainty (1). The statistical error in the average percentage change in earnings as a result of IQ changes and (2) estimates of average lifetime earnings and costs of schooling. Most of the estimated PM-related benefits in this rule accrue to populations exposed to higher levels of $PM_{2.5}$. Of these estimated PM-related mortalities avoided, about 30% occur among populations initially exposed to annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ and about 80% occur among those initially exposed to annual mean $PM_{2.5}$ level of 7.5 $\mu g/m^3$; these are the lowest air quality levels considered in the Laden et al. (2006) and Pope et al. (2002) studies, respectively. This fact is important, because as we estimate PM-related mortality among populations exposed to levels of $PM_{2.5}$ that are successively lower, our confidence in the results diminishes. However, our analysis shows that a substantial portion of the impacts occur at higher exposures.

- There are uncertainties related to the health impact functions used in the analysis. These include: within study variability; across study variation; the application of concentration-response (C-R) functions nationwide; extrapolation of impact functions across population; and various uncertainties in the C-R function, including causality and thresholds. Therefore, benefits may be under- or over-estimates.

- Analysis is for 2016, and projecting key variables introduces uncertainty. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions and source level emissions, as well as population, health baselines, incomes, technology, and other factors.

- This analysis omits certain unquantified effects due to lack of data, time and resources. These unquantified endpoints include other health and ecosystem effects. EPA will continue to evaluate new methods and models and select those most appropriate for estimating the benefits of reductions in air pollution. Enhanced

collaboration between air quality modelers, epidemiologists, toxicologists, ecologists, and economists should result in a more tightly integrated analytical framework for measuring benefits of air pollution policies.

- $PM_{2.5}$ mortality benefits represent a substantial proportion of total monetized benefits (over 90%), and these estimates have following key assumptions and uncertainties.

1. The $PM_{2.5}$-related benefits of the alternative scenarios were derived through a benefit per-ton approach, which does not fully reflect local variability in population density, meteorology, exposure, baseline health incidence rates, or other local factors that might lead to an over-estimate or under-estimate of the actual benefits of controlling SO_2.
2. 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.
3. We assume that the health impact function for fine particles is linear within the range of ambient concentrations under consideration. 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.
4. To characterize the uncertainty in the relationship between $PM_{2.5}$ and premature mortality, 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.

1.6 References

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Chapter 2
INTRODUCTION AND BACKGROUND

2.1 Introduction

2.1.1 Background for Proposed Toxics Rule

2.1.2 NESHAP

This action proposes NESHAP for new and existing coal- and oil-fired electric utility steam generating units (EGUs) meeting the definition found in CAA section 112(a)(8). EPA is proposing these standards to meet its statutory obligation to address HAP emissions from these sources under CAA section 112(d). The proposed NESHAP for new and existing coal- and oil-fired EGUs will be proposed under 40 CFR part 63, subpart UUUUU.

On December 20, 2000 (65 FR 79825), EPA determined that regulation of coal- and oil-fired EGUs under CAA section 112 was appropriate and necessary, in accordance with CAA section 112(n)(1)(A). EPA at the same time added coal- and oil-fired EGUs to the list of industries requiring regulation under CAA section 112(d). The December 2000 listing triggered the deadline established by Congress in CAA section 112(c)(5) under which EPA has two years from the date of listing in which to promulgate "emissions standards under section (d) of this section."

In 2002, EPA initiated a CAA section 112(d) standard setting process for coal- and oil-fired EGUs, and on January 30, 2004, proposed CAA section 112(d) standards for mercury (Hg) emissions from coal-fired EGUs and nickel (Ni) emissions from oil-fired EGUs, and, in the alternative, proposed to remove EGUs from the CAA section 112(c) list based on a finding that it was neither appropriate nor necessary to regulate EGUs pursuant to CAA section 112. EPA never finalized the proposed CAA section 112(d) standard. The removal of EGUs from the CAA section 112 list was challenged in the United States (U.S.) Court of Appeals for the District of Columbia Circuit (D.C. Circuit Court). The Agency finalized the CAA section 111 alternative, after taking and responding to extensive public comments on both sets of regulatory options, by issuing a de-listing rule (Section 112(n) Revision Rule; 70 FR 15994; March 29, 2005) and a final rule (Clean Air Mercury Rule, CAMR) establishing Hg emissions standards for coal-fired EGUs under CAA section 111 on May 18, 2005 (70 FR 28606).

Petitions for reconsideration were filed by a number of parties in summer 2005. EPA responded to the petitions with a final notice of reconsideration on June 9, 2006 (71 FR 33388). Petitions for judicial review were filed on November 29, 2006, by Environmental Petitioners; the National Congress of American Indians and Treaty Tribes; ARIPPA; American Coal for Balanced Mercury Regulations, et al.; United Mine Workers of America; Alaska Industrial Development and Export Authority; the States of New Jersey, California, Connecticut, Delaware, Illinois, Maine, Maryland, Massachusetts, Michigan, Minnesota, New Hampshire, New Mexico, New York, Pennsylvania, Rhode Island, Vermont, and Wisconsin; and the City of Baltimore, MD (*State of New Jersey, et al., v. EPA, 517 F.3d 574)*.

On February 8, 2008, the D.C. Circuit Court vacated the Section 112(n) Revision Rule (*State of New Jersey, et al., v. EPA, 517 F.3d 574)*, and subsequently denied rehearing and rehearing *en banc* of that decision. As a part of the decision, the D.C. Circuit Court also vacated CAMR, reverting to the December 2000 regulatory determination and requiring the development of emission standards under CAA section 112(d) (MACT standards) for coal- and oil-fired EGUs. The litigation process continued until, on January 29, 2009, EPA requested of the Department of Justice (DOJ) that the Government's appeals be withdrawn.

On December 18, 2008, several environmental and public health organizations ("Plaintiffs")[1] filed a complaint in the D.C. District Court (Civ. No. 1:08-cv-02198 (RMC)) alleging that the Agency had failed to perform a nondiscretionary duty under CAA section 304(a)(2), by failing to promulgate final section 112(d) standards for HAP from coal- and oil-fired EGUs by the statutorily-mandated deadline, December 20, 2002, 2 years after such sources were listed under section 112(c). EPA settled that litigation. A Consent Decree was issued on April 15, 2010, that calls for EPA to, no later than March 16, 2011, sign for publication in the Federal Register a notice of proposed rulemaking setting forth EPA's proposed emission standards for coal- and oil-fired EGUs and, no later than November 16, 2011, sign for publication in the Federal Register a notice of final rulemaking.

In response to the D.C. Circuit Court's vacatur, we are proposing CAA section 112(d) NESHAP for all coal- and oil-fired EGUs that reflect the application of the maximum achievable control technology (MACT) consistent with the requirements of CAA sections 112(d)(2) and (3).

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[1] American Nurses Association, Chesapeake Bay Foundation, Inc., Conservation Law Foundation, Environment America, Environmental Defense Fund, Izaak Walton League of America, Natural Resources Council of Maine, Natural Resources Defense Council, Physicians for Social Responsibility, Sierra Club, The Ohio Environmental Council, and Waterkeeper Alliance, Inc.

This proposed rule would protect air quality and promote public health by reducing emissions of the hazardous air pollutants (HAP) listed in CAA section 112(b).

2.1.3 NSPS

Section 111(b)(1)(b) of the CAA requires EPA to periodically review and revise the new source performance standards (NSPS) as necessary to reflect improvements in methods for the reducing emissions. The NSPS for electric utility steam generating units (40 CFR part 60, subpart Da) were originally promulgated on June 11, 1979 (44 FR 33580). On February 27, 2006, EPA promulgated amendments to the NSPS for particulate matter (PM), sulfur dioxide (SO_2), and nitrogen oxides (NO_X) contained in the standards of performance for electric utility steam generating units (71 FR 9866). EPA was subsequently sued by the offices of multiple states attorneys general and environmental organizations on the amendments. The Petitioners alleged that EPA failed to correctly identify the best system of emission reductions for the amended SO_2 and NO_X standards. The Petitioners also claimed that it is appropriate to establish emission limits for fine particulate matter and condensable particulate matter. Based upon further examination of the record, EPA has determined that certain issues in the rule warrant further consideration. On September 4, 2009, EPA was granted a voluntary remand without vacatur of the 2006 amendments. EPA considers it appropriate to respond to the NSPS voluntary remand in conjunction with the EGU NESHAP since it allows EPA to present a more comprehensive affect on the utility sector. Therefore, even though we are not under any judicial timetable to complete the NSPS remand, we are proposing it in conjunction with the NESHAP. We also are proposing several minor amendments, technical clarifications, and corrections to existing provisions of the fossil fuel-fired EGU and large and small industrial-commercial-institutional steam generating units NSPS, 40 CFR part 60, subparts D, Db, and Dc.

The term "toxics rule" for the remainder of this RIA refers to the combination of the EGU NESHAP and NSPS proposals.

2.2 Appropriate & Necessary Analyses

In 2000, EPA issued a finding that it was both appropriate and necessary to regulate HAP emissions from utilities, in part because Hg, a listed HAP, is both a public health concern and a concern in the environment. This finding was based on the results of the study documented in the Utility Study, as well as subsequent analyses and other available information at the time of the decision. The finding that it is appropriate to regulate HAP emissions from coal- and oil-fired EGUs under CAA section 112 was based on three main points: 1) EGUs are the largest domestic source of Hg emissions, 2) Hg in the environment presents significant hazards to public

health and the environment, and 3) EPA had identified a number of control options which were anticipated to effectively reduce HAP emissions from such units. The finding also noted that remaining uncertainties regarding the extent of the public health impact from HAP emissions from oil-fired EGUs argued for regulation. The finding that it is necessary to regulate HAP emissions from coal- and oil-fired EGUs under CAA section 112 was based on the assessment that implementation of other requirements under the CAA would not adequately address the serious public health and environmental hazards arising from utility HAP emissions which CAA section 112 is intended to address.

Based on the quantitative and qualitative analyses of public health and environmental hazards described above, as well as the analyses of emissions and availability of HAP emission controls, we find that regulation of HAP emissions from coal- and oil-fired EGUs under CAA section 112 is appropriate and necessary. The finding that it is appropriate to regulate emissions from coal- and oil-fired EGUs under CAA section 112 is confirmed because: 1) Hg continues to pose a hazard to public health, 2) U.S. EGU emissions are still the largest domestic source of U.S. Hg emissions (by 2016, EPA projects that U.S. EGU Hg emissions are over 6 times larger than next largest source, which is iron and steel manufacturing), and 3) effective controls for Hg and non-Hg HAP are available for U.S. EGU sources. In addition, new analyses by EPA show that U.S. EGU emissions of non-Hg HAP cause a non-negligible health hazard due to increased cancer risk. The finding that it is necessary to regulate emissions from coal- and oil-fired EGUs under CAA section 112 is confirmed because emissions of Hg and non-Hg HAP causing hazards to public health and the environment will not be explicitly addressed by existing or anticipated requirements under the CAA. For more information on these findings and the analyses to support them, please refer to the preamble or the TSD for the appropriate & necessary analyses.

2.3 Provisions of the Proposed Toxics Rule

2.3.1 What Is the Source Category Regulated by the Proposed Toxics Rule?

The proposed Toxics rule addresses emissions from new and existing coal- and oil-fired EGUs. A major source of HAP emissions is generally a stationary source that emits or has the potential to emit 10 tons per year or more of any single HAP or 25 tons per year or more of any combination of HAP. An area source of HAP emissions is a stationary source that is not a major source. CAA section 112(n)(1)(A) makes no distinction between major and area sources of coal- and oil-fired electric utility steam generating units.

CAA section 112(a)(8) defines an EGU as:

a fossil fuel-fired combustion unit of more than 25 megawatts electric (MWe) that serves a generator that produces electricity for sale. A unit that cogenerates steam and electricity and supplies more than one-third of its potential electric output capacity and more than 25 MWe output to any utility power distribution system for sale is also an electric utility steam generating unit.

This action established 40 CFR part 63, subpart UUUUU, to address HAP emissions from new and existing coal- and oil-fired EGUs. EPA must determine what is the appropriate maximum achievable control technology (MACT) for those units under sections 112(d)(2) and (d)(3) of the CAA.

EPA has divided coal- and oil-fired EGUs into the following subcategories:

- coal-fired units designed for coal > 8,300 Btu/lb;
- coal-fired units designed for coal < 8,300 Btu/lb;
- IGCC units;
- Liquid oil-fired units; and
- Solid oil-derived fuel-fired units.
- The EGU NSPS applies to owners/operators of facilities capable of firing more than 73 megawatts (MW) (250 million Btu per hour(MMBtu/hr)) heat input of fossil fuel and that sells more than 25 MW of electric power to a utility power distribution system. The NSPS also apply to industrial-commercial-institutional cogeneration units over 250 MMBtu/hr that sell more than 25 MW and more than one-third of their potential output capacity to any utility power distribution system.

2.3.2 What Are the Pollutants Regulated by the Rule?

The proposed NESHAP regulates emissions of HAP. Available emissions data show that several HAP, which are formed during the combustion process or which are contained within the fuel burned, are emitted from coal- and oil-fired electric utility steam generating units. The individual HAPs include mercury, arsenic, cadmium, lead, and nickel, among others. EPA described the health effects of these HAP and other HAP emitted from the operation of coal- and oil-fired electric utility steam generating units in the preamble to the proposed rule. These HAP

emissions are known to cause, or contribute significantly to air pollution, which may reasonably be anticipated to endanger public health or welfare.

In addition to reducing HAP, the emission control technologies that will be installed on coal- and oil-fired electric utility steam generating units to reduce HAP will also reduce sulfur dioxide (SO_2) and particulate matter (PM).

The proposed NSPS amendments would revise the PM, SO_2, and NO_X standards. A wide range of human health and welfare effects are linked to the emissions of PM, SO_2, and NO_X. These human health and welfare effects are discussed extensively in Chapter 6 of this RIA.

2.3.3 What Are the Proposed Requirements?

The numerical emission standards that are being proposed for existing coal- and oil-fired electric utility steam generating units are shown in Tables 2-1 and 2-2.

Table 2-1. Emission Limitations for Coal-Fired and Solid Oil-Derived Fuel-Fired EGUs

Subcategory	Total particulate matter	Hydrogen chloride	Mercury
Existing coal-fired unit designed for coal ≥ 8,300 Btu/lb	0.03 lb/MMBtu (0.2 lb/MWh)	0.002 lb/MMBtu (0.02 lb/MWh)	1 lb/TBtu (0.02 lb/GWh)
Existing coal-fired unit designed for coal < 8,300 Btu/lb	0.03 lb/MMBtu (0.2 lb/MWh)	0.002 lb/MMBtu (0.02 lb/MWh)	11 lb/TBtu (0.2 lb/GWh) 4 lb/TBtu* (0.04 lb/GWh*)
Existing - IGCC	0.05 lb/MMBtu (0.3 lb/MWh)	0.0005 lb/MMBtu (0.003 lb/MWh)	3 lb/TBtu (0.02 lb/GWh)
Existing – Solid oil-derived	0.2 lb/MMBtu (2 lb/MWh)	0.005 lb/MMBtu (0.05 lb/MWh)	0.2 lb/TBtu (0.002 lb/GWh)
New coal-fired unit designed for coal ≥ 8,300 Btu/lb	0.05 lb/MWh	0.3 lb/GWh	0.00001 lb/GWh
New coal-fired unit designed for coal < 8,300 Btu/lb	0.05 lb/MWh	0.3 lb/GWh	0.04 lb/GWh

Note: lb/MMBtu = pounds pollutant per million British thermal units fuel input

lb/TBtu = pounds pollutant per trillion British thermal units fuel input

lb/MWh = pounds pollutant per megawatt-electric output

lb/GWh = pounds pollutant per gigawatt-electric output

* Beyond-the-floor limit.

Table 2-2. Emission Limitations for Liquid Oil-Fired EGUs

Subcategory	Total HAP metals	Hydrogen chloride	Hydrogen fluoride	Mercury
Existing – Liquid oil	0.00003 lb/MMBtu (0.0003 lb/MWh)	0.0003 lb/MMBtu (0.003 lb/MWh)	0.0002 lb/MMBtu (0.002 lb/MWh)	0.05 lb/TBtu (0.0006 lb/GWh)
New – Liquid oil	0.0004 lb/MWh	0.0005 lb/MWh	0.0005 lb/MWh	0.0001 lb/GWh

We are also proposing alternate equivalent emission standards (for certain subcategories) to the proposed surrogates in three areas: SO_2 (in addition to HCl), individual non-Hg metals (for PM), and total non-Hg metals (for PM). The proposed emission limitations are provided in Tables 2-3 and 2-4.

Table 2-3. Alternate Emission Limitations for Existing Coal- and Oil-Fired EGUs

Subcategory	Total HAP metals	Hydrogen chloride	Hydrogen fluoride	Mercury
Existing – Liquid oil	0.00003 lb/MMBtu (0.0003 lb/MWh)	0.0003 lb/MMBtu (0.003 lb/MWh)	0.0002 lb/MMBtu (0.002 lb/MWh)	0.05 lb/TBtu (0.0006 lb/GWh)
New – Liquid oil	0.0004 lb/MWh	0.0005 lb/MWh	0.0005 lb/MWh	0.0001 lb/GWh

NA = Not applicable

Table 2-4. Alternate Emission Limitations for New Coal- and Oil-Fired EGUs

Subcategory	Coal-fired unit designed for coal ≥ 8,300 Btu/lb, lb/MWh	Coal-fired unit designed for coal < 8,300 Btu/lb, lb/MWh	Liquid oil, lb/MWh	Solid oil-derived, lb/MWh	IGCC, lb/MWh
SO_2	0.23	0.23	NA	0.71	NA
Total metals	0.000022	0.000022	NA	0.00016	0.00038
Antimony, Sb	1.3×10^{-7}	1.3×10^{-7}	1.1×10^{-6}	7.4×10^{-7}	1.8×10^{-5}
Arsenic, As	5.6×10^{-7}	5.6×10^{-7}	1.6×10^{-6}	1.1×10^{-6}	1.4×10^{-5}
Beryllium, Be	6.1×10^{-8}	6.1×10^{-8}	6.0×10^{-7}	6.1×10^{-8}	1.6×10^{-7}
Cadmium, Cd	3.4×10^{-7}	3.4×10^{-7}	3.9×10^{-7}	5.4×10^{-7}	1.7×10^{-6}
Chromium, Cr	7.1×10^{-6}	7.1×10^{-6}	1.2×10^{-5}	6.1×10^{-6}	2.8×10^{-5}
Cobalt, Co					
Lead, Pb	1.1×10^{-6}	1.1×10^{-6}	5.3×10^{-6}	1.2×10^{-5}	9.2×10^{-6}
Manganese, Mn	1.1×10^{-6}	1.1×10^{-6}	2.4×10^{-5}	6.4×10^{-6}	1.6×10^{-5}
Nickel, Ni	2.9×10^{-6}	2.9×10^{-6}	3.8×10^{-5}	6.5×10^{-6}	2.9×10^{-5}
Selenium, Se	6.8×10^{-7}	6.8×10^{-7}	4.9×10^{-6}	8.4×10^{-7}	2.6×10^{-4}

NA = Not applicable

We analyzed a beyond-the-floor standard for Hg of only 4 lbs/trillion BTUs for all existing and new "coal-fired units designed for coal < 8,300 Btu/lb" based on the availability of activated carbon injection (ACI) for cost-effective Hg control. Most of these units burn lignite coal. We are proposing a beyond-the-floor standard for these units because the Agency considers the cost of incremental reductions beyond the MACT floor standard of 11 lbs/trillion BTUs to be reasonable. While the primary IPM analysis discussed in Chapter 8 included a beyond-the-floor limit, EPA performed a supplemental analysis that estimates the difference in impacts between regulating coal-fired units designed for coal <8,300 Btu/lb at the floor limit and at the beyond-the-floor limit modeled. This analysis (the IPM Beyond the Floor Cost TSD) shows that if the units were only required to meet a standard of 11 lbs/trillion BTUs, the units

would emit approximately an additional 3,854 lbs at a reduced annualized cost of $86.7 million. EPA also performed an alternative analysis which can be found in the Beyond the MACT Floor Analysis TSD.

The proposed NSPS standards are shown in Table 2-5.

Table 2-5. NSPS Emission Standards

Pollutant	Existing Standard	Proposed Standard
PM	0.015 lb/MMBtu (filterable PM)	0.026 lb/MMBtu (total PM)
SO2	1.4 lb/MWh or 95% reduction	1.0 lb/MWh or 97% Reduction
NOX	1.0 lb/MWh	0.70 lb/MWh (option 1) Combined NOX + CO Standard (option 2)

The EGU NESHAP PM and SO_2 standards for new facilities are as stringent or more stringent than the proposed NSPS amendments so we have concluded that there are no costs or benefits associated with these amendments. Thus, the only impacts associated with these amendments are those for the NOx emissions limits for new facilities.

2.3.4 What Are the Operating Limitations?

Instead of emission limitations for the organic HAP, we are proposing that owners or operators of EGUs submit to the delegated authority or EPA, as appropriate, if requested, documentation that an annual performance test meeting the requirements of the proposed rule was conducted. We are proposing that, to comply with the work practice standard, an annual performance test procedure include the following:

(1) Inspect the burner, and clean or replace any components of the burner as necessary,

(2) Inspect the flame pattern and make any adjustments to the burner necessary to optimize the flame pattern consistent with the manufacturer's specifications,

(3) Inspect the system controlling the air-to-fuel ratio, and ensure that it is correctly calibrated and functioning properly,

(4) Minimize total emissions of carbon monoxide (CO) consistent with the manufacturer's specifications,

(5) Measure the concentration in the effluent stream of CO in parts per million, dry volume basis (ppmvd), before and after the adjustments are made,

(6) Submit an annual report containing the concentrations of CO in the effluent stream in ppmvd, and oxygen in percent dry basis, measured before and after the adjustments of the EGU, a description of any corrective actions taken as a part of the combustion adjustment, and the type and amount of fuel used over the 12 months prior to the annual adjustment.

2.4 Startup, Shutdown, and Malfunction

In proposing the standards in this NESHAP, EPA has taken into account startup and shutdown periods and, for the reasons explained below, has not proposed different standards for those periods. The standards that we are proposing are daily or monthly averages. Continuous emission monitoring data obtained from best performing units, and used in establishing the standards, include periods of startup and shutdown. EGUs, especially solid fuel-fired EGUs, do not normally startup and shutdown more than once per day. Thus, we are not establishing a separate emission standard for these periods because startup and shutdown are part of their routine operations and, therefore, are already addressed by the standards. Periods of startup, normal operations, and shutdown are all predictable and routine aspects of a source's operation. We have evaluated whether it is appropriate to have the same standards apply during startup and shutdown as applied to normal operations.

Periods of startup, normal operations, and shutdown are all predictable and routine aspects of a source's operations. However, by contrast, malfunction is defined as a "sudden, infrequent, and not reasonably preventable failure of air pollution control and monitoring equipment, process equipment or a process to operate in a normal or usual manner..." (40 CFR 63.2) EPA has determined that malfunctions should not be viewed as a distinct operating mode and, therefore, any emissions that occur at such times do not need to be factored into development of CAA section 112(d) standards, which, once promulgated, apply at all times.

The existing PM, SO_2, and NO_X NSPS exclude periods of startup and shutdown. The proposed PM, SO_2, and NO_X standards would include periods of startup and shutdown. Periods of malfunction for the PM and NO_X standards and periods of emergency condition for the SO_2 standard are presently excluded from the emissions standards and would continue to be excluded.

2.5 Baseline and Years of Analysis

The Agency considered all promulgated CAA requirements, known state actions, and NSR/PSD enforcement actions in the baseline used to develop the estimates of benefits and costs for the proposed Toxics rule. EPA did not consider actions states may take in the future to

implement the existing ozone and $PM_{2.5}$ NAAQS standards in the baseline for this analysis. The year 2016 is the compliance year for the proposed Toxics rule, though as we explain in Chapters 5,6,8 and 9 we use 2015 as a proxy for compliance in 2016 for our benefits and economic impact analysis due to availability of modeling impacts in that year. All estimates presented in this report represent annualized estimates of the benefits and costs of the proposed Toxics Rule in 2016 rather than the net present value of a stream of benefits and costs in these particular years of analysis.

2.6 Benefits of Emission Controls

The benefits of the proposed Toxics Rule are discussed in Chapter 5 of this report. Annual monetized benefits of $58 to 140 billion (3 percent discount rate) or $52 to 130 billion (7 percent discount rate) are expected for the proposed Toxics rule in 2016.

2.7 Cost of Emission Controls

EPA analyzed the costs of the proposed Toxics Rule using the Integrated Planning Model (IPM). EPA has used this model in the past to analyze the impacts of regulations on the power sector and used an earlier version of this model to analyze the impacts of the CAIR rule and proposed Transport Rule. EPA estimates the private industry annual compliance costs of the rule to the power sector to be $10.9 billion in 2015 (2007 dollars). In estimating the net benefits (benefits – costs) of the rule, EPA uses social costs of the rule that represent the costs to society of this rule. The social costs of the rule are estimated to be $ 10.9 billion (2007 dollars) in 2015. A description of the methodology used to model the costs and economic impacts to the power sector is discussed in Chapter 8 of this report. A description of how the social costs and employment impacts associated with this proposed rule are estimated is provided in Chapter 9 of this report.

2.8 Organization of the Regulatory Impact Analysis

This report presents EPA's analysis of the benefits, costs, and other economic effects of the proposed Toxics Rule to fulfill the requirements of a Regulatory Impact Analysis (RIA). This RIA includes the following chapters:

- Chapter 3, Emissions Impacts, describes the emission inventories and modeling that are essential inputs into the cost and benefit assessments.
- Chapter 4, Air Quality Impacts, describes the air quality data and modeling that are important for assessing the effect on contributions to air quality from the remedy options applied in this proposed rule, and as inputs to the benefits assessment.

- Chapter 5, Mercury and Other HAP Benefits Analysis, describes the methodology and results of the benefits analysis for mercury and other HAP.
- Chapter 6, Co-Benefits Analysis, describes the methodology and results of the benefits analysis for $PM_{2.5}$, Ozone, and other benefit categories.
- Chapter 7, Electric Power Sector Profile, describes the industry affected by the rule.
- Chapter 8, Cost, Economic, and Energy Impacts, describes the modeling conducted to estimate the cost, economic, and energy impacts to the power sector.
- Chapter 9, Economic and Employment Impacts, describes the analysis to estimate the impacts on employment associated with the proposed rule.
- Chapter 10, Statutory and Executive Order Impact Analyses, describes the small business, unfunded mandates, paperwork reduction act, environmental justice, and other analyses conducted for the rule to meet statutory and Executive Order requirements.
- Chapter 11, Comparison of Benefits and Costs, shows a comparison of the social benefits to social costs of the rule.
- Appendix A, Mercury Speciation Fractions Used to Speciate the Mercury Emissions
- Appendix B, Analysis of Trip Travel Distance For Recreational Freshwater Anglers
- Appendix C, Co-Benefit Analysis
- Appendix D, Illustrative End-Use Energy Efficiency Policy Sensitivity
- Appendix E, OAQPS Multimarket Model to Assess the Economic Impact of Environmental Regulation

Chapter 3
EMISSIONS IMPACTS

This chapter summarizes the emissions inventories that are used to create emissions inputs to the air quality modeling that is described in Chapter 4. This chapter provides a summary of the baseline emissions inventories and the emissions reductions that were modeled for this rule. The emissions inventories are processed into a form that is required by the Community Multi-scale Air Quality (CMAQ) model, which simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone, particulate matter (PM) and air toxics. As part of the analysis for this rulemaking, the CMAQ was used to calculate daily and annual particulate matter less than 2.5 microns in diameter ($PM_{2.5}$) concentrations, 8-hr maximum ozone, annual total mercury (Hg) deposition levels and visibility impairment. In the remainder of this Chapter we provide an overview of (1) the emissions components of the modeling platform, (2) the development of the 2005 base-year emissions, (3) the development of the 2016 future-year base case emissions, and (4) the development of the 2016 future year-control case (policy case) emissions. It should be noted that the projected future year inventory used for this analysis is generally representative of several years around 2016 such as 2015.

3.1 Overview of Modeling Platform and Emissions Processing Performed

The inputs to the air quality model; including emissions, meteorology, initial conditions, boundary conditions; along with the methods used to produce the inputs and the configuration of the air quality model are collectively known as a 'modeling platform'. The 2005-based air quality modeling platform used for the proposed Toxics Rule includes 2005 base-year emissions and 2005 meteorology for modeling ozone, $PM_{2.5}$ and mercury (Hg) with CMAQ. Version 4.1 of the 2005-based platform (2005 v4.1 platform) was used for the proposed Toxics Rule, and it is described in the 2005-based, v4.1 platform document: "Technical Support Document: Preparation of Emissions Inventories for the Version 4.1, 2005-based Platform", posted at http://www.epa.gov/ttn/chief/emch/. The Emission Inventories Technical Support Document for the Proposed Toxics Rule entitled "Technical Support Document (TSD) For the Proposed Toxics Rule, Docket No. EPA-HQ-OAR-2009-0234", posted at the same site, describes the development of the future year inventories. It provides more detail on (1) the development of the 2016 base-case emissions inventories for all sectors and (2) the procedures followed to create emissions inputs to CMAQ. Details on the non-emissions portion of the modeling platform used for the RIA are provided in Chapter 4.

Emissions estimates were made for a 2005 base year and for the 2016 future-year scenarios. All inventories include emissions from EGUs, non-EGU point sources, stationary nonpoint sources (previously referred to as stationary area sources), onroad mobile sources, nonroad mobile sources and natural, biogenic emissions. Mercury emissions from volcanic sources, and land and ocean direct and recycled emissions are also included. For each of the modeling scenarios conducted: 2005 base year, 2016 base case, and 2016 control case, the emissions inventory files were processed using the Sparse Matrix Operator Kernel Emissions (SMOKE) Modeling System version 2.6 to produce the gridded model-ready emissions for input to CMAQ. SMOKE was used to create the hourly, gridded emissions data for the species required by CMAQ species to perform air quality modeling for all sectors, including biogenic emissions.

In support of this proposal, EPA processed the emissions in support of air quality modeling for two domains, covering the East and the West (2 separate model runs) of the U.S. and parts of Canada and Mexico using a horizontal grid resolution of 12 x 12 kilometers (km). These 12 km modeling domains were "nested" within a modeling domain covering the lower 48 states using a grid resolution of 36 x 36 km.[1]

3.2 Development of 2005 Base Year Emissions

Emissions inventory inputs representing the year 2005 were developed to provide a base year for forecasting future air quality. These inventories include criteria air pollutants, hydrogen chloride (HCl), chlorine (CL_2) and mercury.[2] Additionally, for some sectors, benzene, formaldehyde, acetaldehyde and methanol are used from the inventory for chemical speciation of volatile organic compounds (VOC). The emission source sectors and the basis for current and future-year inventories are listed and defined in Table 3-1. These are the same sectors as were used in the 2005-based version 4 (v4) platform (www.epa.gov/ttn/chief/emch/index.html#2005), which was the starting point for the v4.1 platform. A comparison of these two platforms is provided in the 2005-based, v4.1 platform document described earlier. The starting point for both platforms was the 2005 National Emission Inventory (NEI), version 2 (v2) from October 6, 2008 (http://www.epa.gov/ttn/chief/net/2005inventory.html). The v4.1 platform utilizes the same 2006 Canadian inventory and a 1999 Mexican inventory as were used in the v4 platform; these

[1] The air quality predictions from the 36 km Continental US (CONUS) domain were used to provide incoming "boundary" concentrations for the 12 km domains.

[2] The mercury emissions used in the version 4.1 platform include changes to the version 4 platform 2005 Hg emissions. These changes were made in support of the analyses for this rule and for the NESHAP for Industrial, Commercial, and Institutional Boilers and Process Heaters (Boiler MACT). These changes are provided in more detail in this section.

were the latest available data from these countries and were used for the portions of Canada and Mexico within the modeling domains.

Table 3-1. Emissions Source Sectors for Current and Future-Year Inventories, 2005-based Platform, Version 4.1

Platform Sector, modeling abbrev. and corresponding 2005 NEI sector	Description and resolution of the data input to SMOKE, 2005 v4.1 platform
EGU sector: ***ptipm*** **NEI Sector: Point**	For all pollutants other than mercury (Hg): 2005 NEI v2 point source EGUs mapped to the Integrated Planning Model (IPM) model using the National Electric Energy Database System (NEEDS) 2006 version 4.10 database. For Hg: 6/18/2010 version of the inventory used for the 2005 National Air Toxics Assessment (NATA) mapped to IPM using NEEDS 2006 version 4.10. The NATA inventory is an update to the 2005 NEI v2 and was divided into EGU and non-EGU sectors consistent with the other pollutants. We additionally removed Hg from sources from the National Emission Standards for Hazardous Air Pollutants for Industrial, Commercial, and Institutional Boilers and Process Heaters (aka "Boiler MACT") Information Collection Request (ICR) database because we included these emissions in the non-EGU sector. For both: Daily emissions input into SMOKE. Annual emissions allocated to months using 3 years of continuous emissions monitor (CEM) data, and allocated to days using month-to day allocations from the 2005 CEM data.
Non-EGU sector: ***ptnonipm*** **NEI Sector: Point**	For all pollutants other than Hg: All 2005 NEI v2 point source records not matched to the ptipm sector, annual resolution. Includes all aircraft emissions. Additionally updated inventory to remove duplicates, improve estimates from ethanol plants, and reflect new information collected from industry from the ICR for the Boiler MACT. Includes point source fugitive dust emissions for which county-specific PM transportable fractions were applied. For Hg: The 6/18/2010 version of NATA inventory was used except for replacement of boiler Hg emissions with the Hg emissions developed for the Boiler MACT. In addition, modified gold mine emissions, and removed Hg from facilities that closed prior to 2005. For both: Annual resolution.
Average-fire sector: ***avefire***	Average-year wildfire and prescribed fire emissions, unchanged from the 2005v4 platform; county and annual resolution.
Agricultural sector: ***ag*** **NEI Sector: Nonpoint**	NH_3 emissions from 2002 NEI nonpoint livestock and fertilizer application, county and annual resolution. Unchanged from the 2005v4 platform.
Area fugitive dust sector: ***afdust*** **NEI Sector: Nonpoint**	PM_{10} and $PM_{2.5}$ from fugitive dust sources (e.g., building construction, road construction, paved roads, unpaved roads, agricultural dust) from the NEI nonpoint inventory (which used 2002 emissions for this sector) after application of county-specific PM transportable fractions. Includes county and annual resolution.
Remaining nonpoint sector: ***nonpt*** **NEI Sector: Nonpoint**	Primarily 2002 NEI nonpoint sources not otherwise included in other SMOKE sectors, county and annual resolution. Also includes updated Residential Wood Combustion emissions, year 2005 non-California WRAP oil and gas Phase II inventory and year 2005 Texas and Oklahoma oil and gas emissions. Removed Hg

Platform Sector, modeling abbrev. and corresponding 2005 NEI sector	Description and resolution of the data input to SMOKE, 2005 v4.1 platform
	emissions from boilers to avoid double counting with Hg emissions added to the non-EGU sector from the Boiler MACT ICR.
Nonroad sector: ***nonroad*** **NEI Sector: Nonroad**	Monthly nonroad emissions from the National Mobile Inventory Model (NMIM) using NONROAD2005 version nr05c-BondBase, which is equivalent to NONROAD2008a, since it incorporated Bond rule revisions to some of the base case inputs and the Bond rule controls did not take effect until later. NMIM was used for all states except California. Monthly emissions for California created from annual emissions submitted by the California Air Resources Board (CARB) for the 2005v2 NEI.
Locomotive, and non-C3 commercial marine vessel (CMV): ***alm_no_c3*** **NEI Sector: Nonroad**	2002 NEI non-rail maintenance locomotives, and category 1 and category 2 commercial marine vessel (CMV) emissions sources, county and annual resolution. Aircraft emissions are included in the Non-EGU sector (as point sources) and category 3 CMV emissions are contained in the seca_c3 sector.
C3 commercial marine: ***seca_c3*** **NEI Sector: Nonroad**	Annual point source-formatted, year 2005 category 3 (C3) CMV emissions, developed for the rule called "Control of Emissions from New Marine Compression-Ignition Engines at or Above 30 Liters per Cylinder", usually described as the Emissions Control Area (ECA) study (http://www.epa.gov/otaq/oceanvessels.htm). Utilized final projections from 2002, developed for the C3 ECA proposal to the International Maritime Organization (EPA-420-F-10-041, August 2010).
Onroad California, NMIM-based, and Motor Vehicle Emissions Simulator (MOVES) sources not subject to temperature adjustments: ***on_noadj*** **NEI Sector: Onroad**	Three, monthly, county-level components: 1) California onroad, created using annual emissions submitted by CARB for the 2005 NEI version 2. NH_3 (not submitted by CARB) from MOVES2010. 2) Onroad gasoline and diesel vehicle emissions from MOVES2010 not subject to temperature adjustments: exhaust carbon monoxide (CO), nitrogen oxides (NO_X), sulfur dioxide (SO_2), VOC, ammonia (NH_3), benzene, formaldehyde, acetaldehyde, 1,3-butadiene, acrolein, naphthalene, brake and tirewear PM, and evaporative VOC, benzene, and naphthalene. 3) Onroad emissions for Hg from NMIM using MOBILE6.2, other than for California.
Onroad cold-start gasoline exhaust mode vehicle from MOVES subject to temperature adjustments: ***on_moves_startpm*** **NEI Sector: Onroad**	2005 monthly, county-level MOVES2010 onroad gasoline vehicle emissions subject to temperature adjustments. Pollutants that are included are limited to PM species and Naphthalene for exhaust mode only. California emissions not included (covered by on_noadj). This sector is limited to cold start mode emissions that contain different temperature adjustment curves from running exhaust (see on_moves_runpm sector).

Platform Sector, modeling abbrev. and corresponding 2005 NEI sector	Description and resolution of the data input to SMOKE, 2005 v4.1 platform
Onroad running gasoline exhaust mode vehicle from MOVES subject to temperature adjustments: ***on_moves_runpm*** **NEI Sector: Onroad**	2005 monthly, county-level MOVES2010 onroad gasoline vehicle emissions subject to temperature adjustments. Pollutants that are included are limited to PM species and Naphthalene for exhaust mode only. California emissions not included. This sector is limited to running mode emissions that contain different temperature adjustment curves from cold start exhaust (see on_moves_startpm sector).
Biogenic: ***biog***	Hour-specific, grid cell-specific emissions generated from the BEIS3.14 model - includes emissions in Canada and Mexico.
Other point sources not from the NEI: ***othpt***	Point sources from Canada's 2006 inventory and Mexico's Phase III 1999 inventory, annual resolution. Also includes annual U.S. offshore oil 2005 NEI v2 point source emissions.
Other point sources not from the NEI, Hg only: ***othpt_hg***	Year 2000 Canada speciated mercury point source emissions; annual resolution.
Other nonpoint and nonroad not from the NEI: ***othar***	Year 2006 Canada (province resolution) and year 1999 Mexico Phase III (municipio resolution) nonpoint and nonroad mobile inventories, annual resolution.
Other nonpoint sources not from the NEI, Hg only: ***othar_hg***	Year 2000 Canada speciated mercury from nonpoint sources; annual resolution.
Other onroad sources not from the NEI: ***othon***	Year 2006 Canada (province resolution) and year 1999 Mexico Phase III (municipio resolution) onroad mobile inventories, annual resolution.

The onroad emissions were primarily based on the 12/21/2009 version of the Motor Vehicle Emissions Simulator (MOVES2010) (http://www.epa.gov/otaq/models/moves/). MOVES was run with a State/month aggregation using average fuels for each state, state/month-average temperatures, and national default vehicle age distributions. The MOVES data were allocated to counties using state-county distributions from the 2005 National Mobile Inventory model (NMIM) results that are part of the 2005 NEI v2. MOVES2010 was used for onroad

sources other than in California[1] for carbon monoxide (CO), nitrogen oxides (NO_X), VOC, $PM_{2.5}$, particulate matter less than ten microns (PM_{10}), sulfur dioxide (SO_2), ammonia (NH_3), naphthalene,[2] and some VOC HAPs.[3] Since MOVES2010 does not provide emissions for all HAPs, the 2005 NEI v2 values, which came from NMIM other than for California, were used for those HAPs not provided by MOVES. Mercury was the only of these NMIM-based HAPs that was used in the modeling. To account for the temperature dependence of $PM_{2.5}$, MOVES-based temperature adjustment factors were applied to gridded, hourly emissions using the same 2005 gridded, hourly 2 meter temperature data used in CMAQ. Additional information on this approach is available in the 2005-based v4.1 platform documentation.

The nonroad emissions utilized the NMIM model (other than California[2]) to create county/month emissions, which are consistent with the annual emissions from the 2005 NEI v2.

Emissions from the point source NEI were primarily from the 2005 NEI v2 inventory, which consisted primarily 2005 values with some 2002 emissions values where 2005 was not available. The point sources are split into "EGU" (aka "ptipm") or "Non-EGU" (aka "ptnonipm") sectors for modeling purposes, based on the matching of the unit level data in the NEI units in the National Electric Energy Database System (NEEDS) version 4.10 database. All units that matched NEEDS were included in the EGU sector so that the future year emissions, which are generated by the Integrated Planning Model (IPM) based on the NEEDS units, would have a consistent universe for the existing sources. We made updates to the 2005 NEI data to remove duplicates and plants or units that were found to shutdown prior to 2005, add estimates for ethanol plants, and revise some of the 2002 data to reflect 2005 emissions based on controls that were discovered to have been put in place between 2002 and 2005.

The mercury emissions used in the modeling were from the inventory developed for the 2005 National Air Toxics Assessment (NATA), with the exceptions that (1) we replaced Hg emissions for boilers and process heaters with the emissions from the database developed as part of the Boiler MACT, which contained unit-specific Hg emissions, (2) we modified some gold mine mercury estimates, and geographic coordinates and stack parameters to account for newer data collected as part of the Gold Mine Ore Processing and Production NESHAP, and (3) we removed Hg from plants that were found to have closed prior to 2005. The NATA inventory started with the 2005 NEI v2, and was updated with data collected for some source categories

[1] California onroad emissions were taken from the California Air Resources Board submission of 2005 data to the NEI. The inventory included all criteria air pollutants other than ammonia and hazardous air pollutants.
[2] Naphthalene emissions were not used in the modeling
[3] 1,3 Butadiene, Acrolein, Formaldehyde, Benzene and Acetaldehyde. Of these, the latter 3 are used in the modeling

during the rule development process, which resulted in major updates to mercury emissions for Portland cement and hazardous waste combustion. The NATA inventory was also revised as a result of comments received as part of the state, local and tribal review. The NATA Hg emissions were also split into the "EGU" and "Non-EGU" sectors for use in the 2005v4.1 platform.

The 2005 annual NO_X and SO_2 emissions for sources in the EGU sector as defined in Table 3-1 are based primarily on data from EPA's Clean Air Markets Division's Continuous Emissions Monitoring (CEM) program, with other pollutants estimated using emission factors and the CEM annual heat input. For mercury, these emission factors were based on the 2002 emissions divided by the 2002 heat input. This approach retained the speciated mercury emissions, which had been generated for 2002 using the same speciation approach as was used for the future year emissions, whereby speciated factors were applied to units based on coal rank, firing type, boiler/burner type, and post-combustion emissions controls. For EGUs without CEMs, emissions were obtained from the state-submitted data in the NEI. Revisions to this sector between version 4 and 4.1 involved the revision and addition of ORIS plant and unit codes,[1] and for a subset of these units, annual emissions were recomputed[2] to reflect the newly matched CEM data.

For the 2005 base year, the annual EGU NEI emissions in the NEI were allocated to hourly emissions values needed for modeling based on the 2004, 2005, and 2006 CEM data. The NO_X CEM data were used to create NO_X-specific profiles, the SO_2 data were used to create SO_2-specific profiles, and the heat input data were used to allocate all other pollutants. The three years of data were used to create monthly profiles by state, while the 2005 data were used to create state-averaged profiles for allocating monthly emissions to daily. These daily values were input into SMOKE, which utilized state-averaged 2005-based hourly profiles to allocate to hourly values. This approach to temporal allocation was used for all base and control cases modeled to provide a temporal consistency that is intended to be a conceivable temporal allocation without tying the approach to a single year.

The nonpoint inventory was augmented with updated oil and gas exploration emissions from Texas and Oklahoma (CO, NO_X, PM, SO_2, VOC). These oil and gas exploration emissions were in addition to data added to the 2005 v4 platform that includes emissions within the

[1] An Oris code is a 4 digit number assigned by the Energy Information Administration (EIA) at the U.S. Department of Energy that is used to track emission generating units under numerous other data systems including the Clean Air Markets Divisions CEM data.

[2] Net change was a decrease in NO_X by 1700 tons and a decrease in SO_2 by 600 tons.

following states: Arizona, Colorado, Montana, Nevada, New Mexico, North Dakota, Oregon, South Dakota, Utah, and Wyoming.

The commercial marine category 3 (C3) vessel emissions (seca_c3 sector) used updated gridded 2005 emissions to reflect the final projections from 2002 developed for the category 3 commercial marine Emissions Control Area (ECA) proposal to the International Maritime Organization (EPA-420-F-10-041, August 2010). These updated emissions include Canada as part of the ECA, and were updated using region-specific growth rates; thus the v4.1 seca_c3 sector inventories contain Canadian province codes.

Other emissions sources included the average-year county-based inventories for emissions from wildfires and prescribed burning. These emissions are intended to be representative for both base and future years and are held constant for each, which minimizes their impact on the modeling results because of post-processing techniques. For Hg, we also used emissions of elemental mercury from natural, recycled and volcanic sources. The same approach was used in the v4 platform except that in the v4.1 platform, we reduced emissions of the natural emissions from land by 90% based on literature[1] indicating that the emissions are 10-12 tons per year as opposed to the 120 tons we had been using previously.

Additionally, the inventories were processed to provide the hourly, gridded emissions for the model-species needed by CMAQ. All of these details are further described in the 2005-based v4.1 platform documentation. Table 3-2 provides summaries of emissions by sector for the 2005 base year, for the v4.1 platform used for the modeling this rule.

[1] Gustin, M. S., Lindberg, S. E., & Weisberg, P. J. (2008). An update on the natural sources and sinks of atmospheric mercury. *Applied Geochemistry, 23*(3), 482-493.

Table 3-2. 2005 Emissions by Sector: VOC, NOX, CO, SO2, NH3, PM10, PM2.5, total and speciated HG, HCL and CL2

Sector Abbrev.	2005 VOC [tons/yr] v4.1	2005 NOX [tons/yr] v4.1	2005 CO [tons/yr] v4.1	2005 SO2 [tons/yr] v4.1	2005 NH3 [tons/yr] v4.1	2005 PM10 [tons/yr] v4.1	2005 PM2_5 [tons/yr] v4.1	2005 Total Hg (sum of 3 species) [tons/yr] v4.1	2005 Elemental Hg [tons/yr] v4.1	2005 Divalent gaseous Hg [tons/yr] v4.1	2005 Particulate Hg [tons/yr] v4.1	2005 HCL [tons/yr] v4.1	2005 CL2 [tons/yr] v4.1
afdust (see note 1)						8,858,992	1,030,391						
ag					3,251,990								
alm_no_c3	67,690	1,924,925	270,007	154,016	773	59,366	56,687	0.142	0.0793	0.0411	0.0212		1.38
seca_c3 (US component)	44,990	647,884	54,049	420,110		53,918	49,541	*	*	*	*		
seca_c3 (non-US component)	18,367	532,181	43,267	321,414		43,326	39,810	*	*	*	*		
nonpt	7,530,564	1,699,532	7,413,762	1,259,635	134,080	1,354,638	1,081,816	4.82	3.1034	1.0605	0.6524	29,001	2,135
nonroad	2,691,844	2,115,408	19,502,718	197,341	1,972	211,807	201,138	0.368	0.2105	0.1041	0.0533		
on_noadj	3,949,362	9,142,274	43,356,130	177,977	156,528	308,497	236,927	0.704	0.5036	0.1402	0.0599		
on_moves_runpm						54,071	49,789						
on_moves_startpm						22,729	20,929						
ptipm	40,950	3,726,459	601,564	10,380,786	21,684	615,095	508,903	52.9	30.1986	21.096	1.6136	351,592	99
ptnonipm	1,310,784	2,238,002	3,221,388	2,089,836	158,837	653,048	440,714	46.2	29.5686	10.4687	6.1291	48,630	4,174
avefire	1,958,992	189,428	8,554,551	49,094	36,777	796,229	684,035						
Canada othar[1]	1,281,095	734,587	3,789,362	95,086	546,034	1,666,188	432,402	2.28	0.86	1.08	0.34		
Canada othon	270,872	524,837	4,403,745	5,309	21,312	14,665	10,395						
Canada othpt	447,313	857,977	1,270,438	1,664,040	21,268	117,669	68,689	5.81	3.59	1.72	.5		
Mexico other	586,842	249,045	644,733	101,047	486,484	143,816	92,861						
Mexico othon	183,429	147,419	1,455,121	8,270	2,547	6,955	6,372						
Mexico othpt	113,044	258,510	88,957	980,359	0	125,385	88,132						
Off-shore othpt	51,240	82,581	89,812	1,961	0	839	837						

[a] fdust emissions in this table and all other summaries represent the emissions after application of the transportable fraction, which was applied to reduce emissions to reflect observed diminished transport from these sources at the scale of our modeling. Application of the transport fraction prevents the overestimation of fugitive dust impacts in the grid modeling as compared to ambient samples.

* due to uncertainty in mercury emissions from this sector, they were removed from the inventories and not used. The amount removed was on the order of 0.001 tons total mercury for U.S. and non-U.S. components of the seca_c3 sector.

Tables 3-3 through 3-5 provide state-level summaries for Hg, SO_2, and $PM_{2.5}$. In the tables below, "Nonpoint" represents the nonpt sector; "Area Fugitive Dust" (which contains only PM_{10} and $PM_{2.5}$) represents the afdust sector; on_noadj, on_startpm and on_runpm sectors are summed into "Onroad"; and nonroad, alm_no_c3 (locomotives and category 1 and 2 marine vessels) and seca_c3 (category 3 marine vessels) sectors are summed into "Nonroad." Mercury emissions are excluded from fires in both the base and future years due to uncertainty associated with these emissions.

Table 3-3. 2005 Base Year Hg Emissions (tons/year) for States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Total
Alabama	2.663	1.499	0.029	0.000	0.007	4.198
Arizona	0.716	0.208	0.030	0.000	0.007	0.961
Arkansas	0.509	0.694	0.017	0.000	0.004	1.224
California	0.005	3.389	1.488	0.480	0.390	5.751
Colorado	0.429	0.746	0.007	0.000	0.006	1.187
Connecticut	0.121	0.184	0.142	0.000	0.004	0.451
Delaware	0.180	0.219	0.000	0.002	0.001	0.402
District of Columbia	0.003	0.001	0.004	0.000	0.000	0.008
Florida	1.173	1.170	0.093	0.001	0.024	2.462
Georgia	1.704	0.690	0.065	0.000	0.013	2.473
Idaho		0.386		0.000	0.002	0.388
Illinois	4.242	1.853	0.108	0.001	0.013	6.217
Indiana	2.879	2.530	0.053	0.000	0.009	5.471
Iowa	1.158	0.705	0.026	0.000	0.004	1.893
Kansas	1.008	0.548	0.020	0.000	0.004	1.580
Kentucky	1.759	0.694	0.029	0.000	0.006	2.488
Louisiana	0.609	1.388	0.022	0.000	0.005	2.025
Maine	0.004	0.127	0.122	0.000	0.002	0.255
Maryland	0.890	0.681	0.129	0.000	0.007	1.707
Massachusetts	0.182	0.237	0.313	0.000	0.007	0.739
Michigan	1.826	1.086	0.088	0.001	0.012	3.013
Minnesota	0.707	1.977	0.043	0.001	0.007	2.734
Mississippi	0.292	0.330	0.015	0.000	0.005	0.643
Missouri	1.854	1.211	0.004	0.000	0.008	3.078
Montana	0.504	0.095	0.008	0.000	0.001	0.608
Nebraska	0.344	0.157	0.011	0.000	0.002	0.514
Nevada	0.310	2.594	0.013	0.000	0.002	2.919
New Hampshire	0.030	0.043	0.050	0.000	0.002	0.125
New Jersey	0.133	0.761	0.233	0.000	0.009	1.137
New Mexico	1.027	0.035	0.010	0.000	0.003	1.076
New York	0.465	0.916	0.614	0.001	0.018	2.014
North Carolina	1.716	0.638	0.091	0.001	0.011	2.456
North Dakota	1.123	0.045	0.011	0.000	0.001	1.180
Ohio	3.662	2.059	0.110	0.001	0.013	5.845

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Total
Oklahoma	0.927	0.379	0.020	0.000	0.006	1.332
Oregon	0.081	1.561	0.060	0.000	0.004	1.706
Pennsylvania	4.979	2.684	0.264	0.001	0.013	7.940
Rhode Island		0.047	0.033	0.000	0.001	0.081
South Carolina	0.581	1.202	0.030	0.000	0.006	1.819
South Dakota	0.048	0.071	0.009	0.000	0.001	0.129
Tennessee	1.251	1.746	0.034	0.000	0.008	3.040
Texas	5.196	4.650	0.073	0.001	0.027	9.947
Tribal Data		0.001		0.000		0.001
Utah	0.148	0.369	0.015	0.000	0.003	0.536
Vermont	0.006	0.001	0.033	0.000	0.001	0.040
Virginia	0.624	1.743	0.100	0.000	0.010	2.477
Washington	0.339	0.202	0.050	0.013	0.007	0.611
West Virginia	2.404	0.454	0.019	0.000	0.002	2.880
Wisconsin	1.147	0.887	0.072	0.001	0.007	2.114
Wyoming	0.949	0.275	0.004	0.000	0.001	1.229
TOTAL	**52.9**	**46.2**	**4.8**	**0.5**	**0.7**	**105.1**

Table 3-4. 2005 Base Year SO2 Emissions (tons/year) for States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	460,123	70,346	52,325	6,392	3,983	983	594,151
Arizona	52,733	23,966	2,571	6,154	3,919	2,888	92,231
Arkansas	66,384	13,066	27,260	5,678	1,998	728	115,114
California	601	33,136	77,672	102,317	4,935	6,735	225,395
Colorado	64,174	1,549	6,810	4,897	3,064	1,719	82,213
Connecticut	10,356	1,831	18,455	2,556	1,375	4	34,576
Delaware	32,378	34,859	5,859	11,746	519	6	85,367
District of Columbia	1,082	686	1,559	414	218	0	3,961
Florida	417,321	57,475	70,490	93,772	13,280	7,018	659,356
Georgia	616,054	54,502	56,829	13,386	7,163	2,010	749,945
Idaho	0	17,151	2,915	2,304	951	3,845	27,166
Illinois	330,382	156,154	5,395	19,303	7,279	20	518,532
Indiana	878,978	87,821	59,775	9,437	4,937	24	1,040,972
Iowa	130,264	64,448	19,832	8,838	2,045	25	225,451
Kansas	136,520	13,235	36,381	8,035	2,241	103	196,515
Kentucky	502,731	25,965	34,229	6,942	3,377	364	573,607
Louisiana	109,851	165,737	2,378	73,001	3,043	892	354,902
Maine	3,887	18,519	9,969	3,752	986	150	37,264
Maryland	283,205	34,988	40,864	17,929	2,706	32	379,723
Massachusetts	84,234	19,620	25,261	25,547	2,819	93	157,575
Michigan	349,877	76,510	42,066	14,597	8,966	91	492,106
Minnesota	101,666	25,169	14,747	10,412	3,111	631	155,736
Mississippi	75,047	29,892	6,796	5,999	2,681	1,051	121,466
Missouri	284,384	78,307	44,573	10,464	5,339	186	423,253
Montana	19,715	11,056	2,600	3,813	912	1,422	39,518

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Total
Nebraska	74,955	6,469	29,575	9,199	1,640	105	121,942
Nevada	53,363	2,253	12,477	2,877	702	1,346	73,018
New Hampshire	51,445	3,245	7,408	805	780	38	63,721
New Jersey	57,044	7,640	10,726	23,659	3,112	61	102,242
New Mexico	30,628	8,062	3,193	3,541	1,879	3,450	50,755
New York	180,847	58,562	125,158	20,990	6,500	113	392,170
North Carolina	512,231	59,433	22,020	43,094	6,506	696	643,980
North Dakota	137,371	9,678	6,455	5,986	525	66	160,082
Ohio	1,116,084	115,165	19,810	15,630	7,715	22	1,274,427
Oklahoma	110,081	40,482	8,556	5,015	3,316	469	167,918
Oregon	12,304	9,825	9,845	13,862	1,872	4,896	52,603
Pennsylvania	1,002,202	83,376	68,349	11,999	6,597	32	1,172,554
Rhode Island	176	2,743	3,365	2,515	265	1	9,065
South Carolina	218,781	31,495	30,016	20,639	3,741	646	305,318
South Dakota	12,215	1,999	10,347	3,412	612	498	29,083
Tennessee	266,148	67,160	32,714	6,288	6,088	277	378,676
Texas	534,949	223,625	115,192	52,643	17,970	1,178	945,556
Tribal	3	1,511		0			1,515
Utah	34,813	9,132	3,577	2,439	1,999	1,934	53,893
Vermont	9	902	5,385	385	346	49	7,078
Virginia	220,248	69,440	32,923	18,523	4,647	399	346,181
Washington	3,409	24,211	7,254	28,345	3,490	407	67,115
West Virginia	469,456	48,314	14,589	2,133	1,289	215	535,996
Wisconsin	180,200	66,806	6,369	7,134	3,735	70	264,314
Wyoming	89,874	22,321	6,721	2,674	807	1,106	123,503
TOTAL	**10,380,786**	**2,089,836**	**1,259,635**	**771,467**	**177,977**	**49,094**	**14,728,796**

*Non-US seca_c3 component not included. These emissions are 321,414 tons/yr.

Table 3-5. 2005 Base Year PM2.5 Emissions (tons/year) for States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Area Fugitive Dust	Total
Alabama	23,487	19,871	23,973	4,237	5,931	13,938	11,582	103,019
Arizona	7,506	3,940	8,596	4,486	7,249	37,151	12,806	81,733
Arkansas	1,761	10,872	23,062	3,803	3,222	10,315	11,681	64,717
California	1,461	21,516	73,873	30,062	22,303	97,302	20,327	266,843
Colorado	4,525	7,114	13,545	3,960	4,554	24,054	11,794	69,546
Connecticut	612	224	10,446	1,740	2,620	56	1,014	16,712
Delaware	2,193	2,225	1,826	1,025	973	87	497	8,826
District of Columbia	17	172	427	277	386	0	162	1,441
Florida	26,142	25,196	38,847	22,728	16,844	99,484	14,108	243,349
Georgia	28,016	12,936	41,847	6,922	12,835	24,082	21,286	147,925
Idaho	1	2,072	27,367	2,140	1,541	52,808	14,125	100,053
Illinois	16,654	15,683	15,181	12,880	13,272	277	58,864	132,812

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Area Fugitive Dust	Total
Indiana	35,056	14,262	32,611	6,515	8,137	344	41,832	138,757
Iowa	8,905	5,904	11,476	6,969	3,706	349	42,837	80,146
Kansas	5,592	7,634	83,174	5,719	3,186	1,468	55,263	162,036
Kentucky	19,936	10,455	18,590	4,762	5,790	5,155	12,655	77,343
Louisiana	5,656	39,591	17,862	15,320	4,474	12,647	10,302	105,851
Maine	98	3,785	13,726	1,627	1,805	2,127	1,312	24,480
Maryland	15,570	6,768	19,764	4,472	5,668	531	3,559	56,332
Massachusetts	3,293	2,245	26,536	5,651	6,091	1,324	4,580	49,720
Michigan	11,375	12,918	24,216	8,702	13,437	1,283	23,506	95,437
Minnesota	3,228	10,651	24,496	8,541	7,019	8,943	49,495	112,372
Mississippi	2,845	10,602	16,769	4,142	4,297	14,897	17,447	71,000
Missouri	6,525	6,948	28,217	7,230	7,992	2,636	48,202	107,750
Montana	2,399	2,729	5,569	2,654	1,496	17,311	24,528	56,686
Nebraska	1,255	1,858	8,655	5,848	2,768	1,483	37,482	59,349
Nevada	3,397	4,095	2,735	2,171	1,301	19,018	7,185	39,902
New Hampshire	2,677	572	12,658	909	1,553	534	658	19,560
New Jersey	5,015	2,599	13,074	6,327	6,219	865	549	34,648
New Mexico	5,670	1,463	5,346	1,959	3,005	48,662	45,353	111,458
New York	10,466	5,000	34,893	9,267	11,582	1,601	13,647	86,456
North Carolina	16,990	12,665	38,389	10,533	9,096	9,870	11,162	108,706
North Dakota	6,397	576	3,241	4,552	1,037	934	38,263	55,001
Ohio	53,570	12,890	23,761	9,868	12,136	316	28,587	141,128
Oklahoma	1,973	6,246	45,804	3,765	4,690	6,644	44,243	113,366
Oregon	479	8,852	49,407	4,751	3,504	65,350	8,738	141,080
Pennsylvania	55,621	14,772	31,263	7,565	11,544	454	13,344	134,564
Rhode Island	47	256	1,107	605	605	14	182	2,816
South Carolina	14,466	4,779	18,139	4,950	5,304	9,163	9,160	65,962
South Dakota	391	2,882	4,463	2,910	1,114	7,062	29,215	48,037
Tennessee	12,872	22,279	20,663	5,072	8,750	3,934	11,900	85,470
Texas	24,900	37,563	50,339	23,551	31,198	21,578	143,698	332,825
Tribal	17	1,569		0	0			1,586
Utah	5,078	3,595	9,079	1,627	2,791	27,412	5,682	55,264
Vermont	37	337	5,415	479	645	696	1,528	9,137
Virginia	12,388	11,504	29,947	7,009	6,943	5,659	8,194	81,644
Washington	2,444	4,618	31,983	7,864	6,878	4,487	13,617	71,890
West Virginia	26,385	5,161	11,130	1,702	2,008	3,050	3,649	53,085
Wisconsin	5,449	7,973	25,407	6,062	6,907	994	11,870	64,662
Wyoming	8,068	10,296	2,922	1,455	1,238	15,686	28,723	68,388
TOTAL	**508,903**	**440,714**	**1,081,816**	**307,367**	**307,645**	**684,035**	**1,030,391**	**4,360,871**

*Non-US seca_c3 component not included. These emissions are 39,810 tons/yr.

3.3 Development of Future Year Base Case Emissions

The 2016 base case scenario represents predicted emissions including known Federal measures for all sectors. It reflects projected economic changes and fuel usage for the EGU and mobile sectors. Emissions from non-EGU stationary sectors have previously been shown to not be well correlated with economic forecasts, and therefore economic impacts were not included for non-EGU stationary sources. Like the 2005 base case, this emissions case includes criteria pollutants, mercury, hydrogen chloride, and chlorine from non-EGU sources, and, for some sectors benzene, formaldehyde, acetaldehyde and methanol from the inventory is used in VOC speciation. It does not include metals nor other non-mercury HAPs except for those mentioned above.

The 2016 base case EGU emissions projections of mercury, hydrogen chloride, SO_2, and PM were obtained from an interim version 4.10 of the Integrated Planning Model (IPM) (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html). The IPM is a multiregional, dynamic, deterministic linear programming model of the U.S. electric power sector. Version 4.10 reflects state rules and consent decrees through December 1, 2010, and incorporates information on existing controls collected through the Information Collection Request (ICR) for the proposed Toxics Rule. Units with SO_2 or NO_X advanced controls (e.g., scrubber, SCR) that were not required to run for compliance with Title IV, New Source Review (NSR), state settlements, or state-specific rules were modeled by IPM to either operate those controls or not based on economic efficiency parameters. Units with advanced mercury controls (e.g., ACI) were assumed to operate those controls in states with mercury requirements. Note that this base case includes the proposed Transport Rule, which will be finalized in June, 2011. Speciated emissions were estimated using mercury speciation factors, which are assigned based on coal rank, firing type, boiler/burner type, and post-combustion emissions controls. These are the same factors as were used in the Clean Air Mercury rule and are provided in Appendix A. Further details on the EGU emissions inventory used for this proposal can be found in the IPM Documentation.

The length of time required to conduct emissions and photochemical modeling precluded the use of the final version IPM version 4.10. Thus the air quality modeling for the proposed Toxics Rule relied on electric generating unit (EGU) emission projections from an interim IPM platform that was subsequently updated during the rulemaking process for the proposed Toxics Rule policy analysis. The updated emissions were not included in the air quality modeling. The updated baseline emission projection was based on an updated IPM platform, which resulted in emissions changes to the EGU sector only. The IPM update reflects additional information obtained primarily from the 2010 ICR and from comments submitted on an IPM Notice of Data

Availability (NODA) in October 2010. Notably, this IPM update included the addition of over 20 GW of existing ACI reported to EPA via the ICR, which explains the majority of the difference in interim and final base case EGU mercury projections. This update also includes additional unit-level updates that were made based on the ICR and public comments on the IPM NODA which identified additional existing pollution controls (such as scrubbers). Additionally, this update corrected an erroneous natural gas emission factor which was responsible for an over-prediction in $PM_{2.5}$ emissions from the EGU sector of 85 thousand tons. Other updates includes adjustments to assumptions regarding the performance of acid gas control technologies, new costs imposed on fuel-switching (e.g., bituminous to sub-bituminous), correction of lignite availability to some plants, incorporation of additional planned retirements, a more inclusive implementation of the scrubber upgrade option, and the availability of a scrubber retrofit to waste-coal fired fluidized bed combustion units without an existing scrubber. Further details on the future year EGU emissions inventory used for this proposal can be found in the IPM v.4.10 Documentation, available at http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html.

Prior to emissions processing through SMOKE, the IPM results were adjusted to account for the impact of the Boiler MACT which resulted in a reduction of roughly 20,000 tons of SO_2 and 460 tons of HCL. This adjustment was not applied to the final IPM version. Mobile source inventories of onroad and nonroad mobile emissions were created for 2016 using a combination of the NMIM and MOVES models in a consistent approach with the 2005 base year. As with the 2005 emissions, the 2016 onroad emissions were based on MOVES 2010. Future-year vehicle miles travelled (VMT) were projected from the 2005 NEI v2 VMT using growth rates from the 2009 Annual Energy Outlook (AEO) data. The same MOVES-based $PM_{2.5}$ temperature adjustment factors were also applied as in 2005 for running mode emissions because these are not dependent on year; however, cold start emissions used 2015-specific temperature adjustment factors.

The 2016 onroad emissions reflect control program implementation through 2016 and include the Light-Duty Vehicle Tier 2 Rule, the Onroad Heavy-Duty Rule, and the Mobile Source Air Toxics (MSAT) final rule. Emission reductions and increases from the Renewable Fuel Standard version 2 (RFS2) are not included.

Nonroad mobile emissions were created only with NMIM using a consistent approach as was used for 2005, but emissions were calculated using NMIM future-year equipment population estimates and control programs for 2015 and then adjusted to 2016 using national level factors. Emissions for locomotives and category 1 and 2 (C1 and C2) commercial marine vessels were

derived for 2016 based on emissions published in the Locomotive Marine Rule, Regulatory Impact Assessment, Chapter 3 (see http://www.epa.gov/otaq/locomotives.htm#2008final).

The future baseline case nonroad mobile emissions reductions for these years include reductions to locomotives, various nonroad engines including diesel engines and various marine engine types, fuel sulfur content, and evaporative emissions standards, including the category 3 marine diesel engines and International Maritime Organization standards which include the establishment of emission control areas for these ships. A summary of the mobile source control programs included in the projected future year baseline is shown in Table 3-6.

Table 3-6. Summary of Mobile Source Control Programs Included in 2016 Baseline

National Onroad Rules:
Tier 2 Rule (*Signature date:* February 28, 2000)
Onroad Heavy-Duty Rule (February 24, 2009)
Final Mobile Source Air Toxics Rule (MSAT2) (February 9, 2007)
Renewable Fuel Standard (March 26, 2010)
Local Onroad Programs:
National Low Emission Vehicle Program (NLEV) (March 2, 1998)
Ozone Transport Commission (OTC) LEV Program (January, 1995)
National Nonroad Controls:
Tier 1 nonroad diesel rule (June 17, 2004)
Phase 1 nonroad SI rule (July 3, 1995)
Marine SI rule (October 4, 1996)
Nonroad diesel rule (October 23, 1998)
Phase 2 nonroad nonhandheld SI rule (March 30, 1999)
Phase 2 nonroad handheld SI rule (April 25, 2000)
Nonroad large SI and recreational engine rule (November 8, 2002)
Clean Air Nonroad Diesel Rule - Tier 4 (June 29, 2004)
Locomotive and marine rule (May 6, 2008)
Nonroad SI rule (October 8, 2008)
Aircraft:
Itinerant (ITN) operations at airports adjusted to year 2016
Locomotives:
Clean Air Nonroad Diesel Final Rule – Tier 4 (June 29, 2004)
Locomotive rule (April 16, 2008)
Locomotive and marine rule (May 6, 2008)
Commercial Marine:
Locomotive and marine rule (May 6, 2008)
Category 3 marine diesel engines Clean Air Act and International Maritime Organization standards (April, 30, 2010)

In the 2016 base case, we used the 2005 base year emissions for Canada and Mexico because appropriate future-year emissions for sources in these countries were not available. The

future-year emissions need to reflect expected percent reductions or increases between the base year and the future year to be considered appropriate for this type of modeling.

For non-EGU point sources, emissions were projected by including emissions reductions and increases from a variety of source data.[11] For non-EGU point sources, other than for certain large municipal waste combustors and airports, emissions were not grown using economic growth projections, but rather were held constant at the emissions levels in 2005. Emissions reductions were applied to non-EGU point source to reflect final federal measures, known plant closures, refinery and other consent decrees. The starting point was the emission projections done for the 2005v4 platform for the proposed Transport Rule. The 2014 projection factors developed for the Transport Rule proposal (see http://www.epa.gov/ttn/chief/emch/index.html#transport) were updated for these 2016 baseline projections. Several additional NESHAP were promulgated since emission projections were done for the proposed Transport Rule, and these were included for the 2016 base case. Emission reductions were also applied to include the impact of the Boiler MACT, which had been proposed at the time of the analysis, and finalized in February 2011. This approach, which utilized information developed between the proposed and final rule, resulted in the reduction of roughly 400,000 tons of SO_2, 5,600 tons HCL and 1.8 tons of Hg nationwide. In addition, the projection includes local controls for NO_X and VOC from the New York State Implementation Plan (SIP) as part of another effort; we do not anticipate that this change significantly impacts the results of this RIA, which are primarily resulting from changes to SO_2 and $PM_{2.5}$.

Since aircraft at airports were treated as point emissions sources in the 2005 NEI v2, we applied projection factors based on activity growth projected by the Federal Aviation Administration Terminal Area Forecast (TAF) system, published December 2008 for these sources.

The mercury emission projections included NESHAP for non-EGU source categories that were finalized or expected to be finalized prior to the proposed Toxics rule including the Boiler MACT (1.8 tons reduction), Portland Cement NESHAP (6.4 tons reduction), Gold Mines NESHAP (1.8 tons reduction), Electric Arc Furnaces NESHAP (2.4 tons reduction), Mercury Cell Chlor-Alkali NESHAP (2.8 tons reduction) and Hazardous Waste Combustion NESHAP (1.1 ton

[11] Controls from the NO_X SIP call were assumed to have been in place by 2005 and captured in the 2005 NEI v2.

reduction[12]) In addition, the projections included reduction of Hg emissions due to the replacement of a smelter with a recovery boiler at a pulp and paper plant (0.7 tons reduction).

Emissions from stationary nonpoint sources were projected using procedures specific to individual source categories. Refueling emissions were projected using the refueling results from the NMIM runs performed for the onroad mobile sector. Portable fuel container emissions were projected using estimates from previous rulemaking inventories compiled by the Office of Transportation and Air Quality (OTAQ). Emissions of ammonia and dust from animal operations were projected based on animal population data from the Department of Agriculture and EPA. Residential wood combustion was projected by replacement of obsolete woodstoves with new woodstoves and a 1 percent annual increase in fireplaces. Landfill emissions were projected using MACT controls. In addition, many of the NY SIP controls applied to nonpoint categories and were included in the projection. All other nonpoint sources were held constant between 2005 and the 2016 future year scenarios.

A summary of all rules and growth assumptions impacting non-EGU stationary sources is provided in Table 3-7. The table is broken out into two sections: (1) the approaches used to project emissions for the proposed Transport Rule that were carried forward for the proposed Toxics Rule and (2) the added controls/reductions used for the proposed Toxics rule that had not been used for the proposed Transport rule.

Table 3-7. Control Strategies and/or Growth Assumptions Included in the 2016 Projection for Non EGU Stationary Sources

Control Strategies and/or Growth Assumptions Applied to 2005 emissions for the 2016 projection	
Projection Approaches Carried Forward from the Proposed Transport Rule[1,2]	
MACT rules, national, VOC: national applied by SCC, MACT	VOC
Consent Decrees and Settlements, including refinery consent decrees, and settlements for: Alcoa, TX and Premcor (formerly MOTIVA), DE	All
Municipal Waste Combustor Reductions –plant level	PM
Hazardous Waste Combustion	PM
Hospital/Medical/Infectious Waste Incinerator Regulations	NOx, PM, SO_2
Large Municipal Waste Combustors – growth applied to specific plants	All
MACT rules, plant-level, VOC: Auto Plants	VOC
MACT rules, plant-level, PM & SO2: Lime Manufacturing	PM, SO_2
MACT rules, plant-level, PM: Taconite Ore	PM
Municipal Waste Landfills: project factor of 0.25 applied	All
Livestock Emissions Growth from year 2002 to year 2016	NH3, PM

[12] Actual reduction for hazardous waste reduction should have been 0.2 tons, but due to an error in the percentage applied, a higher value was reduced.

Residential Wood Combustion Growth and Change-outs from year 2005 to Year 2016	All
Gasoline Stage II growth and control from year 2005 to year 2016	VOC
Portable Fuel Container Mobile Source Air Toxics Rule 2: inventory growth and control from year 2005 to year 2016	VOC
Additional Projection Approaches For the Proposed Toxics Rule[3]	
NESHAP: Portland Cement (09/09/10) – plant level based **on Industrial Sector Integrated Solutions (ISIS)** policy emissions in 2013. The ISIS results are from the ISIS-Cement model runs for the NESHAP and NSPS analysis of July 28, 2010 and include closures.	Hg, NO_X, SO2, PM, HCL
NESHAP: Industrial, Commercial, Institutional (ICI) Boilers, aka "Boiler MACT" (signed 02/21/2011)	Hg, SO_2, HCL, PM
NESHAP: Gold Mine Ore Processing and Production Area Source Category (based on proposed rule 04-15-10) – finalized 12/2010	Hg
NESHAP: Mercury Emissions From Mercury Cell Chlor-Alkali Plants-Final Rule (12/19/03)	Hg
Pulp and Paper Project smelter replacement for Georgia Pacific plant in VA (12/2009)	Hg
NESHAP: Electric Arc Furnace Steelmaking Facilities (12/28/2007)	Hg
NESHAP: Hazardous Waste Combustion (12/19/2005)	Hg
New York ozone SIP standards	VOC, HAP VOC, NO_X
Additional Plant and Unit closures provided by state, regional, and EPA agencies	All
Emission Reductions resulting from controls put on specific boiler units (not due to MACT) after 2005, identified through analysis of the control data gathered from the ICR from the ICI Boiler NESHAP.	NO_X, SO_2, HCL
NESHAP: Reciprocating Internal Combustion Engines (RICE)[4]	NO_X, CO, PM
Use Phase II WRAP 2018 Oil and Gas, and apply RICE controls to these emissions	VOC, SO_2, NO_X, CO
Use 2008 Oklahoma and Texas Oil and Gas, and apply RICE controls to these emissions	VOC, SO_2, NO_X, CO, PM

1. They were changed to reflect a 2016 future year, rather than 2012 / 2014
2. We inadvertently did not apply closures that had been applied for the Transport Rule proposal; emissions from these plants sum to 3300 tons VOC, 178 tons PM2.5, 1982 tons SO2, 1639 tons NOX, 6 tons NH3 and 379 tons CO. At the state level, the largest impact is in West Virginia (717 tons NOX, which is 2% of emissions in ptnonipm) and 1604 tons SO2 which is 7% of the ptnonipm sector. When considering emissions from other sectors, the percentages will be much smaller. All other errors are under 500 tons (less than 1% of the ptnonipm sector). This omission is expected to have negligible impacts on our analysis since the reductions were omitted from both the base and policy cases.
3. Note that SO2 reductions are expected to occur to due fuel sulfur limits but were excluded from the projection. They were expected to reduce SO2 by 27,000 tons, nationwide. This omission is expected to have negligible impacts on our analysis since the reductions were omitted from both the base and policy cases.
4. Due to a software issue, emission reductions from the LaFarge and SaintGobain consent decrees (January 2010) were not included in the projection. The resulting emissions are therefore too high in CA, IL, IN, KS, LA, MA, MI, MO, NC, OH, OK, PA, TX, WA, and WI, and are summarized nationally below. Although these missed reductions are large, they have a minimal impact on our overall analysis because the modeling analysis for the RIA captures an appropriate difference between the future base and policy cases and that difference is unaffected by this omission since it was omitted from both the base and the policy cases.

CO (tons)	NOX (tons)	PM10 (tons)	PM2_5 (tons)	SO2 (tons)	VOC (tons)
110	13,214	269	210	16,270	6

Table 3-8 shows a summary of the 2005 and 2016 modeled base case emissions for the sum of the lower 48 states. Tables 3-9 to 3-11 below provide summaries of Hg, SO_2 and $PM_{2.5}$ in the 2016 base case for each sector by state.

Table 3-8. Summary of Modeled Base Case Annual Emissions (tons/year) for 48 States by Sector: Hg, SO_2 and $PM_{2.5}$

Source Sector Hg Emissions	**2005**	**2016**
EGU Point	53	29
Non-EGU Point	46	29
Nonpoint	4.8	5
Nonroad	0.5	0.5
On-road	0.7	0.7
Average Fire		
Total HG, All Sources	105	64
Source Sector SO_2 Emissions		
EGU Point	10,380,786	3,577,698
Non-EGU Point	2,089,836	1,349,038
Nonpoint	1,259,635	1,250,300
Nonroad	771,467	35,616
On-road	177,977	26,784
Average Fire	49,094	49,094
Total SO_2, All Sources	**14,728,795**	**6,288,530**
Source Sector $PM_{2.5}$ Emissions		
EGU Point	508,903	384,320*
Non-EGU Point	440,714	404,926
Nonpoint plus Area Fugitive Dust	2,112,207	2,071,484
Nonroad	307,366	169,144
On-road	307,645	188,320**
Average Fire	684,035	684,035
Total $PM_{2.5}$, All Sources	**4,360,870**	**3,902,229**

*PM2.5 based on modeled value. Subsequent IPM run with updated base case and correction to natural gas emission factor resulted in 285,253 tons.

**On-road PM2.5 for 2016 had two errors which were not able to be corrected prior to the AQ modeling, resulting in a national level over-estimate of 86,000 tons in the 2016 case, which is 2% of the total PM2.5 emissions from the continental U.S.

Table 3-9. 2016 Base Case Hg Emissions (tons/year) for Lower 48 States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Total
Alabama	1.2550	0.6869	0.0293	0.0003	0.0068	1.98
Arizona	0.7487	0.0742	0.0300	0.0003	0.0070	0.86
Arkansas	0.7246	0.5523	0.0171	0.0002	0.0038	1.30
California	0.1322	2.0271	1.4881	0.4799	0.3898	4.52
Colorado	0.0832	0.6339	0.0068	0.0003	0.0058	0.73
Connecticut	0.0069	0.1839	0.1422	0.0002	0.0039	0.34
Delaware	0.0090	0.0353	0.0001	0.0019	0.0011	0.05
District of Columbia		0.0008	0.0040	0.0000	0.0005	0.01
Florida	0.4859	0.6206	0.0929	0.0013	0.0243	1.23
Georgia	0.5115	0.2056	0.0653	0.0005	0.0130	0.80
Idaho		0.3758		0.0001	0.0016	0.38
Illinois	0.4879	1.5530	0.1080	0.0007	0.0129	2.16
Indiana	1.5583	2.0018	0.0528	0.0004	0.0085	3.62
Iowa	0.9994	0.3602	0.0258	0.0003	0.0038	1.39
Kansas	0.9551	0.3645	0.0201	0.0002	0.0035	1.34
Kentucky	0.8278	0.4658	0.0289	0.0002	0.0058	1.33
Louisiana	1.0188	0.3517	0.0220	0.0004	0.0053	1.40
Maine	0.0129	0.0889	0.1221	0.0001	0.0018	0.23
Maryland	0.1144	0.4264	0.1287	0.0003	0.0068	0.68
Massachusetts	0.0094	0.2339	0.3130	0.0003	0.0070	0.56
Michigan	1.5010	0.6395	0.0884	0.0009	0.0122	2.24
Minnesota	0.1610	1.8691	0.0432	0.0005	0.0066	2.08
Mississippi	0.4048	0.2666	0.0155	0.0002	0.0048	0.69
Missouri	1.9487	0.8189	0.0041	0.0004	0.0083	2.78
Montana	0.0968	0.0708	0.0079	0.0001	0.0013	0.18
Nebraska	0.4228	0.1092	0.0108	0.0001	0.0023	0.55
Nevada	0.0874	0.7880	0.0127	0.0001	0.0024	0.89
New Hampshire	0.0108	0.0272	0.0499	0.0002	0.0016	0.09
New Jersey	0.0257	0.6580	0.2333	0.0005	0.0093	0.93
New Mexico	0.2958	0.0110	0.0101	0.0001	0.0029	0.32
New York	0.0510	0.6600	0.6138	0.0009	0.0181	1.34
North Carolina	0.4868	0.5493	0.0911	0.0005	0.0105	1.14
North Dakota	0.9364	0.0268	0.0114	0.0001	0.0009	0.98
Ohio	1.5759	1.2379	0.1096	0.0006	0.0132	2.94
Oklahoma	1.0284	0.2605	0.0204	0.0002	0.0056	1.32
Oregon	0.0075	0.2949	0.0601	0.0003	0.0041	0.37
Pennsylvania	1.6132	1.9226	0.2642	0.0006	0.0130	3.81
Rhode Island		0.0466	0.0333	0.0000	0.0011	0.08
South Carolina	0.3472	0.8218	0.0302	0.0003	0.0058	1.21
South Dakota	0.0272	0.0241	0.0088	0.0001	0.0010	0.06

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Total
Tennessee	0.7427	0.7705	0.0341	0.0003	0.0082	1.56
Texas	3.3673	3.6445	0.0728	0.0011	0.0268	7.11
Tribal Data		0.0011		0.0000		0.00
Utah	0.1838	0.2064	0.0150	0.0001	0.0030	0.41
Vermont		0.0010	0.0327	0.0001	0.0009	0.03
Virginia	0.2842	0.7885	0.1000	0.0004	0.0099	1.18
Washington	0.1666	0.1044	0.0504	0.0127	0.0065	0.34
West Virginia	0.8600	0.3142	0.0191	0.0001	0.0023	1.20
Wisconsin	0.8701	0.8148	0.0720	0.0006	0.0071	1.76
Wyoming	1.2596	0.1922	0.0042	0.0000	0.0011	1.46
Total	**28.7**	**29.2**	**4.8**	**0.510**	**0.704**	**63.9**

Table 3-10. 2016 Base Case SO_2 Emissions (tons/year) for Lower 48 States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Total
Alabama	172,198	65,649	52,312	197	513	983	291,850
Arizona	23,140	24,206	2,566	52	626	2,888	53,477
Arkansas	93,754	12,910	27,255	142	286	728	135,075
California	4,740	22,148	77,610	8,489	2,216	6,735	121,938
Colorado	55,588	1,425	6,469	47	529	1,719	65,778
Connecticut	2,643	1,832	18,438	100	275	4	23,291
Delaware	1,717	6,299	5,857	715	79	6	14,673
District of Columbia	0	686	1,559	3	36	0	2,284
Florida	122,123	40,662	70,479	4,530	1,901	7,018	246,713
Georgia	91,885	42,407	56,812	430	1,108	2,010	194,652
Idaho	0	17,137	2,911	21	167	3,845	24,082
Illinois	148,934	85,834	5,380	319	1,036	20	241,524
Indiana	229,248	64,088	59,764	160	675	24	353,959
Iowa	98,518	19,010	19,816	85	291	25	137,745
Kansas	61,622	12,708	36,374	55	257	103	111,119
Kentucky	123,010	18,773	34,208	257	436	364	177,048
Louisiana	98,808	146,371	2,371	3,979	402	892	252,824
Maine	1,123	7,803	9,943	194	131	150	19,345
Maryland	36,211	13,623	40,850	1,055	513	32	92,284
Massachusetts	4,236	16,168	25,235	1,368	497	93	47,597
Michigan	169,853	24,072	42,066	440	919	91	237,440
Minnesota	51,952	18,728	14,727	252	500	631	86,789
Mississippi	55,317	22,327	6,785	244	332	1,051	86,055
Missouri	172,031	65,392	44,540	214	652	186	283,016
Montana	13,234	7,858	1,959	24	105	1,422	24,603
Nebraska	74,642	4,777	29,569	55	181	105	109,329
Nevada	11,283	2,134	12,474	25	187	1,346	27,449
New Hampshire	4,348	2,578	7,391	22	120	38	14,496
New Jersey	8,507	6,758	10,711	1,300	661	61	27,998
New Mexico	11,370	8,065	2,833	24	237	3,450	25,978
New York	28,911	20,812	125,199	979	1,303	113	177,318
North Carolina	82,544	45,264	21,992	2,177	811	696	153,484
North Dakota	76,081	9,678	5,766	35	62	66	91,688
Ohio	204,291	58,216	19,810	422	969	22	283,731
Oklahoma	139,800	31,097	7,535	45	436	469	179,382
Oregon	11,102	8,597	9,846	787	369	4,896	35,598

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Total
Pennsylvania	152,929	46,609	68,322	458	981	32	269,332
Rhode Island	0	2,725	3,364	129	72	1	6,291
South Carolina	128,070	22,746	30,001	1,037	462	646	182,963
South Dakota	29,711	1,947	10,298	22	76	498	42,552
Tennessee	106,762	39,433	32,695	173	695	277	180,036
Texas	334,636	138,883	110,147	2,103	2,084	1,178	589,030
Tribal	0	1,495		0			1,495
Utah	31,343	8,034	3,425	25	297	1,934	45,057
Vermont	0	903	5,379	7	90	49	6,428
Virginia	45,345	47,045	32,897	771	756	399	127,213
Washington	2,804	19,131	7,227	1,432	654	407	31,655
West Virginia	127,826	23,305	14,580	75	161	215	166,162
Wisconsin	77,871	18,573	6,370	123	554	70	103,561
Wyoming	55,636	22,118	6,180	18	86	1,106	85,146
Total	**3,577,698**	**1,349,038**	**1,250,300**	**35,616**	**26,784**	**49,094**	**6,288,529**
*Non-US seca_c3 component not included. These emissions are 957,065 tons/yr.							

Table 3-11. 2016 Base Case PM2.5 Emissions (tons/year) for Lower 48 States by Sector

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Area Fugitive Dust	Total
Alabama	14,801	17,064	22,982	2,576	1,631	13,938	11,591	84,583
Arizona	10,196	3,804	8,178	2,836	1,817	37,151	12,806	76,788
Arkansas	3,805	9,905	22,683	2,191	1,108	10,315	11,681	61,689
California	9,718	20,859	69,736	17,963	17,777	97,302	20,386	253,741
Colorado	4,972	7,007	12,854	2,490	4,373	24,054	11,794	67,544
Connecticut	1,632	225	9,303	1,090	2,988	56	1,014	16,308
Delaware	643	1,906	1,675	477	514	87	497	5,801
District of Columbia	0	172	407	151	229	0	162	1,121
Florida	26,114	18,264	37,931	10,096	4,168	99,484	14,126	210,183
Georgia	14,411	12,161	40,435	4,131	3,803	24,082	21,286	120,309
Idaho	187	2,067	27,023	1,267	1,555	52,808	14,154	99,060
Illinois	11,157	14,266	13,753	7,429	10,062	277	58,864	115,808
Indiana	21,198	13,572	31,618	3,769	5,586	344	41,832	117,919
Iowa	5,223	5,688	10,176	3,593	3,816	349	42,837	71,682
Kansas	4,634	7,556	82,581	3,078	1,736	1,468	55,263	156,315
Kentucky	13,598	10,341	16,928	2,899	2,342	5,155	12,655	63,917
Louisiana	5,219	36,644	17,365	6,491	1,000	12,647	10,302	89,669
Maine	712	3,143	11,958	985	1,876	2,127	1,312	22,114
Maryland	3,791	6,153	18,742	2,304	3,584	531	3,559	38,665
Massachusetts	2,754	2,127	24,749	2,531	5,278	1,324	4,580	43,343
Michigan	7,188	11,115	22,374	5,048	10,955	1,283	23,506	81,470
Minnesota	9,011	9,665	22,535	5,035	10,917	8,943	49,495	115,600
Mississippi	2,554	9,491	15,685	2,495	876	14,897	17,454	63,451
Missouri	8,040	6,334	25,550	4,217	4,335	2,636	48,202	99,315
Montana	2,453	2,528	4,925	1,427	1,239	17,311	24,528	54,412

State	EGU	NonEGU	Nonpoint	Nonroad	Onroad	Fires	Area Fugitive Dust	Total
Nebraska	2,657	1,857	8,177	3,177	1,760	1,483	37,482	56,593
Nevada	10,903	4,029	2,612	1,364	732	19,018	7,185	45,843
New Hampshire	1,138	508	11,543	610	1,588	534	658	16,578
New Jersey	3,380	2,577	11,837	3,358	5,483	865	549	28,049
New Mexico	5,785	1,445	5,006	1,220	1,178	48,662	45,353	108,648
New York	7,580	4,442	37,074	5,432	13,467	1,601	13,647	83,242
North Carolina	12,185	11,775	36,080	4,746	3,172	9,870	11,162	88,990
North Dakota	5,338	569	2,807	2,293	1,735	934	38,263	51,940
Ohio	19,844	12,251	22,428	5,908	8,425	316	28,587	97,759
Oklahoma	7,412	5,669	45,423	2,165	1,856	6,644	44,243	113,412
Oregon	1,653	8,161	47,545	2,517	1,917	65,350	8,738	135,881
Pennsylvania	21,187	13,237	29,061	4,839	8,838	454	13,344	90,961
Rhode Island	598	256	1,035	281	758	14	182	3,124
South Carolina	11,831	4,477	16,869	2,372	1,548	9,163	9,162	55,421
South Dakota	768	2,145	3,959	1,445	1,128	7,062	29,215	45,722
Tennessee	6,637	21,495	19,126	3,129	3,034	3,934	11,900	69,254
Texas	37,320	34,923	47,953	13,048	6,101	21,578	143,814	304,737
Tribal	32	1,557		0	0			1,589
Utah	5,011	3,564	8,859	1,021	2,328	27,412	5,682	53,877
Vermont	0	337	4,882	325	1,250	696	1,528	9,018
Virginia	7,141	10,840	27,774	3,938	4,315	5,659	8,194	67,861
Washington	1,927	4,197	30,049	3,737	3,665	4,487	13,617	61,680
West Virginia	16,198	4,921	10,405	1,114	1,084	3,050	3,649	40,423
Wisconsin	6,376	7,430	24,646	3,639	8,423	994	11,870	63,379
Wyoming	7,406	10,207	2,620	896	967	15,686	28,723	66,505
Grand Total	**384,320**	**404,926**	**1,029,916**	**169,144**	**188,320**	**684,035**	**1,030,631**	**3,891,291**
*Non-US seca_c3 component not included. These emissions are 120,617 tons/yr.								

3.4 Development of Future Year Control Case Emissions for Air Quality Modeling

For the future year control case (policy case) air quality modeling, the emissions for *all* sectors were unchanged from the base case modeling except for those from EGUs. The IPM model was used to prepare the 2016 policy case (i.e., the proposed Toxics Rule) for EGU emissions as described in the IPM v.4.10 Documentation, available at http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html. As with the base case projections, photochemical modeling of the policy case is based on interim IPM v.4.10. The final IPM 4.10 includes all of the updates incorporated in the base case. In addition, the mercury removal from some new fabric filters is correctly accounted for in this update. The policy modeled in this final scenario reflects the emissions limits that EPA is proposing. This differs from interim policy case modeling, which was conducted before a comprehensive review of ICR data was able to inform the proposed emissions limits. Using limited ICR data available early in the rulemaking process,

EPA's preliminary policy case reflected lower HCL and mercury emissions standards than are being proposed today. The changes in EGU Hg, SO_2, and $PM_{2.5}$ emissions as a result of the interim policy case (utilized in the air quality modeling) for the lower 48 states are summarized in Table 3-12. State-specific summaries of EGU Hg, SO_2 and $PM_{2.5}$ for the sum of the lower 48 states are shown in Tables 3-13 through 3-15, respectively.

Table 3-12. Summary of Emissions Changes for the Proposed Toxics Rule in the Lower 48 States

Item	Pollutant		
2016 EGU Emissions	**HG**	**SO2**	**PM2.5**
Base Case EGU Emissions (tons)	28.70	3,577,698	384,320
Control EGU Emissions (tons)	6.84	1,220,379	291,044
Reductions to Base Case in Control Case (tons)	21.87	2,357,319	93,276
Percentage Reduction of Base EGU Emissions	76.2%	65.9%	24.3%
Total 2016 Manmade Emissions*			
Total Base Case Emissions (tons)	63.92	6,288,530	3,891,292
Total Control Case Emissions (tons)	42.05	3,931,211	3,798,016
Percentage Reduction of All Manmade Emissions	34.2%	37.5%	2.4%
* In this table, man-made emissions includes average fires. Non-US seca_c3 emissions are not included: 957,065 SO2; and 120,617 PM2.5			

Table 3-13. State Specific Changes in Annual EGU Hg for the Lower 48 States

State	2016 Base Case Hg (tons)	2016 Policy Case Hg (tons)	EGU Hg reduction (tons)	EGU Hg reduction (%)
Alabama	1.255	0.192	1.063	85%
Arizona	0.749	0.089	0.660	88%
Arkansas	0.725	0.066	0.658	91%
California	0.132	0.084	0.048	36%
Colorado	0.083	0.090	-0.007	-8%
Connecticut	0.007	0.006	0.000	7%
Delaware	0.009	0.021	-0.012	-134%
District of Columbia	0.000	0.000	0.000	
Florida	0.486	0.193	0.293	60%

State	2016 Base Case Hg (tons)	2016 Policy Case Hg (tons)	EGU Hg reduction (tons)	EGU Hg reduction (%)
Georgia	0.512	0.215	0.296	58%
Idaho	0.000	0.000	0.000	
Illinois	0.488	0.287	0.201	41%
Indiana	1.558	0.380	1.178	76%
Iowa	0.999	0.152	0.847	85%
Kansas	0.955	0.097	0.858	90%
Kentucky	0.828	0.313	0.514	62%
Louisiana	1.019	0.166	0.853	84%
Maine	0.013	0.000	0.013	100%
Maryland	0.114	0.116	-0.001	-1%
Massachusetts	0.009	0.010	-0.001	-11%
Michigan	1.501	0.174	1.327	88%
Minnesota	0.161	0.074	0.087	54%
Mississippi	0.405	0.053	0.352	87%
Missouri	1.949	0.242	1.706	88%
Montana	0.097	0.045	0.052	54%
Nebraska	0.423	0.084	0.338	80%
Nevada	0.087	0.056	0.031	36%
New Hampshire	0.011	0.011	0.000	0%
New Jersey	0.026	0.026	0.000	0%
New Mexico	0.296	0.087	0.209	71%
New York	0.051	0.043	0.008	17%
North Carolina	0.487	0.207	0.280	57%
North Dakota	0.936	0.063	0.874	93%
Ohio	1.576	0.640	0.936	59%
Oklahoma	1.028	0.105	0.924	90%
Oregon	0.008	0.008	0.000	0%
Pennsylvania	1.613	0.517	1.096	68%
Rhode Island	0.000	0.000	0.000	
South Carolina	0.347	0.142	0.205	59%
South Dakota	0.027	0.012	0.015	56%
Tennessee	0.743	0.153	0.590	79%
Texas	3.367	0.536	2.831	84%
Utah	0.184	0.078	0.105	57%
Vermont	0.000	0.000	0.000	
Virginia	0.284	0.114	0.170	60%
Washington	0.167	0.020	0.147	88%
West Virginia	0.860	0.505	0.355	41%
Wisconsin	0.870	0.146	0.724	83%
Wyoming	1.260	0.220	1.040	83%
Total	**28.7**	**6.8**	**21.9**	**76%**

Table 3-14. State Specific Changes in Annual EGU SO_2 for the Lower 48 States

State	2016 Base Case SO2 (tons)	2016 Policy Case SO2 (tons)	EGU SO2 reduction (tons)	EGU SO2 reduction (%)
Alabama	172,198	38,346	133,852	78%
Arizona	23,140	21,632	1,508	7%
Arkansas	93,754	7,314	86,440	92%
California	4,740	4,148	592	12%
Colorado	55,588	19,698	35,890	65%
Connecticut	2,643	2,041	601	23%
Delaware	1,717	3,359	(1,642)	-96%
District of Columbia			-	
Florida	122,123	57,439	64,684	53%
Georgia	91,885	40,767	51,118	56%
Idaho	0	0	-	
Illinois	148,934	47,403	101,531	68%
Indiana	229,248	111,741	117,507	51%
Iowa	98,518	22,208	76,309	77%
Kansas	61,622	12,781	48,841	79%
Kentucky	123,010	97,707	25,304	21%
Louisiana	98,808	32,624	66,184	67%
Maine	1,123	0	1,123	100%
Maryland	36,211	11,528	24,683	68%
Massachusetts	4,236	2,556	1,680	40%
Michigan	169,853	27,922	141,931	84%
Minnesota	51,952	27,805	24,147	46%
Mississippi	55,317	10,595	44,722	81%
Missouri	172,031	32,412	139,619	81%
Montana	13,234	9,071	4,163	31%
Nebraska	74,642	34,551	40,091	54%
Nevada	11,283	4,735	6,548	58%
New Hampshire	4,348	730	3,618	83%
New Jersey	8,507	6,997	1,511	18%
New Mexico	11,370	9,357	2,013	18%
New York	28,911	13,468	15,443	53%
North Carolina	82,544	34,946	47,598	58%
North Dakota	76,081	11,955	64,126	84%
Ohio	204,291	77,852	126,439	62%
Oklahoma	139,800	14,196	125,605	90%
Oregon	11,102	1,423	9,679	87%
Pennsylvania	152,929	73,714	79,215	52%
Rhode Island	0	0	-	
South Carolina	128,070	35,223	92,847	72%
South Dakota	29,711	7,490	22,220	75%
Tennessee	106,762	44,110	62,652	59%
Texas	334,636	81,000	253,636	76%
Utah	31,343	14,261	17,083	55%

State	2016 Base Case SO2 (tons)	2016 Policy Case SO2 (tons)	EGU SO2 reduction (tons)	EGU SO2 reduction (%)
Vermont	0	0	-	
Virginia	45,345	16,029	29,317	65%
Washington	2,804	2,804		0%
West Virginia	127,826	44,129	83,696	65%
Wisconsin	77,871	24,481	53,390	69%
Wyoming	55,636	25,831	29,805	54%
Total	**3,577,698**	**1,220,379**	**2,357,319**	**66%**

Table 3-15. State Specific Changes in Annual EGU $PM_{2.5}$ for the Lower 48 States

State	2016 Base Case PM2.5 (tons)	2016 Policy Case PM2.5 (tons)	EGU PM2.5 reduction (tons)	EGU PM2.5 reduction (%)
Alabama	14,801	9,829	4,972	34%
Arizona	10,196	7,260	2,936	29%
Arkansas	3,805	2,803	1,002	26%
California	9,718	9,550	169	2%
Colorado	4,972	4,778	194	4%
Connecticut	1,632	1,537	95	6%
Delaware	643	815	-171	-27%
District of Columbia				
Florida	26,114	20,494	5,620	22%
Georgia	14,411	10,648	3,762	26%
Idaho	187	187	0	0%
Illinois	11,157	9,235	1,921	17%
Indiana	21,198	14,992	6,206	29%
Iowa	5,223	4,148	1,075	21%
Kansas	4,634	2,755	1,879	41%
Kentucky	13,598	9,009	4,589	34%
Louisiana	5,219	5,345	-125	-2%
Maine	712	699	13	2%
Maryland	3,791	3,069	723	19%
Massachusetts	2,754	2,452	302	11%
Michigan	7,188	5,170	2,019	28%
Minnesota	9,011	4,440	4,571	51%
Mississippi	2,554	2,583	-29	-1%
Missouri	8,040	5,719	2,321	29%
Montana	2,453	1,803	651	27%

State	2016 Base Case PM2.5 (tons)	2016 Policy Case PM2.5 (tons)	EGU PM2.5 reduction (tons)	EGU PM2.5 reduction (%)
Nebraska	2,657	4,024	-1,368	-51%
Nevada	10,903	10,816	87	1%
New Hampshire	1,138	917	220	19%
New Jersey	3,380	3,210	170	5%
New Mexico	5,785	5,287	498	9%
New York	7,580	6,719	861	11%
North Carolina	12,185	7,651	4,534	37%
North Dakota	5,337	1,787	3,551	67%
Ohio	19,844	13,671	6,173	31%
Oklahoma	7,412	5,973	1,439	19%
Oregon	1,653	1,548	106	6%
Pennsylvania	21,187	13,119	8,068	38%
Rhode Island	598	609	-11	-2%
South Carolina	11,831	7,085	4,746	40%
South Dakota	768	567	201	26%
Tennessee	6,637	4,758	1,879	28%
Texas	37,320	32,181	5,139	14%
Utah	5,011	4,399	611	12%
Vermont	0	0	0	-98%
Virginia	7,141	6,391	750	11%
Washington	1,927	1,650	278	14%
West Virginia	16,198	9,386	6,812	42%
Wisconsin	6,376	4,653	1,724	27%
Wyoming	7,406	5,292	2,114	29%
Tribal Data	32	32	0	0%
Total	**384,319**	**291,044**	**93,275**	**24%**

APPENDIX A.
MERCURY SPECIATION FRACTIONS USED TO SPECIATE THE MERCURY EMISSIONS

Category	Particulate	Divalent Gaseous	Elemental
Bituminous Coal and Pet. Coke, PC Boiler with ESP-CS	0.0117	0.4656	0.5227
Bituminous Coal, Coal Gasification	0.0051	0.0847	0.9102
Bituminous Coal, PC Boiler with Dry Sorbent Injection and ESP-CS	0.0016	0.6710	0.3274
Bituminous Coal, PC Boiler with ESP-CS	0.0611	0.6820	0.2570
Bituminous Coal, PC Boiler with ESP-CS and Wet FGD	0.0022	0.0778	0.9200
Bituminous Coal, PC Boiler with ESP-HS	0.0490	0.5784	0.3726
Bituminous Coal, PC Boiler with ESP-HS and Wet FGD	0.0063	0.2068	0.7870
Bituminous Coal, PC Boiler with FF Baghouse	0.0398	0.6258	0.3344
Bituminous Coal, PC Boiler with FF Baghouse and Wet FGD	0.0648	0.3300	0.6052
Bituminous Coal, PC Boiler with PM Scrubber	0.0180	0.1951	0.7869
Bituminous Coal, PC Boiler with SCR and SDA/FF Baghouse	0.0506	0.4604	0.4890
Bituminous Coal, PC Boiler with SDA/FF Baghouse	0.0917	0.2886	0.6197
Bituminous Coal, PC Boiler with SNCR and ESP-CS	0.2032	0.2712	0.5256
Bituminous Coal, Stoker Boiler with SDA/FF Baghouse	0.1996	0.1794	0.6211
Bituminous Coal/Pet. Coke, Cyclone with ESP-CS and Wet FGD	0.0007	0.1130	0.8863
Bituminous Coal/Pet. Coke, PC Boiler with FF Baghouse	0.0220	0.7841	0.1939
Bituminous Coal/Pet. Coke, Fluidized Bed Combustor with SNCR and FF Baghouse	0.4244	0.2787	0.2970
Bituminous Waste, Fluidized Bed Combustor with FF Baghouse	0.0212	0.3881	0.5907
Lignite Coal, Cyclone Boiler with ESP-CS	0.0004	0.1699	0.8297
Lignite Coal, Cyclone Boiler with SDA/FF Baghouse	0.0995	0.1707	0.7298
Lignite Coal, Fluidized Bed Combustor with ESP-CS	0.0137	0.1164	0.8700
Lignite Coal, Fluidized Bed Combustor with FF Baghouse	0.0042	0.7118	0.2840
Lignite Coal, PC Boiler with ESP-CS	0.0009	0.0362	0.9629
Lignite Coal, PC Boiler with ESP-CS and FF Baghouse	0.0019	0.6449	0.3532
Lignite Coal, PC Boiler with ESP-CS and Wet FGD	0.0082	0.1345	0.8574
Lignite Coal, PC Boiler with PM Scrubber	0.0016	0.0298	0.9686
Lignite Coal, PC Boiler with SDA/FF Baghouse	0.0036	0.1262	0.8702
Subbituminous Coal, Fluidized Bed Combustor with SNCR and FF Baghouse	0.0027	0.0342	0.9632
Subbituminous Coal, PC Boiler with ESP-CS	0.0016	0.3083	0.6901
Subbituminous Coal, PC Boiler with ESP-CS and Wet FGD	0.0043	0.0294	0.9663

Category	Particulate	Divalent Gaseous	Elemental
Subbituminous Coal, PC Boiler with ESP-HS	0.0006	0.1252	0.8741
Subbituminous Coal, PC Boiler with ESP-HS and Wet FGD	0.0117	0.0446	0.9437
Subbituminous Coal, PC Boiler with FF Baghouse	0.0149	0.8283	0.1568
Subbituminous Coal, PC Boiler with PM Scrubber	0.0145	0.0511	0.9344
Subbituminous Coal, PC Boiler with SDA/ESP	0.0032	0.0382	0.9586
Subbituminous Coal, PC Boiler with SDA/FF Baghouse	0.0099	0.0435	0.9467
Subbituminous Coal/Pet. Coke, Cyclone Boiler with ESP-HS	0.0093	0.0752	0.9155

Chapter 4
AIR QUALITY BENEFITS OF EMISSIONS REDUCTIONS

4.1 Air Quality Modeling Platform

This section describes the air quality modeling performed by EPA in support of the Toxics Rule. A national scale air quality modeling analysis was performed to estimate the impact of the sector emissions changes on future year annual and 24-hour PM2.5 concentrations, 8-hr maximum ozone, total mercury deposition, as well as visibility impairment. Air quality benefits are estimated with the Community Multi-scale Air Quality (CMAQ) model. CMAQ simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone, particulate matter and air toxics. In addition to the CMAQ 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 may be slightly different than the final utility sector inventory. Similarly, the projected future year inventory used for this analysis is generally representative of several years around 2016 such as 2015. 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. Photochemical grid models use state of the science numerical algorithms to estimate pollutant formation, transport, and deposition over a variety of spatial scales that range from urban to continental. Emissions of precursor species are injected into the model where they react to form secondary species such as ozone and then transport around the modeling domain before ultimately being removed by deposition or chemical reaction.

The 2005-based CMAQ modeling platform was used as the basis for the air quality modeling for this 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. More information about the modeling platform is available in the modeling technical support document for this rule (USEPA, 2011).

4.1.1 Photochemical Model Background

The Community Multi-scale Air Quality (CMAQ) model v4.7.1 (www.cmaq-model.org) is a state of the science three-dimensional Eularian "one-atmosphere" photochemical transport

model used to estimate air quality (Appel et al., 2008; Appel et al., 2007; Byun and Schere, 2006). CMAQ simulates the formation and fate of photochemical oxidants, ozone, primary and secondary PM concentrations, and air toxics over regional and urban spatial scales for given input sets of meteorological conditions and emissions. CMAQ is applied with the AERO5 aerosol module, which includes the ISORROPIA inorganic chemistry (Nenes et al., 1998) and a secondary organic aerosol module (Carlton et al., 2010). The CMAQ model is applied with sulfur and organic oxidation aqueous phase chemistry (Carlton et al., 2008) and the carbon-bond 2005 (CB05) gas-phase chemistry module (Gery et al., 1989).

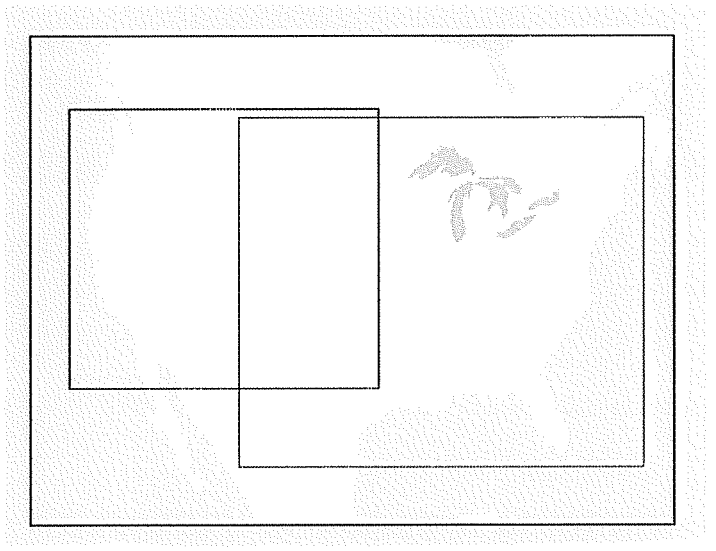
4.1.2 Model Setup, Application, and Post-Processing

The modeling analyses were performed for a domain covering the continental United States, as shown in Figure 4.1. This domain has a parent horizontal grid of 36 km with two finer-scale 12 km grids over portions of the eastern and western U.S. 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 vary in time and space. 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 emissions changes. Table 4.1 provides geographic information about the photochemical model domains.

Table 4-1. 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 (see Table II-3)		

Figure 4-1. Map of the Photochemical Modeling Domains. 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.



The 36 km and 12 km modeling domains were modeled for the entire year of 2005 and projected year 2016. Data from the entire year were utilized when looking at the estimation of PM2.5, total mercury deposition, and visibility impacts from the regulation. Data from April through October is used to estimate ozone impacts. All air quality impacts are based on improvements in future year pollution based on emissions changes from this source sector.

As part of the analysis for this rulemaking, the modeling system was used to calculate daily and annual PM2.5 concentrations, 8-hr maximum ozone, annual total mercury deposition levels and visibility impairment. Model predictions are used to estimate future-year design values of PM2.5 and ozone. Specifically, we compare a 2016 baseline scenario, a scenario without the boiler sector controls, to a 2016 control scenario which includes the adjustments to the boiler 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 (USEPA, 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 mercury deposition were analyzed using absolute model changes, although percent changes between the control case and two future baselines are also estimated.

4.1.3 Emissions Input Data

The emissions data used in the base year and future baseline and future emissions adjustment case are based on the 2005 v4.1 platform. The emissions cases use different emissions data for some pollutants 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 mercury emissions from the National Air Toxics Assessment Inventory and some industrial boiler sector mercury emissions more consistent with the engineering analysis for the Industrial/Commercial/Institutional Boilers and Process Heaters NESHAP. Emissions for the future years for the EGU sector utilized information collected from the utility MACT information collection request. Emissions are processed to photochemical model inputs with the SMOKE emissions modeling system (Houyoux et al., 2000).

The 2016 baseline (or reference) case is intended to represent the emissions associated with growth and controls in that year projected from the 2005 simulation year. The United States EGU point source emissions estimates for the future year baseline and control case are based on an Integrated Planning Model (IPM) run for criteria pollutants, hydrochloric acid, and mercury in 2016. Both control and growth factors were applied to a subset of the 2005 non-EGU point and non-point to create the 2016 baseline case. The 2005 v4 platform 2014 projection factors were the starting point for most of the 2016 SMOKE-based projections. The mercury projections for non–EGU point sources accounted for emission reductions expected in the future due to NESHAP for various non-EGU source categories that were finalized or expected to be finalized prior to the Utility proposal including the Boiler MACT, Gold Mine NESHAP and Electric Arc Furnace NESHAP. The estimated total anthropogenic emissions and emissions for the utility sector used in the modeling assessment are shown in Table 4-2. More details on these emissions can be found in Chapter 3.

Table 4-2. Estimated Total Inventory and EGU Sector Emissions for Each Modeling Scenario

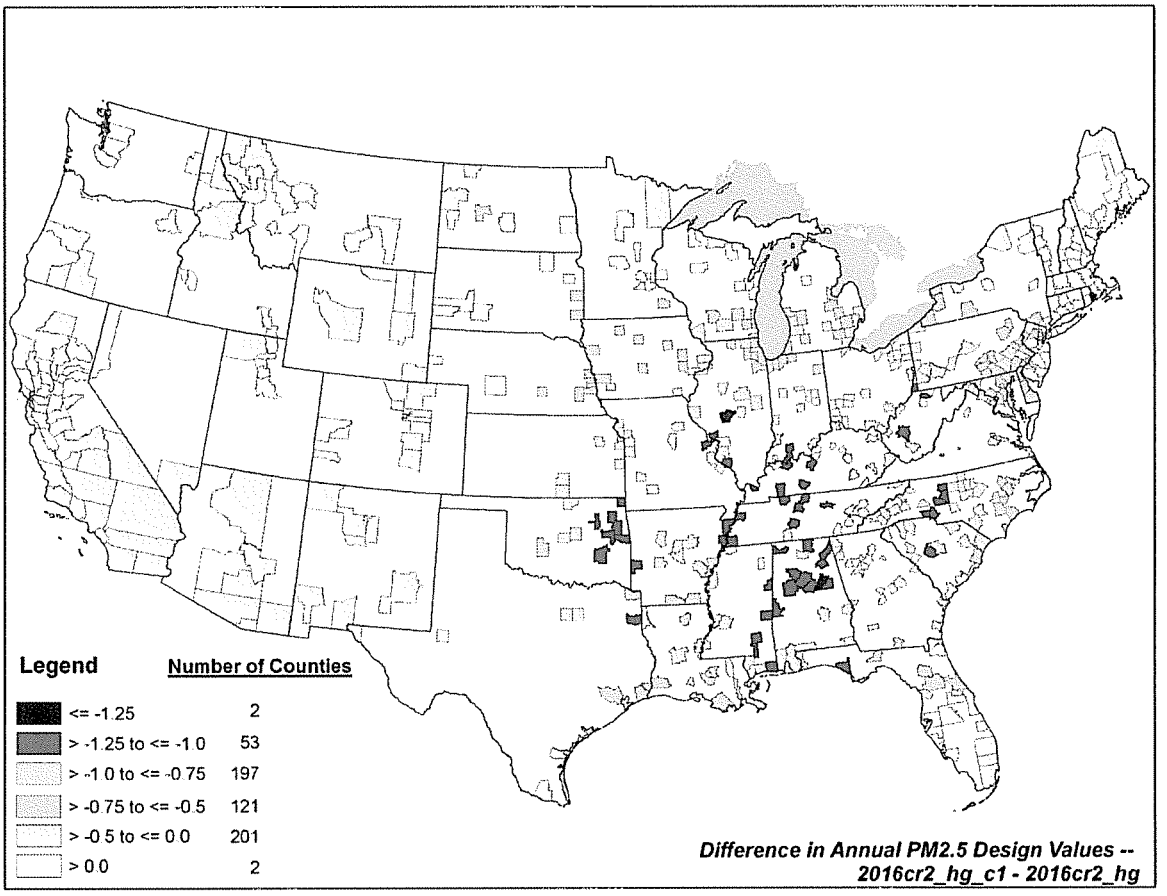
		Emissions (tons/year)					
Scenario	Sector	VOC	NOx	CO	SO2	PM10	PM2.5
2005 baseline	EGU (PTIPM)	40,950	3,726,459	601,564	10,380,786	615,095	508,903
	All	17,613,543	22,216,093	83,017,436	15,050,209	13,031,716	4,400,680
2016 baseline	EGU (PTIPM)	40,845	1,769,764	691,310	3,577,698	523,504	384,320
	All	14,390,421	15,019,836	59,148,384	7,245,595	12,772,091	4,022,846
2016 control case	EGU (PTIPM)	38,217	1,618,199	656,245	1,220,379	358,165	291,044
	All	14,387,792	14,868,270	59,113,319	4,888,276	12,606,752	3,929,570

		Emissions (tons/year)					
Scenario	Sector	HG2	HG0	HG_PM25	HCL	CL2	NH3
2005 baseline	EGU (PTIPM)	21	30	1.6	351,592	99	21,684
	All	33	64	8.5	429,223	6,409	3,762,641
2016 baseline	EGU (PTIPM)	7	21	0.7	74,089		36,655
	All	16	42	5.9	140,638	6,050	3,897,033
2016 control case	EGU (PTIPM)	2	5	0.4	8,802		36,982
	All	11	26	5.6	75,351	6,050	3,897,360

4.2 Impacts of Sector on Future Annual $PM_{2.5}$ Levels

This section summarizes the results of our modeling of annual average PM2.5 air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2016 baseline scenario to a 2016 control scenario (the proposed Toxics Rule interim values). The modeling assessment indicates a decrease up to 1.49 $\mu g/m^3$ in annual PM2.5 design values is possible given an area's proximity to controlled sources. The median reduction in annual PM2.5 design value over all monitor locations is 0.70 $\mu g/m^3$.

Figure 4-2. Change in Design Values between the 2016 Baseline and 2016 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline.



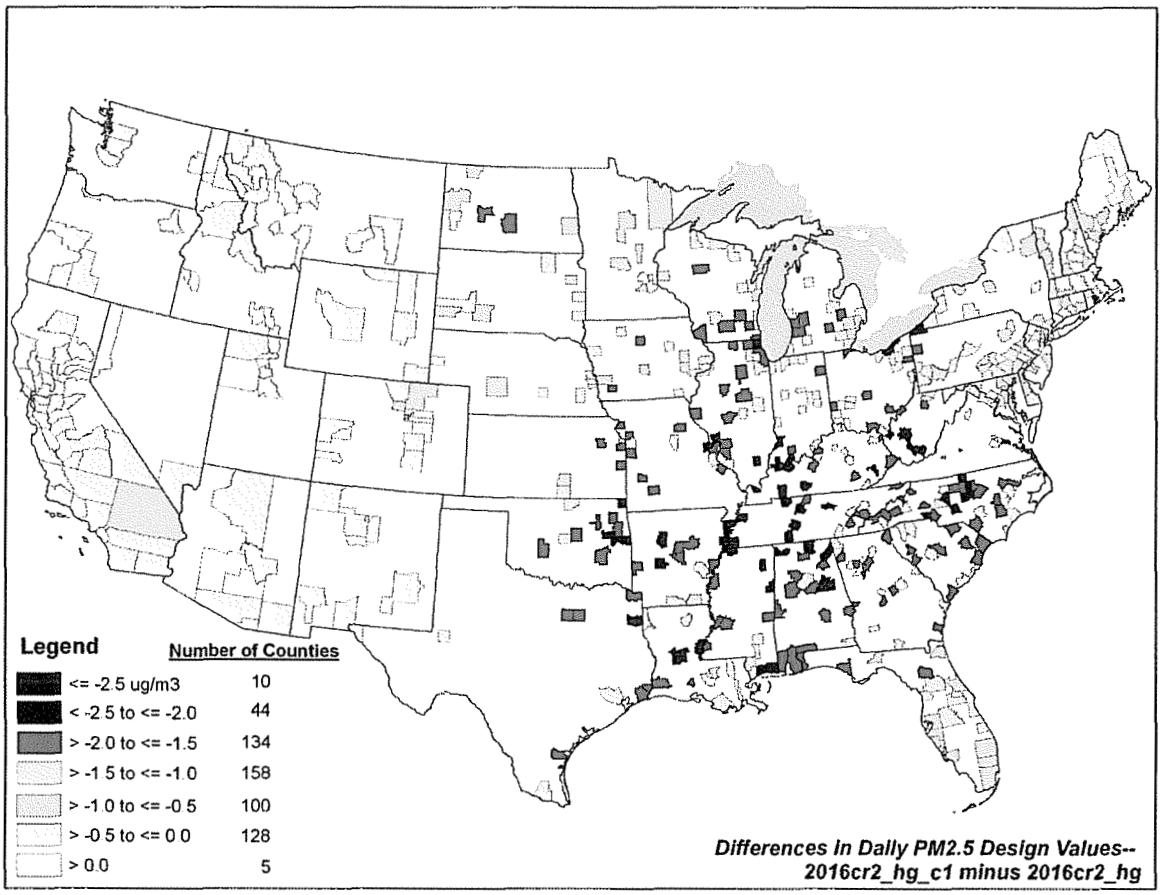
An annual $PM_{2.5}$ design value is the concentration that determines whether a monitoring site meets the annual NAAQS for $PM_{2.5}$. The full details involved in calculating an annual $PM_{2.5}$ design value are given in appendix N of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by United States Environmental Protection Agency modeling guidance (USEPA, 2007).

4.3 Impacts of Sector on Future 24-hour $PM_{2.5}$ Levels

This section summarizes the results of our modeling of 24-hr average $PM_{2.5}$ air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2016 baseline scenario to a 2016 control scenario (the interim results for the proposed Toxics Rule). A decrease up to 3.1 $\mu g/m^3$ in 24-hr average $PM_{2.5}$ design value at monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions from those sources. A median decrease of 1.2 $\mu g/m^3$ in 24-hr average $PM_{2.5}$

design value at monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions from those sources.

Figure 4-3. Change in Design Values between the 2016 Base Case and 2016 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline.



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 United States Environmental Protection Agency modeling guidance (USEPA, 2007).

4.4 Impacts of Sector on Future Visibility Levels

Air quality modeling conducted for this rule was used to project visibility conditions in 138 mandatory Class I federal areas across the U.S. in 2016 (USEPA, 2007). The level of visibility impairment in an area is based on the light-extinction coefficient and a unitless

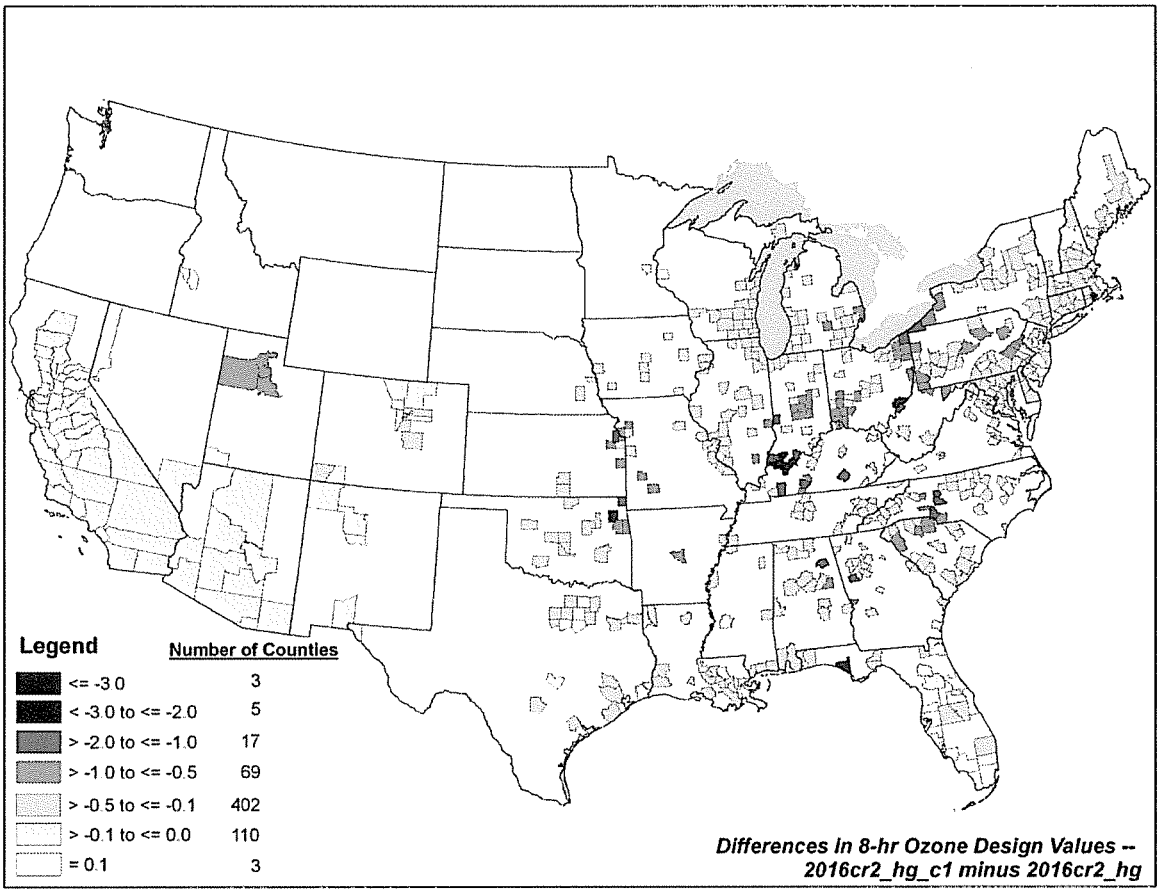
visibility index, called a "deciview", which is used in the valuation of visibility. The deciview metric provides a scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. Higher deciview values are indicative of worse visibility. Thus, an improvement in visibility is a decrease in deciview value.

The modeling assessment indicates a median visibility improvement of 0.06 deciviews in annual 20% worst visibility days over all Class I area monitors. An improvement in visibility up to 2.68 deciviews on the 20% worst visibility days at Class I monitor locations in the United States is possible given an area's proximity to controlled sources and the amount of reduced emissions from these sources.

4.5 Impacts of Sector on Future Ozone Levels

This section summarizes the results of our modeling of 8-hr maximum ozone air quality impacts in the future due to reductions in emissions from this sector. Specifically, we compare a 2016 baseline scenario to a 2016 control scenario. The modeling assessment indicates a decrease of up to 3.5 ppb in 8-hr averaged ozone design value is possible given an area's proximity to controlled sources and the amount of reduced emissions from these sources. A median decrease of 0.20 ppb in 8-hr averaged ozone design value is possible given an area's proximity to controlled sources and the amount of reduced emissions from these sources. The full details involved in calculating design value are given in appendix P of 40 CFR part 50. Projected air quality benefits are estimated using procedures outlined by United States Environmental Protection Agency modeling guidance (USEPA, 2007).

Figure 4-4. Change in Design Values between the 2016 Baseline and 2016 Control Simulations. Negative numbers indicate lower (improved) design values in the control case compared to the baseline.



4.6 Impacts of Sector on Total Mercury Deposition

This section summarizes the results of our modeling of total mercury deposition impacts in the future based on changes to source sector emissions. Available data indicate that the mercury emissions from these sources in the 2016 baseline scenario are a mixture of gaseous elemental mercury (73%), inorganic divalent mercury (reactive gas phase mercury) (24%), and particulate bound mercury (2%). Model results for the continental United States indicate that total mercury deposition (wet and dry forms) reductions from this sector would be 24,000 $\mu g/m^2$ (1.0% of total mercury deposition from all sources).

Figure 4-5. Difference in Total Mercury Deposition between 2016 Base Case and 2016 Control Scenarios

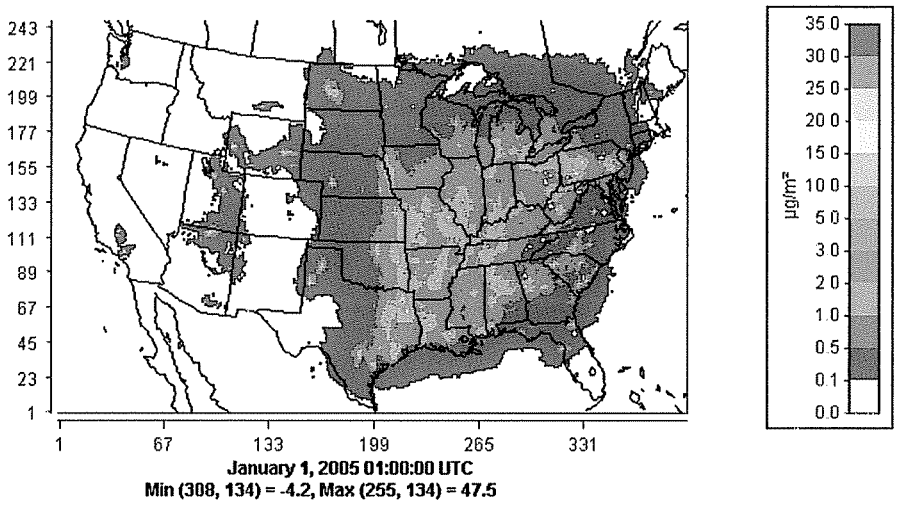
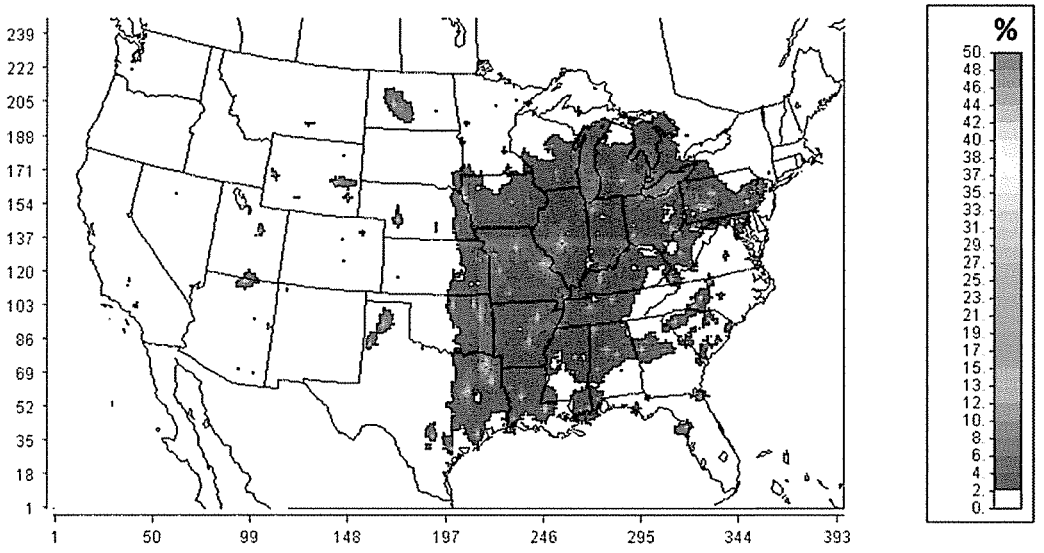


Figure 4-6. Percent Difference in Total Mercury Deposition between 2016 Base Case and 2016 Control Scenarios



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Chapter 5
MERCURY AND OTHER HAP BENEFITS ANALYSIS

5.1 Introduction

This chapter provides an analysis of the benefits of the proposed Toxics Rule from mercury and reductions of other HAP. This analysis builds on the methodologies developed previously for the 2005 Clean Air Mercury Rule (CAMR). This is a national scale assessment which focuses on the exposures to methylmercury in populations who consume self-caught freshwater fish (recreational fishers and their families). While there are other routes of exposure, including self-caught saltwater fish and commercially purchased fresh and saltwater fish, these exposures are not evaluated because 1) for self-caught saltwater fish, we are unable to estimate the reduction in fish tissue methylmercury that would be associated with reductions in mercury deposition from U.S. EGUs, and 2) for commercially purchased ocean fish, it is nearly impossible to determine the source of the methylmercury in those fish, and thus we could not attribute mercury levels to U.S. EGUs. This benefits analysis focuses on reductions in lost IQ points in the population, because of the discrete nature of the effect, and because we are able to assign an economic value to IQ points. There are other neurological effects associated with exposures to methylmercury, including impacts on motor skills and attention/behavior and therefore, risk estimates based on IQ will not cover these additional endpoints and therefore could further underestimate overall neurodevelopmental impacts. In addition, the NRC (2001) noted that "there remains some uncertainty about the possibility of other health effects at low levels of exposure. In particular, there are indications of immune and cardiovascular effects, as well as neurological effects emerging later in life, that have not been adequately studied." These limitations suggest that the benefits of mercury reductions are understated by our analysis, however, the magnitude of the additional benefits is highly uncertain

In Section 5.2, we discuss the potential health effects of mercury. Section 5.3 provides a discussion of mercury in the environment, including potential impacts on wildlife. Section 5.4 describes the resulting change in mercury deposition from air quality modeling of the proposed Toxics rule. Section 5.5 presents information on key data and assumptions used in conducting the benefits analysis. Section 5.6 presents information on a dose-response function that relates mercury consumption in women of childbearing with changes in IQ seen in children that were exposed prenatally. IQ is used as a surrogate for the neurobehavioral endpoints that EPA relied upon for setting the methylmercury reference dose (RfD). Section 5.7 presents exposure modeling and benefit methodologies applied to a no-threshold model (i.e., a model that assumes no threshold in effects at low doses of mercury exposure). Section 5.8 presents the final benefits

and risk estimates for recreational freshwater anglers and selected high-risk subpopulations. Section 5.9 presents a qualitative description of the benefits from reductions in HAPs other than mercury that will take place as a result of the proposed Toxics Rule.

For this benefits assessment, EPA chose to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal mercury exposure as the initial endpoint for quantification and valuation of mercury health benefits. Reasons for this initial focus on IQ included the availability of thoroughly-reviewed, high-quality epidemiological studies assessing IQ or related cognitive outcomes suitable for IQ estimation, and the availability of well-established methods and data for economic valuation of avoided IQ deficits, as applied in EPA's previous benefits analyses for childhood lead exposure.

The quantitative estimates of human health benefits and risk levels provided in Section 5.2 consist of two primary sets of analysis: 1) A national-scale assessment of economic benefits associated with avoided IQ loss due to reduced methylmercury (MeHg) exposure among recreational freshwater anglers; and 2) Modeled risk levels, in terms of IQ loss, for six high-risk subpopulations as a means of estimating potential disproportionate impacts on demographic groups with traditionally subsistence or near-subsistence rates of fish consumption.

The first analysis (Section 5.2.1) estimates benefits from avoided IQ loss under various regulatory scenarios for all recreational freshwater anglers in the 48 contiguous U.S. states. The average effect on individual avoided IQ loss in 2016 is 0.00209 IQ points, with total nationwide benefits estimated between $0.5 and $6.1 million.[1] In contrast, the subpopulations analyses (Section 5.2.2) focus on specific demographic groups with relatively high levels of fish consumption. For example, an African-American child in the Southeast born in 2016 to a mother consuming fish at the 90th percentile of published subsistence-like levels is estimated to experience a loss of 7.711 IQ points as a result of in-utero MeHg exposure from all sources in the absence of a Toxics Rule.[2] The implementation of the Toxics Rule would reduce the expected IQ loss for this child by an estimated 0.176 IQ points.

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[1] Monetized benefits estimates are for an immediate change in MeHg levels in fish. If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response.

[2] We do note that overall confidence in IQ loss estimates above approximately 7 points decreases because we begin to apply the underlying IQ loss function at exposure levels (ppm hair levels) above those reflected in epidemiological studies used to derive those functions. The 39.1 ppm was the highest measured ppm level in the Faroes Island study, while ~86 was the highest value in the New Zealand study (USEPA, 2005) (a 7 IQ points

5.2 Impact of Mercury on Human Health

5.2.1 Introduction

Mercury is a persistent, bioaccumulative toxic metal that is emitted from power plants in three forms: gaseous elemental Hg (Hg^0), oxidized Hg compounds (Hg^{+2}), and particle-bound Hg (Hg_P). Elemental Hg does not quickly deposit or chemically react in the atmosphere, resulting in residence times that are long enough to contribute to global scale deposition. Oxidized Hg and HgP deposit quickly from the atmosphere impacting local and regional areas in proximity to sources. Methylmercury (MeHg) is formed by microbial action in the top layers of sediment and soils, after Hg has precipitated from the air and deposited into waterbodies or land. Once formed, MeHg is taken up by aquatic organisms and bioaccumulates up the aquatic food web. Larger predatory fish may have MeHg concentrations many times, typically on the order of one million times, that of the concentrations in the freshwater body in which they live. Although Hg is toxic to humans when it is inhaled or ingested, we focus in this rulemaking on exposure to MeHg through ingestion of fish, as it is the primary route for human exposures in the U.S., and potential health risks do not likely result from Hg inhalation exposures associated with Hg emissions from utilities.

In 2000, the National Research Council (NRC) of the NAS issued the NAS Study, which provides a thorough review of the effects of MeHg on human health. There are numerous studies that have been published more recently that report effects on neurologic and other endpoints.

5.2.2 Reference and Benchmark Doses

In 1995, EPA set a health-based ingestion rate for chronic oral exposure to MeHg termed an oral Reference Dose (RfD), at 0.0001 milligrams per kilogram per day (mg/kg-day).[1] The RfD was based on effects reported for children exposed in utero during the Iraqi Hg poisoning episode, in which children were exposed to high levels of Hg when their mothers consumed contaminated grain (Marsh et al., 1987). Subsequent research from large epidemiological studies in the Seychelles (Davidson et al., 1995), Faroe Islands (Grandjean et al., 1997), and New Zealand (Kjellstrom et al., 1989) added substantially to the body of knowledge on neurological effects from MeHg exposure. In 2001 EPA established a revised RfD based on the advice of the NAS and an independent review panel convened as part of the Integrated Risk Information

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loss is approximately associated with a 40 ppm hair level given the concentration-response function we are using).

[1] MeHg exposure is measured as milligrams of MeHg per kilogram of bodyweight per day, thus normalizing for the size of fish meals and the differences in bodyweight among exposed individuals.

System (IRIS) process. In their analysis, the NAS examined in detail the epidemiological data from the Seychelles, the Faroe Islands, and New Zealand, as well as other toxicological data on MeHg. The NAS recommended that neurobehavioral deficits as measured in several different tests among these studies be used as the basis for the RfD.

The NAS proposed that the Faroe Islands cohort was the most appropriate study for defining an RfD, and specifically selected children's performance on the Boston Naming Test (a neurobehavioral test) as the key endpoint. Results from all three studies were considered in defining the RfD, as published in the "2001 Water Quality for the Protection of Human Health: Methylmercury" and in the Integrated Risk Information System (IRIS) summary for MeHg: "Rather than choose a single measure for the RfD critical endpoint, EPA based this RfD for this assessment on several scores from the Faroes' measures, with supporting analyses from the New Zealand study, and the integrative analysis of all three studies." (USEPA, 2002).

EPA defined the updated RfD of 0.0001 mg/kg-day in 2001 (USEPA, 2002). Although derived from a more complete data set and with a somewhat different methodology, the current RfD is numerically the same as the previous (1995) RfD (0.0001 mg/kg-day, or 0.1 µg/kg-day).

This RfD, consistent with the standard definition, is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 2002). In general EPA believes that exposures at or below the RfD are unlikely to be associated with appreciable risk of deleterious effects. However, no RfD defines an exposure level corresponding to zero risk; moreover the RfD does not represent a bright line, above which individuals are at risk of adverse effects. EPA's interpretation for this assessments is that any exposures to MeHg above the RfD are of concern given the nature of the data available for mercury that is not necessarily available for many other chemicals, where exposures have often had to be significantly above the RfD before they might be considered as causing a hazard to public health. The scientific basis for the mercury RfD includes extensive human data and extensive data on sensitive subpopulations, including pregnant mothers; therefore, the RfD does not include extrapolations from animals to humans, and from the general population to sensitive subpopulations. In addition, there was no evidence of a threshold for MeHg-related neurotoxicity within the range of exposures in the Faroe Islands study which served as the primary basis for the RfD. This additional confidence in the basis for the RfD suggests that all exposures above the RfD can be interpreted with more confidence as causing a potential hazard to public health. Studies published since the current MeHg RfD was released include new analyses of children's neuropsychological effects from the existing Seychelles and Faroe Islands

cohorts, including formation of a new cohort in the Faroe Islands study. There are also a number of new studies that were conducted in population-based cohorts in the U.S. and other countries. A comprehensive assessment of the new literature has not been completed by EPA. However, data published since 2001 are generally consistent with those of the earlier studies that were the basis of the RfD, demonstrating persistent effects in the Faroe Island cohort, and in some cases associations of effects with lower MeHg exposure concentrations than in the Faroes. These new studies provide additional confidence that exposures above the RfD are contributing to risk of adverse effects, and that reductions in exposures above the RfD can lead to incremental reductions in risk.

5.2.3 Neurologic Effects

In its review of the literature, the NAS found neurodevelopmental effects to be the most sensitive and best documented endpoints and appropriate for establishing an RfD (NRC, 2000); in particular NAS supported the use of results from neurobehavioral or neuropsychological tests. The NAS report (NRC, 2000) noted that studies in animals reported sensory effects as well as effects on brain development and memory functions and support the conclusions based on epidemiology studies. The NAS noted that their recommended endpoints for an RfD are associated with the ability of children to learn and to succeed in school. They concluded the following: "The population at highest risk is the children of women who consumed large amounts of fish and seafood during pregnancy. The committee concludes that the risk to that population is likely to be sufficient to result in an increase in the number of children who have to struggle to keep up in school."

5.2.4 Cardiovascular Impacts

The NAS summarized data on cardiovascular effects available up to 2000. Based on these and other studies, the NRC (2000) concluded that "Although 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 MeHg toxicity in humans and animals." The NRC also stated that "additional studies are needed to better characterize the effect of methylmercury exposure on blood pressure and cardiovascular function at various stages of life."

Additional cardiovascular studies have been published since 2000. EPA did not to develop a quantitative dose-response assessment for cardiovascular effects associated with MeHg exposures, as there is no consensus among scientists on the dose-response functions for these effects. In addition, there is inconsistency among available studies as to the association between MeHg exposure and various cardiovascular system effects. The pharmacokinetics of

some of the exposure measures (such as toenail Hg levels) are not well understood. The studies have not yet received the review and scrutiny of the more well-established neurotoxicity data base.

5.2.5 Genotoxic Effects

The Mercury Study noted that MeHg is not a potent mutagen but is capable of causing chromosomal damage in a number of experimental systems. The NAS concluded that evidence that human exposure to MeHg caused genetic damage is inconclusive; they note that some earlier studies showing chromosomal damage in lymphocytes may not have controlled sufficiently for potential confounders. One study of adults living in the Tapajós River region in Brazil (Amorim et al., 2000) reported a direct relationship between MeHg concentration in hair and DNA damage in lymphocytes; as well as effects on chromosomes. Long-term MeHg exposures in this population were believed to occur through consumption of fish, suggesting that genotoxic effects (largely chromosomal aberrations) may result from dietary, chronic MeHg exposures similar to and above those seen in the Faroes and Seychelles populations.

5.2.6 Immunotoxic Effects

Although exposure to some forms of Hg can result in a decrease in immune activity or an autoimmune response (ATSDR, 1999), evidence for immunotoxic effects of MeHg is limited (NRC, 2000).

5.2.7 Other Human Toxicity Data

Based on limited human and animal data, MeHg is classified as a "possible" human carcinogen by the International Agency for Research on Cancer (IARC, 1994) and in IRIS (USEPA, 2002). The existing evidence supporting the possibility of carcinogenic effects in humans from low-dose chronic exposures is tenuous. Multiple human epidemiological studies have found no significant association between Hg exposure and overall cancer incidence, although a few studies have shown an association between Hg exposure and specific types of cancer incidence (e.g., acute leukemia and liver cancer) (NAS, 2000).

There is also some evidence of reproductive and renal toxicity in humans from MeHg exposure. However, overall, human data regarding reproductive, renal, and hematological toxicity from MeHg are very limited and are based on either studies of the two high-dose poisoning episodes in Iraq and Japan or animal data, rather than epidemiological studies of chronic exposures at the levels of interest in this analysis.

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5.3 Impact of Mercury on Ecosystems and Wildlife

5.3.1 Introduction

Deposition of mercury to waterbodies can also have an impact on ecosystems and wildlife. Mercury contamination is present in all environmental media with aquatic systems experiencing the greatest exposures due to bioaccumulation. Bioaccumulation refers to the net uptake of a contaminant from all possible pathways and includes the accumulation that may occur by direct exposure to contaminated media as well as uptake from food.

Atmospheric mercury enters freshwater ecosystems by direct deposition and through runoff from terrestrial watersheds. Once mercury deposits, it may be converted to organic methylmercury mediated primarily by sulfate-reducing bacteria. Methylation is enhanced in anaerobic and acidic environments, greatly increasing mercury toxicity and potential to bioaccumulate in aquatic foodwebs. A number of key biogeochemical controls influence the production of methylmercury in aquatic ecosystems. These include sulfur, pH, organic matter, iron, mercury "aging", and bacteria type and activity (Munthe et al., 2007).

Wet and dry deposition of oxidized mercury is a dominant pathway for bringing mercury to terrestrial surfaces. In forest ecosystems, elemental mercury may also be absorbed by plants stomatally, incorporated by foliar tissues and released in litterfall (Ericksen et al., 2003). Mercury in throughfall, direct deposition in precipitation, and uptake of dissolved mercury by roots (Rea et al., 2002) are also important in mercury accumulation in terrestrial ecosystems.

Soils have significant capacity to store large quantities of atmospherically deposited mercury where it can leach into groundwater and surface waters. The risk of mercury exposure extends to insectivorous terrestrial species such as songbirds, bats, spiders, and amphibians that receive mercury deposition or from aquatic systems near the forest areas they inhabit (Bergeron et al., 2010a, b; Cristol et al., 2008; Rimmer et al., 2005; Wada et al., 2009 & 2010).

Numerous studies have generated field data on the levels of mercury in a variety of wild species. Many of the data from these environmental studies are anecdotal in nature rather than representative or statistically designed studies. The body of work examining the effects of these

exposures is growing but still incomplete given the complexities of the natural world. A large portion of the adverse effect research conducted to date has been carried out in the laboratory setting rather than in the wild; thus, conclusions about overarching ecosystem health and population effects are difficult to make at this time. In the sections that follow numerous effects have been identified at differing exposure levels.

5.3.2 Effects on Fish

A review of the literature on effects of mercury on fish (Crump and Trudeau, 2009) reports results for numerous species including trout, bass (large and smallmouth), northern pike, carp, walleye, salmon and others from laboratory and field studies. The effects studied are reproductive and include deficits in sperm and egg formation, histopathological changes in testes and ovaries, and disruption of reproductive hormone synthesis. These studies were conducted in areas from New York to Washington and while many were conducted by adding MeHg to water or diet many were conducted at current environmental levels. While we cannot determine at this time whether these reproductive deficits are affecting fish populations across the United States it should be noted that it is possible that over time reproductive deficits could have an effect on populations. Lower fish populations would conceivably impact the ecosystem services like recreational fishing derived from having healthy aquatic ecosystems quite apart from the effects of consumption advisories due to the human health effects of mercury.

The Integrated Science Assessment for Oxides of Nitrogen and Sulfur – Ecological Criteria (Final Report, 2008) presents information regarding the possible complementary effects of sulfur and mercury deposition. The ISA has concluded that there is a causal relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments. This suggests that lowering the rate of sulfur deposition would also reduce mercury methylation thus alleviating the effects of aquatic acidification as well as the effects of mercury on fish.

5.3.3 Effects on Birds

In addition to effects on fish, mercury also affects avian species. In previous reports (EPA 1997 and CAMR 2005) much of the focus has been on large piscivorous species, in particular the common loon. The loon is most visible to the public during the summer breeding season on northern lakes and they have become an important symbol of wilderness in these areas (McIntyre and Barr 1997). A multitude of loon watch, preservation, and protection groups have formed over the past few decades and have been instrumental in promoting conservation, education, monitoring, and research of breeding loons (McIntyre and Evers 2000, Evers 2006). Significant

adverse effects on breeding loons from mercury have been found to occur, including behavioral (reduced nest-sitting), physiological (flight feather asymmetry), and reproductive (chicks fledged/territorial pair) effects (Evers, 2008, Burgess, 2008) and reduced survival (Mitro et al., 2008). Additionally Evers et al. (2008) report that they believe that results from their study integrating the effects on the endpoints listed above and evidence from other studies the weight of evidence indicates that population-level effects negatively impacting population viability occur in parts of Maine and New Hampshire, and potentially in broad areas of the loon's range.

Recently attention has turned to other piscivorous species such as the white ibis and great snowy egret. While considered to be fish-eating generally these wading birds have a diverse diet including crayfish, crabs, snails, insects and frogs. These species are experiencing a range of adverse effects due to exposure to mercury. The white ibis has been observed to have decreased foraging efficiency (Adams and Frederick, 2008). Additionally ibises have been shown to exhibit decreased reproductive success and altered pair behavior at chronic exposure to levels of dietary MeHg commonly encountered by wild birds (Frederick and Jayasena, 2010). These effects include significantly more unproductive nests, male/male pairing, reduced courtship behavior (head bobbing and pair bowing) and lower nestling production by exposed males. In this study a worst-case scenario suggested by the results could involve up to a 50% reduction in fledglings due to MeHg in diet. These estimates may be conservative if male/male pairing in the wild resulted in a shortage of partners for females and the effect of homosexual breeding were magnified. In egrets mercury has been implicated in the decline of the species in south Florida (Sepulveda et al., 1999) and Hoffman (2010) has shown that egrets experience liver and possibly kidney effects. While ibises and egrets are most abundant in coastal areas and these studies were conducted in south Florida and Nevada, the ranges of ibises and egrets extend to a large portion of the United States. Ibis territory can range inland to Oklahoma, Arkansas and Tennessee. Egret range covers virtually the entire United States except the mountain west. Insectivorous birds have also been shown to suffer adverse effects due to current levels of mercury exposure. These songbirds such as Bicknell's thrush, tree swallows and the great tit have shown reduced reproduction, survival, and changes in singing behavior. Exposed tree swallows produced fewer fledglings (Brasso, 2008), lower survival (Hallinger, 2010) and had compromised immune competence (Hawley, 2009). The great tit has exhibited reduced singing behavior and smaller song repertoire in an area of high contamination in the vicinity of a metallurgic smelter in Flanders (Gorissen, 2005). While these effects were small and would likely have little effect on population viability in such a short-lived species.

5.3.4 Effects on Mammals

In mammals adverse effects have been observed in mink and river otter collected in the wild in the northeast where atmospheric deposition from municipal waste incinerators and electric utilities are the largest sources (USEPA, 1999), both fish eating species. For otter from Maine and Vermont maximum concentrations on Hg in fur nearly equal or exceed a concentration associated with mortality. Concentrations in liver for mink in Massachusetts/Connecticut and the levels in fur from mink in Maine exceed concentrations associated with acute mortality (Yates, 2005). Adverse sub-lethal effects may be associated with lower Hg concentrations and consequently be more widespread than potential acute effects. These effects may include increased activity, poorer maze performance, abnormal startle reflex, and impaired escape and avoidance behavior (Scheuhammer et al., 2007). Conclusions

The studies cited here provide a glimpse of the scope of mercury effects on wildlife particularly reproductive and survival effects at current exposure levels. These effects range across species from fish to mammals and spatially across a wide area of the United States. The literature is far from complete however. Much more research is required to establish a link between the ecological effects on wildlife and the effect on ecosystem services (services that the environment provides to people) for example recreational fishing, bird watching and wildlife viewing. EPA is not, however, currently able to quantify or monetize the benefits of reducing mercury exposures affecting provision of ecosystem services.

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5.4 Mercury Risk and Exposure Analyses – Data Inputs and Assumptions

5.4.1 Introduction

This section provides information regarding key data inputs and assumptions used in this assessment. The section begins with a description of the populations modeled in this assessment, follows with information about the data used to estimate MeHg concentrations in fish, and closes with a summary of the science and related assumptions used in this assessment to link changes in modeled mercury deposition to changes in fish tissue concentrations.

5.4.2 Data Inputs

Populations Assessed For the National Aggregate Estimates of Exposed Populations in Freshwater Fishing Households

The main source of data for identifying the size and location of the potentially exposed populations is the Census 2000 data, summarized at the tract-level. There are roughly 64,500 tracts in the continental United States, with populations generally ranging between 1,500 and 8,000 inhabitants. For the national aggregate analysis of exposure levels, the specific population of interest drawn from these data is the number of women aged 15 to 44 (i.e., childbearing age) in each tract. To predict populations in later years (2005 and 2016), we applied county-level population growth projections for the corresponding population category (Woods and Poole, 2008) to the 2000 tract-level data. To specifically estimate the portion of these populations that are pregnant in any given year, we applied state-level 2006 fertility rate (live births per 1,000 women aged 15 to 44 years) data from U.S. Vital Statistics (DHHS, 2009).

Two main sources of national-level recreation activity data are available and suitable for estimating the size and spatial distribution of freshwater recreational angler populations and activities in the United States:

- the National Survey of Fishing, Hunting, and Wildlife-Associated Recreation (FHWAR), maintained by the Department of the Interior (DOI) (DOI and DOC, 1992, 1997, 2002, 2007) and

- the National Survey of Recreation and the Environment (USDA, 1994).

FHWAR Angler Data. The FHWAR, conducted by the U.S. Census Bureau about every 5 years since 1955, includes data on the number and characteristics of participants as well as time and money spent on hunting, fishing, and wildlife watching. The most recent survey and report are for recreational activities conducted in 2006 (DOI and DOC, 2007). Data from this report were used to provide the most recent estimate of the percentage of the resident population in each state (16 years old or older) that engaged in freshwater fishing during the year. As shown in Table 5-1, these percentages vary from 3% (New Jersey) to 27% (Minnesota).

Table 5-1. Summary of FWHAR State-Level Recreational Fishing Characteristics

State	Freshwater Anglers as Percentage of State Population[a]	Percentage of Freshwater Fishing Trips[b]	
		Lakes	Rivers
Alabama	15.7%	59.9%	40.1%
Arizona	7.0%	79.2%	20.8%
Arkansas	19.5%	81.1%	18.9%
California	4.1%	53.5%	46.5%
Colorado	13.2%	63.7%	36.3%
Connecticut	6.4%	58.7%	41.3%
Delaware	5.0%	52.8%	47.2%
Florida	7.9%	67.4%	32.6%
Georgia	12.6%	70.4%	29.6%
Idaho	18.4%	44.4%	55.6%
Illinois	7.3%	76.4%	23.6%
Indiana	12.3%	77.8%	22.2%
Iowa	16.8%	55.1%	44.9%
Kansas	14.8%	84.7%	15.3%
Kentucky	17.5%	80.0%	20.0%
Louisiana	14.2%	71.2%	28.8%
Maine	19.4%	73.7%	26.3%
Maryland	5.5%	40.7%	59.3%
Massachusetts	5.1%	75.5%	24.5%
Michigan	14.2%	85.6%	14.4%
Minnesota	26.9%	89.0%	11.0%
Mississippi	19.6%	79.0%	21.0%
Missouri	18.9%	80.2%	19.8%
Montana	22.8%	46.8%	53.2%
Nebraska	12.3%	80.6%	19.4%
Nevada	5.9%	80.5%	19.5%
New Hampshire	8.9%	67.9%	32.1%
New Jersey	3.1%	68.9%	31.1%
New Mexico	10.9%	56.1%	43.9%
New York	4.7%	67.2%	32.8%
North Carolina	10.7%	68.7%	31.3%
North Dakota	17.3%	87.2%	12.8%
Ohio	11.8%	78.8%	21.2%
Oklahoma	18.8%	83.1%	16.9%
Oregon	13.6%	39.0%	61.0%

(continued)

Table 5-1. Summary of FWHAR State-Level Recreational Fishing Characteristics (continued)

State	Freshwater Anglers as Percentage of State Population[a]	Percentage of Freshwater Fishing Trips[b]	
		Lakes	Rivers
Pennsylvania	8.1%	44.0%	56.0%
Rhode Island	4.4%	73.5%	26.5%
South Carolina	14.2%	75.6%	24.4%
South Dakota	14.6%	69.7%	30.3%
Tennessee	13.8%	68.6%	31.4%
Texas	9.7%	79.3%	20.7%
Utah	15.6%	68.0%	32.0%
Vermont	12.6%	71.1%	28.9%
Virginia	7.5%	70.4%	29.6%
Washington	9.5%	50.0%	50.0%
West Virginia	19.7%	50.1%	49.9%
Wisconsin	22.8%	79.5%	20.5%
Wyoming	23.5%	64.0%	36.0%

[a] Based on FHWAR 2006 data for residents 16 years and older.

[b] Based on FHWAR 2001 data for residents 16 years and older.

The methodology for assessing mercury exposures also requires a further breakdown of freshwater fishing activities into two categories: rivers (including rivers and streams) and lakes (including lakes, ponds, reservoirs, and other flat water). Data at this level of detail are not reported in the summary national reports for the FHWAR; however, they are available from the FHWAR survey household-level data. For this analysis, data from a previous analysis and summary of the 2001 FHWAR household-level survey data (EPA, 2005) were used to provide estimates of the percentage of freshwater fishing days by residents in each state that were to either the lake or river category.[1] As shown in Table 5-1, the highest percentage going to lakes is in Minnesota (89%) and the highest to rivers is in Oregon (61%).

NSRE Angler Data. The NSRE, formerly known as the National Recreation Survey (NRS), is a nationally administered survey, which has been conducted periodically since 1962. It is designed to assess outdoor recreation participation in the United States and elicit information

[1] Although the total *number* of fishing trips varies from year to year, there is little reason to expect that the *ratio* of river trips to lake trips would have changed significantly since 2001. For this reason, given resource and timetable limitations, we did not update this input to the analysis.

regarding people's opinions about their natural environment. The NSRE sample of freshwater anglers is smaller than the FHWAR sample, but it is nonetheless a useful resource because it provides a wide variety of information about fishing activities. Importantly, it includes relatively detailed information about the nature and location of recent freshwater trips. Because the sampling procedure is designed to be representative, inferences may be drawn about the relative popularity of particular types of freshwater bodies (e.g., lakes, rivers) among the general public and the average distance traveled to reach these sites. Although more recent NSRE surveys have been conducted in 2000 and 2009, data from 1994 survey (NSRE, 1994) is used for this analysis because it contains the most detailed information regarding fishing trip destinations.

The NSRE 1994 elicited information from respondents about *the most recent* fishing trip. One of the main advantages of NSRE 1994 is that it includes geocoded data for reported fishing destinations. To specify the location of the last fishing trip, respondents were asked to provide the name of the waterbody, the nearest town to the waterbody, and an estimate of the distance and direction from their home to the waterbody. Appendix B describes how these data were used in this analysis to estimate the percentage of freshwater fishing trips that were in different distance intervals from respondents' homes. Using the demographic data from the NSRE, these estimates were further differentiated according to the income level and urban versus nonurban location of the respondents.

High-Consuming Subpopulations in the United States

Based on a detailed review of the literature, we identified several subpopulations with particularly high potential risks of mercury exposure due to relatively high rates of freshwater fish consumption (Moya, 2004; Burger 2002, Shilling et al. 2010, Dellinger, 2004). The analysis of potentially high-risk groups focuses on six subpopulations:

- low-income African-American recreational/subsistence fishers in the Southeast region[1]
- low-income white recreational/subsistence fishers in the Southeast region
- low-income female recreational/subsistence fishers
- Hispanic subsistence fishers

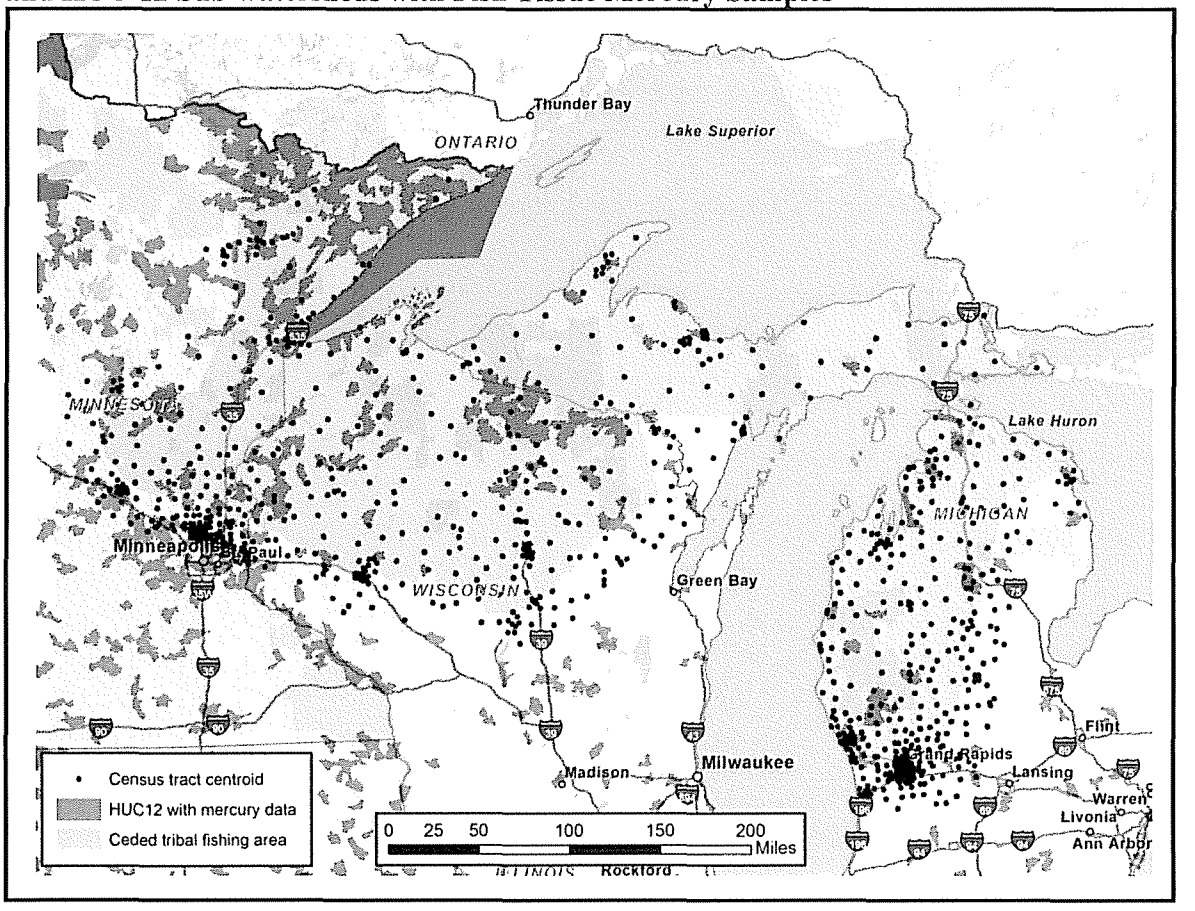
[1] Southeast for purposes of this analysis comprises Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, and West Virginia.

- Laotian subsistence fishers
- Chippewa/Ojibwe Tribe members in the Great Lakes area

To identify the size and spatial distribution of these potentially high-risk groups, we again used Census 2000 tract-level population data. These data identify tract-level populations in the year 2000 for each of the specified racial/ethnic definitions and, more specifically, for low-income African-American, white, and female populations. For this part of the analysis, the low-income designation is based on the tract-level estimates of subpopulations living in poverty. Population size projections for future years (beyond 2000) in each selected tract were based on county-level growth projections for the full population (all ages and both sexes) in the most closely corresponding race category (Woods and Poole, 2008). For example, the Asian and Native American categories in the county-level growth projection data were used for the Laotian and Chippewa population projections, respectively.

For the analysis of Chippewa subpopulation exposures, the analysis was spatially restricted to only include census tracts in Minnesota, Wisconsin, and Michigan with centroids that are located no more than 20 miles from the main tribal fishing area (the justification for this travel distance limitation is discussed below). The Chippewa tribal fishing areas in these states were defined as the territories around the Great Lakes that have been ceded to the Chippewa for tribal fishing rights. The boundaries of this tribal fishing area are shown in yellow in Figure 5-1.

Figure 5-1. Map of the Chippewa Tribal Fishing Area, Nearby Census Tract Centroids, and HUC-12 Sub-watersheds with Fish Tissue Mercury Samples



5.4.3 Mercury Concentrations in Freshwater Fish

Data Sources for Fish Tissue Concentrations

To characterize the spatial distribution of mercury concentration estimates in freshwater fish across the country, we compiled data from three main sources, which are described below.

National Listing of Fish Advisory (NLFA) database. The NLFA, managed by EPA (http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/), collects and compiles fish tissue sample data from all 50 states and from tribes across the United States. In particular, it contains data for over 43,000 mercury fish tissue samples collected from 1995 to 2007.

U.S. Geologic Survey (USGS) compilation of mercury datasets. As part of its Environmental Mercury Mapping and Analysis (EMMA) program, USGS compiled mercury fish tissue sample data from a wide variety of sources (including the NLFA) and has posted these

data at http://emmma.usgs.gov/datasets.aspx. The compilation includes (1) state-agency collected and reported data (including Delaware, Iowa, Indiana, Louisiana, Minnesota, Ohio, South Carolina, Virginia, Wisconsin, and West Virginia) from over 40,000 fish tissue samples, covering the period 1995 to 2007 and (2) over 10,000 fish tissue samples from several other sources, including the National Fish Tissue Survey, the National Pesticide Monitoring Program (NPMP), the National Contaminant Biomonitoring Program (NCBP), the Biomonitoring of Environmental Status and Trends (BEST) datasets of the USFWS and USGS (http://www.cerc.cr.usgs.gov/data/data.htm), and the Environmental Monitoring and Analysis Program (EMAP) (http://www.epa.gov/emap/).

EPA's National River and Stream Assessment (NRSA) study data. These data include nearly 600 fish tissue mercury samples collected at randomly selected freshwater sites across the United States during the period 2008 to 2009.

Approach for Compiling Fish Tissue Dataset for Exposure Analysis

Data from these three datasets were combined into a single master fish tissue dataset covering the period 1995 to 2009. One problem encountered in combining these datasets is the potential duplication of samples in the NLFA and USGS state-collected data. Unfortunately, these two datasets do not contain directly comparable and unique identifiers that allow duplicate samples to be easily identified and removed. Therefore, as an alternative, the samples from these two datasets were subdivided into data groups according to the year and state in which they were collected. If both datasets contained a data group for the same year and the same state, then the data group with the fewer number of observations was excluded from the master data.

The following criteria were also applied to exclude data from the master fish tissue dataset to be used in the analysis. Samples were excluded if they:

- did not include useable latitude-longitude coordinates for spatial identification;
- were located at sites outside the tidal boundaries of the continental United States (i.e., if they were not sampled from freshwater sites);
- did not come from fish species found in freshwater; or
- did not come from sampled fish that were at least 7 inches in length (i.e., unlikely to be consumed).

Each remaining sample was then categorized as either a river or lake sample based on information about the sampling site location. First, specific character strings in the site names (e.g., "river," "creek," "lake," "pond," and "reservoir") were used to classify sites. Second, remaining sites were categorized based on a GIS analysis that linked the sites' latitude-longitude coordinates to the nearest waterbody and its category.

The resulting master fish tissue mercury concentration dataset contains 26,940 sample concentration estimates from 3,876 river sites and 23,206 estimates from 2,167 lake sites.

A new dataset was then created by spatially grouping and averaging the river and lake concentration estimates at the HUC-12 sub-watershed level. First, all of the mercury sampling sites included in the master data were mapped and matched to the HUC-12 sub-watersheds in which they are located. A total of 3,884 HUC-12s in the continental United States (4.6%) contain at least one river or lake mercury sample.[1] Second, site-specific average mercury concentration values were generated by computing the mean concentration estimate at each site. Third, HUC-level average lake concentration estimates were computed as the mean of the site-specific average lake concentration estimates for each HUC containing at least one lake sampling site (1,396 HUCs). Fourth, HUC-level average river concentration estimates were computed as the mean of the site-specific average river concentration estimates for each HUC containing at least one river sampling site (2,655 HUCs).

Summary of Fish Tissue Mercury Concentration Estimates Used in the Exposure Analysis

The resulting HUC-level mercury concentration dataset is summarized in Table 5-2. The average HUC-level mercury concentration estimate for lakes is 0.29 ppm and for rivers is 0.26 ppm. The large standard deviations and ranges reported in the table also reflect the considerable spatial variation in lake and river concentration estimates across samples. As described below, the analysis uses this inter-watershed spatial variation (rather than just the average point estimate across watersheds) to estimate mercury exposures. However, in this analysis, exposure estimates were only generated for populations linked to these HUCs containing at least one river or lake mercury fish tissue sample.

[1] This number excludes 15 HUC-12s containing mercury samples. These HUC-12s were excluded from the analysis due to their proximity to potentially significant non-air sources of mercury, including gold mines or non-EGU mercury sources included in the 2008 Toxic Release Inventory.

Table 5-2. Summary of HUC-level Average Mercury Fish Tissue Concentration Estimates

	N[a]	*Mean*	*Std. Dev.*	*Min*	*Max*
Lake Fish Tissue Concentrations					
HUC-level average mercury concentration (ppm)	1,396	0.286	0.231	0.000	3.56
Number of lake samples per HUC	1,396	16.62	31.61	1	458
Number of lake sampling sites per HUC	1,396	1.55	1.97	1	33
River Fish Tissue Concentrations					
HUC-level average mercury concentration (ppm)	2,655	0.261	0.259	0.006	4.97
Number of river samples per HUC	2,655	10.15	22.45	1	288
Number of river sampling sites per HUC	2,655	1.46	1.10	1	16

[a] Number of HUC-12s with at least one river or lake sampling site

5.5 Linking Changes in Modeled Mercury Deposition to Changes in Fish Tissue Concentrations

5.5.1 Introduction

In the United States, humans are exposed to MeHg mainly by consuming fish that contain MeHg. Accordingly, to estimate changes in human exposure EPA must analyze how changes in Hg deposition from U.S. coal-fired power plants translate into changes in MeHg concentrations in fish. Quantifying the linkage between different levels of Hg deposition and fish tissue MeHg concentration is an important step in the risk assessment process and the focus of the material described in this chapter.

To effectively estimate fish MeHg concentrations in a given ecosystem, it is important to understand that the behavior of Hg in aquatic ecosystems is a complex function of the chemistry, biology, and physical dynamics of different ecosystems. The majority (95 to 97 percent) of the Hg that enters lakes, rivers, and estuaries from direct atmospheric deposition is in the inorganic form (Lin and Pehkonen, 1999). Microbes convert a small fraction of the pool of inorganic Hg in the water and sediments of these ecosystems into the organic form of Hg (MeHg). MeHg is the only form of Hg that biomagnifies in organisms (Bloom, 1992). Ecosystem-specific factors that affect both the bioavailability of inorganic Hg to methylating microbes (e.g., sulfide, dissolved

organic carbon) and the activity of the microbes themselves (e.g., temperature, organic carbon, redox status) determine the rate of MeHg production and subsequent accumulation in fish (Benoit et al., 2003). The extent of MeHg bioaccumulation is also affected by the number of trophic levels in the food web (e.g., piscivorous fish populations) because MeHg biomagnifies as large piscivorous fish eat smaller organisms (Watras and Bloom, 1992; Wren and MacCrimmon, 1986). These and other factors can result in considerable variability in fish MeHg levels among ecosystems at the regional and local scale.

Use of Mercury Maps to Project Changes in Fish Tissue Concentrations

To analyze the relationship between Hg deposition and MeHg concentrations in fish in freshwater aquatic ecosystems across the U.S. for the national scale benefits assessment, EPA applied EPA's Office of Water's Mercury Maps (MMaps) approach (US EPA, 2001a). MMaps implements a simplified form of the IEM-2M model applied in EPA's Mercury Study Report to Congress (USEPA, 1997). By simplifying the assumptions inherent in the freshwater ecosystem models that were described in the Report to Congress, the MMaps model showed that these models converge at a steady-state solution for MeHg concentrations in fish that are proportional to changes in Hg inputs from atmospheric deposition (i.e.,., over the long term, fish concentrations are expected to decline proportionally to declines in atmospheric loading to a waterbody). The temporal response time for a change in fish tissue MeHg levels following a change in mercury deposition can range from years to decades or more depending on the attributes of the watershed and waterbody involved.[1]

MMaps has several limitations:

1. The MMaps approach is based on the assumption of a linear, steady-state relationship between concentrations of MeHg in fish and present day air deposition mercury inputs. We expect that this condition will likely not be met in many waterbodies because of recent changes in mercury inputs and other environmental variables that affect mercury bioaccumulation. For example, the US has recently reduced human-caused emissions while international emissions have increased.

[1] Research has suggested that fish tissue MeHg levels in some locations may display a multi-phase response following a discrete change in mercury deposition, with the first phase lasting a few years to a decade or more and primarily involving changes in aerial loading directly to the waterbody and the second phase lasting decade (to a century or more) and reflecting longer-term changes in watershed erosion and runoff to the waterbody (Knights et al., 2009, Harris et al., 2007).

2. The requirement that environmental conditions remain constant over the time required to reach steady state inherent in the MMaps methodology may not be met, particularly in systems that respond slowly to changes in mercury inputs.
3. Many water bodies, particularly in areas of historic gold and mercury mining, contain significant non-air sources of mercury. The MMaps methodology will yield biased results when applied to such waterbodies. As a simple illustrative example, if we have mercury deposition of 100 at a given location and a MeHg fish concentration of 6 in a local fish tissue sample, and a new emissions rule reduces deposition by half to 50, then, in the absence of other non-air deposition sources, we would assume that the MeHg fish concentration is reduced by the same proportion, to 3 ((50 / 100) x 6). However, if total pre-control mercury loading to the system is actually 100 *plus* another unaccounted for source (for example, an additional 100 due to area gold mining), then the MeHg fish concentration of 6 is actually due to 200 in total mercury loading. In this case, reducing mercury air deposition from 100 to 50 would only reduce the total loading by 25%, to 150, which, based on the MMaps methodology, would result in a MeHg fish concentration of 4.5 ((150 / 200) x 6) rather than 3. In areas where on-air deposition sources are unaccounted for, MMaps-based estimates of changes in MeHg fish tissue concentrations due to reduced mercury air emissions would therefore be biased high.
4. Finally, MMaps does not provide for a calculation of the time lag between a reduction in mercury deposition and a reduction in the MeHg concentrations in fish and, as noted earlier, depending on the nature of the watersheds and waterbodies involved, the temporal response time for fish tissue MeHg levels following a change in mercury deposition can range from years to decades.[1]

This methodology therefore applies only to situations where air deposition is the sole significant source of Hg to a water body, and where the physical, chemical, and biological characteristics of the ecosystem remain constant over time. EPA recognizes that concentrations of MeHg in fish across all ecosystems may not reach steady state and that ecosystem conditions affecting mercury dynamics are unlikely to remain constant over time. EPA further recognizes that many water bodies, particularly in areas of historic gold and Hg mining in western states,

[1] As noted earlier in Section 5.1,monetized benefits estimates are for an immediate change in MeHg levels in fish (i.e., the potential lag period associated with fully realizing fish tissue MeHg levels was not reflected in benefits modeling). If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response.

contain significant non-air sources of Hg. Finally, EPA recognizes that MMaps does not provide for a calculation of the time lag between a reduction in Hg deposition and a reduction in the MeHg concentrations in fish. While acknowledging these limitations, EPA is unaware of any other tool for performing a national-scale assessment of the change in fish MeHg concentrations resulting from reductions in atmospheric deposition of Hg. The following paragraphs provide additional details on the above limitations, as well as a brief assessment of the degree to which conditions match those assumptions. The MMaps model (US EPA, 2001a) assumes that for long-term steady-state conditions, reductions in fish tissue concentrations are expected to track linearly with reductions in air deposition watershed loads.

The MMaps model represents a reduced form of the IEM-2M and MCM models used in the Mercury Study Report to Congress (USEPA, 1997), as well as the subsequent Dynamic MCM (D-MCM) model (Harris et al., 1996). That is, the equations of these mercury fate and transport models are reduced to steady state and consolidated into a single equilibrium equation equating the ratio of future/current air deposition rates to future/current fish tissue concentrations. At certain sites, the MMaps model has been shown to produce results equivalent to those of these complex models over the long term, under a specific set of conditions.

Though plainly stated, the steady-state assumption is a compilation of a number of individual conditions. For example, fish tissue data may not represent average, steady-state concentrations for two major reasons:

- Fish tissue and deposition rate data for the base period are not at steady state. Where deposition rates have recently changed, the watershed or waterbody may not have had sufficient time to fully respond. The pool of mercury in different media could be sufficiently large relative to release rates, and thus needs more time to achieve a new equilibrium. This is more likely to occur in deeper lakes and lakes with large catchments where turnover rates are longer and where the watershed provides significant inputs of mercury.
- Fish tissue data do not represent average conditions (or conditions of interest for forecast fish levels). Methylation and bioaccumulation are variable and dynamic processes. If fish are sampled during a period of high or low methylation or bioaccumulation, they would not be representative of the average, steady-state or dynamic equilibrium conditions of the waterbody. This effect is significantly more pronounced in small and juvenile fish. Examples include tissue data collected during a drought or during conditions of fish starvation. Other examples include areas in which seasonal fluctuations in fish mercury levels are significant due, for example, from seasonal runoff of contaminated soils from

abandoned gold and mercury mines or areas geologically rich in mercury. In such a case, MMaps predictions would be valid for similar conditions (e.g. wet year/dry year, or season) in the future, rather than typical or average conditions. Alternatively, sufficient fish tissue would need to be collected to get an average concentration that represents a baseline dynamic equilibrium.

Other ecosystem conditions might cause projections from the MMaps approach to be inaccurate for a particular ecosystem. Watershed and waterbody conditions can undergo significant changes in capacity to transport, methylate, and bioaccumulate mercury. Examples of this include regions where sulfate and/or acid deposition rates are changing (in turn affecting MeHg production independently of total mercury loading), and where the trophic status of a waterbody is changing. A number of other water quality parameters have been correlated with increased fish tissue concentrations (e.g. low pH, high DOC, lower algal concentrations), but these relationships are highly variable among different waterbodies. MMaps will be biased when waterbody characteristics change between when fish were initially sampled, and the new conditions of the waterbody.

As stated above, the relationship between the change in mercury deposition from air to the change in fish tissue concentration holds only when air deposition is the predominant source of the mercury load to a waterbody. Due to this requirement in the model, the national application of the MMaps approach screened out those watersheds that either contained active gold mines or had other substantial non-US EGU anthropogenic releases of mercury. Identification of watersheds with gold mines was based on a 2005 USGS data set characterizing mineral and metal operations in the United States. The data represent commodities monitored by the National Minerals Information Center of the USGS, and the operations included are those considered active in 2003 (online link: http://tin.er.usgs.gov/mineplant/). The identification of watersheds with substantial non-EGU anthropogenic emissions was based on a TRI-net query for 2008 of non-EGU mercury sources with total annual on-site Hg emissions (all media) of 39.7 pounds or more. This threshold value corresponds to the 25th percentile annual US-EGU mercury emission value as characterized in the 2005 NATA. EPA considered the 25th percentile US-EGU emission level to be a reasonable screen for additional substantial non-US EGU releases to a given watershed.

It should be noted that MMaps was designed to address an important, but very specific issue – that of eventual response of fish tissue to air deposition reductions. As such it responds to a need to understand how mercury reductions, independent of other changes in the environment, will impact fish contamination and human health. More complex models are required in cases

where more complete descriptions are needed. A dynamic model is essential for modeling waterbody recovery during the period in which waterbody response lags reductions in mercury loads. A dynamic model is also essential for understanding seasonal fluctuations, as well as year-to-year fluctuations due to meteorological variability. Finally, a more complex model would be essential for assessing the impact of other watershed and water quality changes (e.g. erosion, wetlands coverage, and acid deposition) that might affect mercury bioaccumulation in fish. These complex models are used to derive the MMaps approach, and are themselves based on a number of assumptions. While these assumptions are considered reasonable given the state of the science of environmental modeling and mercury in the environment, the validity of assumptions inherent in both the MMaps approach and dynamic ecosystem scale models will need to be reevaluated as the science of mercury fate and transport evolves.

The MMaps methodology was peer reviewed by a set of national experts in the fate and transport of mercury in watersheds (US EPA, 2001a). While two reviewers felt it could be used to predict future fish tissue concentrations, a third cautioned it should not be considered a robust predictor until scientific data can be generated to validate the approach. Reviewers systematically identified a set of implicit assumptions that compose the steady state assumption in the MMaps approach. They pointed out that due to evolving and complex nature of the science of mercury, some features of the complex models are assumptions themselves, and thus cannot be wholly relied upon as ultimate predictors of mercury fate and transport. The reviewers pointed out that there is limited scientific information to directly verify this approach, and that some scientific data appears to refute individual components of the overall steady state assumption. One reviewer did perform a D-MCM and MMaps comparison, and found that, under these assumptions, MMaps model did produce comparable steady-state results as the D-MCM model. There was considerable discussion about how best to aggregate the data, to scale up to a deposition reduction requirement, from fish-specific and waterbody specific information. The description of the approach and the methodologies as applied in this analysis are largely consistent with the peer review recommendations.

The MMaps report (US EPA, 2001a) presented a national-scale application of Mercury Maps to determine the percent reductions in air deposition that would be needed in watersheds across the country for average fish tissue concentrations to achieve the national MeHg criterion. In this national-scale assessment, fish tissue concentrations were aggregated at the scale of large watersheds, thus presenting average results for each watershed. The use of other scales of aggregation, e.g., waterbody specific, is consistent with the MMaps approach to the degree to which different mercury loads can be discerned.

5.5.2 The Science of Mercury Processes and Variability in Aquatic Ecosystems

The set of physical, chemical, and biological processes controlling mercury fate in watersheds and water bodies can be grouped into specific categories: mercury cycle chemistry; mercury processes in the atmosphere, soils and water; bioavailability of mercury in water; and mercury accumulation in the food web. The following is a review of these categories, discussing the related scientific developments that have added to our understanding of mercury processes. This review builds upon the work previously summarized in EPA's Mercury Report to Congress (USEPA, 1997).

Mercury Cycle Chemistry

Mercury occurs naturally in the environment as several different chemical species. The majority of mercury in the atmosphere (95-97%) is present in a neutral, elemental state (Hg^0) (Lin and Pehkonen, 1999), while in water, sediments and soils the majority of mercury is found in the oxidized, divalent state (Hg(II)) (Morel et al., 1998). A small fraction (percent) of this pool of divalent mercury is transformed by microbes into MeHg (CH3Hg(II)/ MeHg) (Jackson, 1998). MeHg is retained in fish tissue and is the only form of mercury that biomagnifies in aquatic food webs (Kidd et al., 1995). As a result, MeHg concentrations in higher trophic level organisms such as piscivorous fish, birds and wildlife are often 104-106 times higher than aqueous MeHg concentrations (Jackson, 1998). Transformations among mercury species within and between environmental media result in a complicated chemical cycle. Mercury emissions from both natural and anthropogenic sources are predominantly as Hg(II) species and Hg^0 (Landis and Keeler, 2002; Seigneur et al., 2004). Anthropogenic point sources of mercury consist of combustion (e.g., utility boilers, municipal waste combustors, commercial/industrial boilers, medical waste incinerators) and manufacturing sources (e.g., chlor-alkali, cement, pulp and paper manufacturing) (USEPA, 1997). Natural sources of mercury arise from geothermic emissions such as crustal degassing in the deep ocean and volcanoes as well as dissolution of mercury from geologic sources (Rasmussen, 1994).

Mercury Processes in the Atmosphere

The relative contributions of local, regional and long range sources of mercury to fish mercury levels in a given water body are strongly affected by the speciation of natural and anthropogenic emissions sources. Elemental mercury is oxidized in the atmosphere to form the more soluble mercuric ion (Hg(II)) (Schroeder et al., 1989). Particulate and reactive gaseous phases of Hg(II) are the principle forms of mercury deposited onto terrestrial and aquatic systems because they are more efficiently scavenged from the atmosphere through wet and dry deposition than Hg0 (Lindberg and Stratton, 1998). Because Hg(II) species or reactive gaseous mercury

(RGM) and particulate mercury (Hg(p)) in the atmosphere tend to be deposited more locally than Hg^0, differences in the species of mercury emitted affect whether it is deposited locally or travels longer distances in the atmosphere (Landis et al., 2004).

Mercury Processes in Soils

A portion of the mercury deposited in terrestrial systems is re-emitted to the atmosphere. On soil surfaces, sunlight may reduce deposited Hg(II) to Hg^0, which may then evade back to the atmosphere (Carpi and Lindberg, 1997; Frescholtz and Gustin, 2004; Scholtz et al., 2003). Significant amounts of mercury can be co-deposited to soil surfaces in throughfall and litterfall of forested ecosystems (St. Louis et al., 2001), and exchange of gaseous Hg^0 by vegetation has been observed (e.g., (Gustin et al., 2004).

Hg(II) has a strong affinity for organic compounds such that inorganic Hg in soils and wetlands is predominantly bound to dissolved organic matter (Mierle and Ingram, 1991). MeHg likewise forms stable complexes with solid and dissolved organic matter (Hintelmann and Evans, 1997). These complexes can dominate MeHg speciation under aerobic conditions (Karlsson and Skyllberg, 2003). Truly dissolved and dissolved organic carbon (DOC)-complexed Hg(II) and MeHg are transported by percolation to shallow groundwater, and by runoff to adjacent surface waters (Ravichandran, 2004). Sorbed Hg(II) and MeHg are transported by erosion fluxes to depositional areas on the watershed and to adjacent surface waters (e.g., (Hurley et al., 1998).

Concentrations of MeHg in soils are generally very low. In contrast, wetlands are areas of enhanced MeHg production and account for a significant fraction of the external MeHg inputs to surface waters that have watersheds with a large portion of wetland coverage (e.g., St. Louis et al., 2001). Accordingly, there is a positive relationship between MeHg yield and percent wetland coverage (Hurley et al., 1995). Hydrology exerts an important control on the magnitude and flux of MeHg in wetland ecosystems (Branfireun and Roulet, 2002), as well as the transport of inorganic mercury deposited in a given watershed to surface waters (Babiarz et al., 2001).

Mercury Processes in Water

In a water body, deposited Hg(II) is reduced to Hg^0 by ultraviolet and visible wavelengths of sunlight as well as microbially mediated reduction pathways (Amyot et al., 2000; Mason et al., 1995). In turn, Hg^0 is oxidized back to Hg(II), driven by sunlight as well as by "dark" chemical or biochemical processes (Lalonde et al., 2001; Zhang and Lindberg, 2001). Driven by wind and water currents, dissolved Hg^0 in the water column is volatilized, which can be a significant removal mechanism for mercury in surface waters and a net source of mercury to the atmosphere (Siciliano et al., 2002).

In the water column and sediments, Hg(II) partitions strongly to silts and biotic solids, sorbs weakly to sands, and complexes strongly with dissolved and particulate organic material. The abundance of various inorganic ligands (e.g., OH^-, Cl^-, S^{2-}, DOC) in freshwater and saltwater ecosystems plays an important role in both oxidation and reduction of inorganic mercury as well as its bioavailability to methylating microbes. For example, reduction of Hg(II) is hypothesized to be a function of the predominance of $Hg(OH)_2$, which is inversely correlated with pH (Mason et al., 1995). Reduction of Hg(II) to Hg^0 and subsequent volatilization from the water column is important because it effectively reduces the pool of inorganic mercury that could potentially undergo conversion to MeHg.

Hg(II) and MeHg sorbed to solids settle out of the water column and accumulate on the surface of the benthic sediment layer. Surficial sediments interact with the water column via resuspension and bioturbation. The burial of sediments below the surficial zone can be a significant removal mechanism for contaminants in surface sediments (e.g., Gobas et al., 1998; Gobas et al., 1995). The depth of the active sediment layer is a highly sensitive parameter for predicting the temporal response of different ecosystems to changes in mercury loading in environmental fate models. This is because the reservoir of Hg(II) potentially available for conversion to MeHg in the sediments is a function of the depth and volume of the active sediment layer. The compartment conducive for methylation is similarly affected (Harris and Hutchison, 2003; Sunderland et al., 2004). Physical characteristics of different ecosystem types affect estuarine mixing and sediment resuspension, which also affect the production of MeHg in the water and sediments (Rolfhus et al., 2003; Sunderland et al., 2004; Tseng et al., 2001).

Bioavailability of Inorganic Mercury to Methylating Microbes

The amount of bioavailable MeHg in water and sediments of aquatic systems is a function of the relative rates of mercury methylation and demethylation. In the water, MeHg is degraded by two microbial processes and sunlight (Barkay et al., 2003; Sellers et al., 1996). Recent research has shown that demethylating Hg-resistant bacteria may adapt to systems that are highly contaminated with total mercury, helping to explain the paradox of low MeHg and fish Hg levels in these systems (Schaefer et al., 2004).

Mass balances for a variety of lakes and coastal ecosystems show that in situ production of MeHg is often one of the main sources of MeHg in the water and sediments (Benoit et al., 1998; Bigham and Vandal, 1994; Gbundgo-Tugbawa and Driscoll, 1998; Gilmour et al., 1998; Mason et al., 1999). Sulfate-reducing bacteria (SRB) are thought to be the principle agents responsible for the majority of MeHg production in aquatic systems (Beyers et al., 1999;

Compeau and Bartha, 1987; Gilmour and Henry, 1991). SRB thrive in the redoxocline, where the maximum gradient between oxic and anoxic conditions exists (Hintelmann et al., 2000). Thus, in addition to the presence of bioavailable Hg(II), MeHg production and accumulation in aquatic systems is a function of the geochemical parameters that enhance or inhibit the activity of methylating microbes, especially sulfur concentrations, redox potential (Eh) and the composition and availability of organic carbon.

A number of factors affect the bioavailabilty of Hg(II). A strong inverse relationship between complexation of Hg(II) by sulfides and MeHg production has been demonstrated in a number of studies (Benoit et al., 1999a; Benoit et al., 1999b; Craig and Bartlett, 1978; Craig and Moreton, 1986). Passive diffusion of dissolved, neutral inorganic mercury species is hypothesized as one of the main modes of entry across the cell membranes of methylating microbes (Benoit et al., 1999a; Benoit et al., 2003; Benoit et al., 1999b). Thus, the formation of neutral, dissolved mercury species such as $HgCl_2$, $Hg(OH)_2$, HgClOH, and HgS^0(aq.), which depend on the availability of constituent ligands in the surface and interstitial waters, may strongly influence the availability of inorganic mercury to SRB, although our understanding of the forms of mercury that are bioavailable to methylating microbes is currently incomplete (Benoit et al., 2001; Benoit et al., 1999a; King et al., 2001). See Section 5.7.5.1 below for additional detail on the relationship between sulfur deposition and mercury methylation.

Changes in the bioavailability of inorganic mercury and the activity of methylating microbes as a function of sulfur, carbon and ecosystem specific characteristics mean that ecosystem changes and anthropogenic "stresses" that do not result in a direct increase in mercury loading to the ecosystem but alter the rate of MeHg formation may also affect mercury levels in organisms (Grieb et al., 1990). Because mercury concentrations in fish can increase even when there has been no change in the total amount of mercury deposited in the ecosystem, environmental changes such as eutrophication, which may alter microbial activity and the chemical dynamics of mercury within an ecosystem, must be considered together with emission control strategies to effectively manage mercury accumulation in the food web.

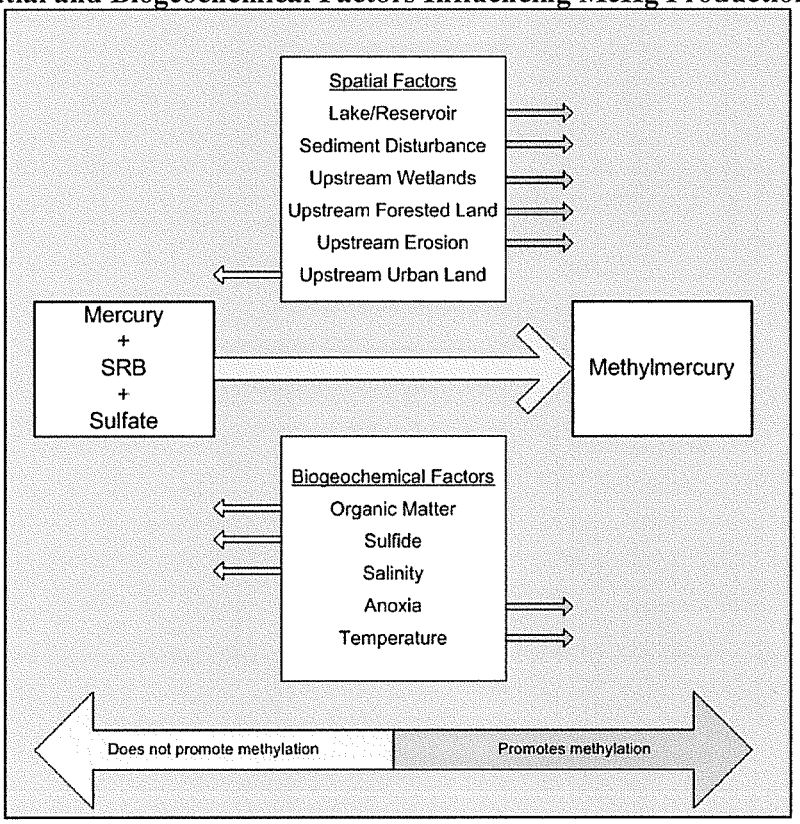
Recent research indicates that the bioavailability or reactivity of newly deposited Hg(II) may be greater than older "legacy" mercury in the system (Hintelmann et al., 2002). These results suggest that lakes receiving the bulk of their mercury directly from deposition to the lake surface (e.g., some seepage lakes) would see fish mercury concentrations respond more rapidly to changes in atmospheric deposition than lakes receiving most of their mercury from watershed runoff. The implications of these data are also that systems with a greater surface area to watershed area ratio that receive most of their inputs directly from the atmosphere (e.g., seepage

lakes) may respond more rapidly to changes in emissions and deposition of mercury than those receiving significant inputs of mercury from the catchment area.

Sulfur and Mercury Methylation

EPA's *2008 Integrated Science Assessment (ISA) for Oxides of Nitrogen and Sulfur–Ecological Criteria (Final Report)* concluded that evidence is sufficient to infer a casual relationship between sulfur deposition and increased mercury methylation in wetlands and aquatic environments. Specifically, there appears to be a relationship between SO_4^{2-} deposition and mercury methylation; however, the rate of mercury methylation varies according to several spatial and biogeochemical factors whose influence has not been fully quantified (see Figure 5-2). Therefore, the correlation between SO_4^{2-} deposition and MeHg could not be quantified for the purpose of interpolating the association across waterbodies or regions. Nevertheless, because changes in MeHg in ecosystems represent changes in significant human and ecological health risks, the association between sulfur and mercury cannot be neglected (EPA, 2008, Sections 3.4.1 and 4.5).

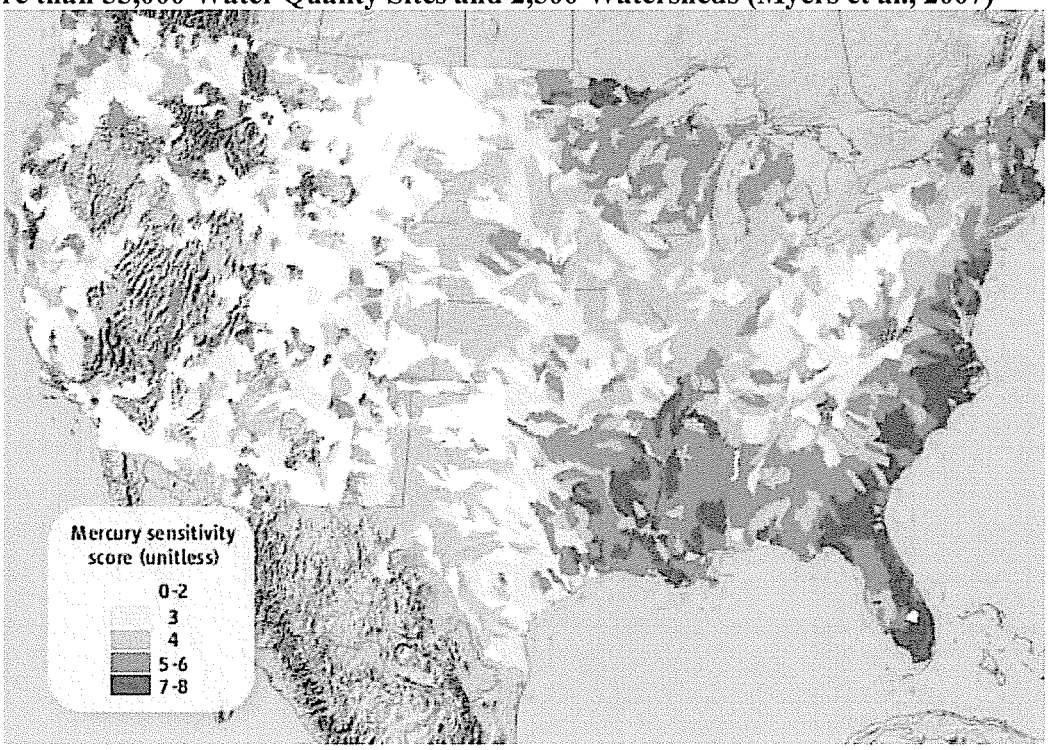
Figure 5-2. Spatial and Biogeochemical Factors Influencing MeHg Production



As research evolves and the computational capacity of models expands to meet the complexity of mercury methylation processes in ecosystems, the role of interacting factors may be better parsed out to identify ecosystems or regions that are more likely to generate higher concentrations of MeHg. Figure 5-3 illustrates the type of current and forward-looking research being developed by the U.S. Geological Survey (USGS) to synthesize the contributing factors of mercury and to develop a map of sensitive watersheds. The mercury score referenced in Figure 5-3 is based on SO_4^{2-} concentrations, acid neutralizing capacity (ANC), levels of dissolved organic carbon and pH, mercury species concentrations, and soil types to gauge the methylation sensitivity (Myers et al., 2007).

Interdependent biogeochemical factors preclude the existence of simple sulfate-related mercury methylation models (see Figure 5-3). It is clear that decreasing sulfate deposition is likely to result in decreased MeHg concentrations. Future research may allow for the characterization of a usable sulfate-MeHg response curve; however, no regional or classification calculation scale can be created at this time because of the number of confounding factors.

Figure 5-3. Preliminary USGS Map of Mercury Methylation–Sensitive Watersheds Derived from More than 55,000 Water Quality Sites and 2,500 Watersheds (Myers et al., 2007)



Decreases in SO_4^{2-} deposition have already shown promising reductions in MeHg. Observed decreases in MeHg fish tissue concentrations have been linked to decreased

acidification and declining SO_4^{2-} and mercury deposition in Little Rock Lake, WI (Hrabik and Watras, 2002), and to decreased SO_4^{2-} deposition in Isle Royale in Lake Superior, MI (Drevnick et al., 2007). Although the possibility exists that reductions in SO_4^{2-} emissions could generate a pulse in MeHg production because of decreased sulfide inhibition in sulfate-saturated waters, this effect would likely involve a limited number of U.S. waters (Harmon et al., 2007). Also, because of the diffusion and outward flow of both mercurysulfide complexes and SO_4^{2-}, increased mercury methylation downstream may still occur in sulfate-enriched ecosystems with increased organic matter and/or downstream transport capabilities.

Remediation of sediments heavily contaminated with mercury has yielded significant reductions of MeHg in biotic tissues. Establishing quantitative relations in biotic responses to MeHg levels as a result of changes in atmospheric mercury deposition, however, presents difficulties because direct associations can be confounded by all of the factors discussed in this section. Current research does suggest that the levels of MeHg and total mercury in ecosystems are positively correlated, so that reductions in mercury deposited into ecosystems would also eventually lead to reductions in MeHg in biotic tissues. Ultimately, an integrated approach that involves the reduction of both sulfur and mercury emissions may be most efficient because of the variability in ecosystem responses. Reducing SO_X emissions could have a beneficial effect on levels of MeHg in many waters of the United States.

Mercury Accumulation in the Food Web

Dissolved Hg(II) and MeHg accumulate in aquatic vegetation, phytoplankton, and benthic invertebrates. Unlike Hg(II), MeHg biomagnifies though each successive trophic level in both benthic and pelagic food chains such that mercury in predatory, freshwater fish is found almost exclusively as MeHg (Bloom, 1992; Watras et al., 1998). Thus, trophic position and food-chain complexity plays an important role in MeHg bioaccumulation (Kidd et al., 1995). The chemical and physical characteristics of different ecosystems affect MeHg uptake at the base of the food chain, driving bioaccumulation at higher trophic levels. At the base of pelagic freshwater food-webs, MeHg uptake by plankton is thought to be a combination of passive diffusion and facilitated transport (Laporte et al., 2002; Watras et al., 1998). Uptake of MeHg by plankton can be enhanced or inhibited by the presence of different ligands bound to MeHg (Lawson and Mason, 1998). Similarly, the assimilation efficiency of MeHg at the base of the food chain is also affected by the type of dissolved MeHg-complexes in the water and sediments. This may be a function of differences in the ability of organisms to solubilize MeHg through digestive processes with different MeHg complexes (Lawrence and Mason, 2001; Leaner and Mason, 2002). The presence of organic ligands and high concentrations of DOC in aquatic

ecosystems are generally thought to limit MeHg uptake by biota (Driscoll et al., 1995; Sunda and Huntsman, 1998; Watras et al., 1998).

In fish, MeHg bioaccumulation is a function of several uptake (diet, gills) and elimination pathways (excretion, growth dilution) (Gilmour et al., 1998; Greenfield et al., 2001). As a result, the highest mercury concentrations for a given fish species correspond to smaller, long-lived fish that accumulate MeHg over their life span with minimal growth dilution (e.g., (Doyon et al., 1998). In general, higher mercury concentrations are expected in top predators, which are often large fish relative to other species in a waterbody.

5.5.3 Summary

In the United States, humans are exposed to MeHg mainly by consuming fish that contain MeHg. Aquatic ecosystems respond to changes in mercury deposition in a highly variable manner as a function of differences in their chemical, biological and physical properties. Depending on the characteristics of a given ecosystem, methylating microbes convert a small but variable fraction of the inorganic mercury in the sediments and water derived from human activities and natural sources into MeHg. MeHg is the only form of mercury that biomagnifies in the food web. Concentrations of MeHg in fish are generally on the order of a million times the MeHg concentration in water. In addition to mercury deposition, key factors affecting MeHg production and accumulation in fish include the amount and forms of sulfur and carbon species present in a given waterbody. Thus, two adjoining water bodies receiving the same deposition can have significantly different fish mercury concentrations.

For this analysis, EPA used the Mercury Maps (MMaps) model to estimate changes in freshwater fish mercury concentrations resulting from changes in mercury deposition after regulation of mercury emissions from U.S. coal-fired power plants. MMaps, a simplified form of the IEM-2M model applied in EPA's 1997 Mercury Study Report to Congress, is a static model that assumes a proportional relationship between declines in atmospheric mercury deposition and concentrations in fish at steady state. This means, for example, that a 50% decrease in mercury deposition rates is projected to lead to a 50% decrease in mercury concentrations in fish. MMaps does not consider the dynamics of relevant ecosystem specific factors that can affect the methylation and bioaccumulation in fish in different water bodies over time, nor does it consider the inputs of non-air sources to the watershed. In all cases, the MMaps model does not address the lag time of different ecosystems to reach steady state (i.e., when fish mercury concentrations reflect changes in atmospheric deposition). In addition, applying the MMaps model assumes that atmospheric deposition is the principle source of mercury to the waterbodies being investigated

and environmental factors that affect MeHg production and accumulation in organisms will remain constant, allowing each ecosystem to reach steady state. While MMaps has several limitations, EPA knows of no alternative tool for performing a national-scale assessment of such changes.

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5.7 Analysis of the Dose-Response Relationship Between Maternal Mercury Body Burden and Childhood IQ

5.7.1 Introduction

In considering possible health endpoints for quantification and monetization, EPA reviewed the scientific literature on the health effects of mercury, including the "Toxicological Effects of Methylmercury," published by the National Research Council (NRC) in 2000 (NRC, 2000).

EPA chose to focus on quantification of intelligence quotient (IQ) decrements associated with prenatal mercury exposure as the initial endpoint for quantification and valuation of mercury health benefits. Reasons for this initial focus on IQ included the availability of thoroughly-reviewed, high-quality epidemiological studies assessing IQ or related cognitive outcomes suitable for IQ estimation, and the availability of well-established methods and data for economic valuation of avoided IQ deficits, as applied in EPA's previous benefits analyses for childhood lead exposure.

Epidemiological studies of prenatal mercury exposure conducted in the Faroe Islands (Grandjean et al., 1997), New Zealand (Kjellstrom et al., 1989; Crump et al., 1998), and the Seychelles Islands (Davidson et al., 1998; Myers et al., 2003) have examined neurodevelopmental outcomes through the administration of tests of cognitive functioning. Each of these studies included some but not all of the following tests: full-scale IQ, performance IQ, problem solving, social and adaptive behavior, language functions, motor skills, attention, memory and other functions. The NRC reviewed the studies and determined that "Each of the studies was well designed and carefully conducted, and each examined prenatal MeHg exposures within the range of the general U.S. population exposures" (NRC, 2000).

As part of previous analyses, EPA attempted to identify the appropriate dose-response coefficients from the Faroe Islands, New Zealand, and Seychelles Islands studies, and devised a statistical approach for combining those coefficients to provide an integrated estimate of the IQ dose-response coefficient.

For this assessment, EPA used a more recently revised estimate of the IQ dose-response function, based on a peer-reviewed study by Axelrad et al. (2007) ("the Axelrad study"). The Axelrad study estimated a dose-response relationship between maternal mercury body burden and subsequent childhood decrements in IQ using a Bayesian hierarchical model to integrate data from the Faroe Islands, New Zealand, and Seychelles Islands studies.

The Axelrad study used a linear model that goes through the origin to fit population-level dose-response relationships to the pooled data from the three studies. The application of a linear model should not be interpreted to suggest that any of the three studies used have data showing health effects from MeHg exposure at or below the RfD. The RfD is an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (EPA, 2002). EPA believes that exposures at or below the RfD are unlikely to be associated with appreciable risk of deleterious effects. It is important to note, however, that the RfD does not define an exposure level corresponding to zero risk; mercury exposure near or below the RfD could pose a very low level of risk which EPA deems to be non-appreciable. It is also important to note that the RfD does not define a bright line, above which individuals are necessarily at risk of adverse effect. Use of a linear model that goes through the origin, rather than one that reflects a threshold effect is technically more simple and practical. It associates an increment of IQ benefit with a given reduction in exposure. A linear model allows us to estimate the benefits of reductions in exposure due to power plants without a complete assessment of other sources of exposure. Other models would require information on the joint distribution of exposure from power plants and other sources to estimate the benefits of reducing the exposure due to power plants, which would require much more precise information about consumption patterns.

5.7.2 Epidemiological Studies of Mercury and Neurodevelopmental Effects

The IQ dose-response estimate is based on data from three major prospective studies investigating potential neurotoxicity of low-level, chronic mercury exposure: the Faroe Islands study, the New Zealand study, and the Seychelles Child Development Study.

In assembling the New Zealand sample, Kjellstrom et al. (1989) ascertained the fish consumption of 10,930 of 16,293 pregnant women in the study area. They identified 935 women

who reportedly consumed fish at least 3 times per week. Hair samples were obtained from these women, and 73 were found to have a hair mercury level of 6 parts per million (ppm) or greater. In this group, the mean was 8.3 ppm, with a range of 6 to 86 ppm, although only one woman had a level greater than 20 ppm. Each woman with 6 ppm hair mercury or greater was matched to 3 controls - one with hair mercury between 3-6 ppm, one with hair mercury less than 3 ppm and high fish consumption, and one with hair mercury less than 3 ppm and low fish consumption. Ethnic group, age, smoking, residence time in New Zealand, and child sex were also used to select controls. The final study group included 237 children, including 57 fully matched sets of 4 children. Although children were assessed at 4 and 6 years of age, only the data collected at the older age is considered in this analysis, as the reliability and validity of neurodevelopmental testing generally increases with child age.

The Faroe Islands investigators assembled a birth cohort of 1,353 newborns recruited from three hospitals over a 21-month period in 1986-1987. In 1,022 women, two biomarkers of prenatal mercury exposure were collected: cord-blood mercury, and maternal hair mercury at delivery. Neurodevelopmental assessments of 917 children were conducted at age 7 (Grandjean et al., 1997). For these 917 children, the geometric mean concentration of mercury in cord-blood was 22.6 parts per billion (ppb) (inter-quartile range 13.1 – 40.5 ppb, full range 0.9 – 351 ppb). The geometric mean concentration of mercury in maternal hair was 4.2 ppm (inter-quartile range: 2.5-7.7 ppm, full range 0.2 – 39.1 ppm) (Budtz-Jorgensen et al., 2004a). Neurodevelopmental assessments of the children were conducted at age 7 years (Grandjean et al., 1997).

In assembling the Seychelles Child Development Study sample, investigators obtained hair samples from 779 pregnant women and ultimately enrolled a study sample consisting of 740 newborns. The mean maternal hair mercury level was 6.8 ppm (range 0.9-25.8 ppm) (Davidson et al., 1998). Neurodevelopmental assessments were conducted when the children were 6.5, 19, 29, and 66 months, and at 9 years. The mean maternal hair mercury level for the 643 children who participated in the assessment at age 9 years was 6.9 ppm (standard deviation 4.5 ppm) (Myers et al., 2003).

5.7.3 Statistical Analysis

Previous statistical analysis conducted by Ryan (2005) produced a dose-response relationship, integrating data from all three studies, with a central estimate of an IQ change of -0.13 IQ points (95% confidence interval -0.28, -0.03) for every ppm of mercury in maternal hair. Axelrad et al. (2007) conducted a more recent statistical analysis integrating data from the Faroe

Islands, New Zealand, and Seychelles Islands studies to produce a single estimate of the IQ dose-response relationship, which is used in this RIA. Additional details of the analysis are reported in the Axelrad study and in its Supplemental Material (available at http://www.ehponline.org/docs/2007/9303/suppl.pdf). The information is summarized below.

The Axelrad study used a Bayesian hierarchical statistical model to estimate the integrated dose-response coefficient. This is similar to the approach used by the NRC panel to calculate a benchmark dose value integrating data from all three studies (NRC, 2000). The model makes use of dose-response coefficients for IQ, and also considered all other cognitive endpoints reported in the three studies in an effort to obtain more robust estimates of the IQ relationship that account for within-study (endpoint-to-endpoint) variability as well as variability across studies.

The Axelrad study assumed a linear relationship between mercury body burdens and neurodevelopmental outcomes, in keeping with the recommendation of the NRC committee (NRC, 2000). In the New Zealand and Seychelles Islands studies, all information necessary for the model was obtained from the published papers, including linear regression coefficients (Crump et al., 1998; Myers et al., 2003). The Faroe Islands publications, however, reported results with cord blood and maternal hair mercury transformed to the log scale and provided no results of linear models (Grandjean et al., 1997, 1999). A report by the Faroe Islands investigators (Budtz-Jorgensen et al., 2005) provided the additional details needed for the analysis.

The Wechsler Intelligence Scales for Children (WISC) is a standard test of childhood IQ that was used in each of the three studies. The version of the test administered in the Seychelles Islands (3rd ed.; WISC-III) was different from the earlier version used in New Zealand and the Faroe Islands (revised ed.; WISC-R). In a sample of approximately 200 children, the correlation between the Full-Scale IQ scores for the two versions was 0.89; thus the WISC-R and WISC-III appear to measure the same constructs and generate scores with similar dispersion (Wechsler, 1991).

The WISC-R includes 10 core subtests and three supplementary subtests. For the Faroe Islands study, the investigators administered only three subtests of the WISC-R: Digit Span and Similarities (core subtests) and Block Design (a supplementary subtest). The Axelrad study used data for these three subtests to estimate an IQ–mercury coefficient for the Faroe Islands cohort. The Faroe Islands investigators fit data for these three subtests in a structural equation model (SEM) to estimate a standardized coefficient for a hypothetical Full-Scale IQ (Budtz-Jorgensen

et al., 2005). In the SEM analysis of IQ, the three WISC-R subtests are viewed as representative of an underlying latent IQ variable.

To estimate the association between mercury and IQ using information from the three studies, the Axelrad study used a hierarchical random-effects model that includes study-to-study as well as endpoint–to–endpoint variability. Axelrad et al. (2007) implemented the model with a Bayesian approach, using WinBUGS version 1.4 (http://www.mrc-bsu.cam.ac.uk/bugs/). Although the Axelrad study's Bayesian analysis yields highest posterior density (HPD) intervals, the authors refer to these as confidence intervals to aid in the interpretation of results (Axelrad et al., 2007).

The integrated analysis produced a central estimate of -0.18 (95% CI, -0.378 to -0.009) IQ points for each part per million maternal hair mercury, similar to the results found for both the Faroe Islands and Seychelles studies, and lower than the estimate found in the New Zealand study. This central estimate was used as the basis for estimating IQ loss associated with prenatal MeHg exposure in this assessment.

5.7.4 Strengths and Limitations of the IQ Dose-Response Analysis

The Axelrad study produced an estimate of the relationship between maternal mercury body burdens during pregnancy and childhood IQs that incorporates data from all three epidemiologic studies judged by the NRC to be of high quality and suitable for risk assessment. The statistical approach makes use of all the available data (including information on results for related tests of cognitive function), and can be used to produce population-based estimates of a health outcome that can be readily monetized for use in benefit-cost analysis.[1]

There are several aspects of IQ as a metric for neurodevelopmental effects in this benefit-cost analysis that are important to recognize. Full-Scale IQ is a composite index that averages a child's performance across many functional domains, providing a good overall picture of cognitive health. An extensive body of data documents the predictive validity of full-scale IQ, as measured at school age, and late outcomes such as academic and occupational success (Neisser et al., 1996). In addition, methods are readily available for valuing shifts in IQ and thus conducting a benefits analysis of interventions that shift the IQ distribution in a population.

[1] There is limited evidence directly linking IQ and methylmercury exposure in the three large epidemiological studies that were evaluated by the NAS and EPA. Based on its evaluation of the three studies, EPA believes that children who are prenatally exposed to low concentrations of methylmercury may be at increased risk of poor performance on neurobehavioral tests, such as those measuring attention, fine motor function, language skills, visual-spatial abilities (like drawing), and verbal memory. For this analysis, EPA is adopting IQ as a surrogate for the neurobehavioral endpoints that NAS and EPA relied upon for the RfD.

Methods for monetization of the other tests administered in the three studies have not been developed.

It is important to recognize, however, that full-scale IQ might not be the cognitive endpoint that is most sensitive to prenatal mercury exposure. Significant inverse associations were found, in both the New Zealand and Faroe Islands studies, between prenatal mercury levels and neurobehavioral endpoints other than IQ. If the effects of mercury are highly focal, affecting only specific cognitive functions, taking full-scale IQ as the primary endpoint for a benefits analysis might underestimate the impacts. In averaging performance over diverse functions in order to compute full-scale IQ, the specific effects of mercury on only certain of these functions would be "diluted," and the estimated magnitude of the change in performance per unit change in the mercury biomarker would be underestimated.

Moreover, it is well known that there may be substantial deficits in cognitive wellbeing even in individuals with normal or above average IQ. The criterion most frequently used to identify children with learning disabilities for the purposes of assignment to special education services is a discrepancy between IQ and achievement. Specifically, the child's achievement in reading, math, or other academic areas is significantly lower than what would be expected, given his or her full-scale IQ. Thus, there are deficits in cognitive functioning that are not captured by IQ scores. For example, two of the most sensitive endpoints in the Faroe Islands study were the Boston Naming Test, which assesses word retrieval, and the California Verbal Learning Test-Children, which assesses the acquisition and retention of information presented verbally. Depending on the severity of the deficits, a child who has deficits in either of these skills could be at a considerable disadvantage in the classroom setting and at substantial educational risk. Neither of these abilities is directly assessed by the WISC-R or WISC-III, however, and so do not explicitly contribute to a child's IQ score. Therefore, benefits calculations relying solely on IQ decrements are likely to underestimate the benefits to cognitive functioning of reduced mercury exposures. In additions, impacts on other neurological domains (such as motor skills and attention/behavior) are not represented by IQ scores and thus are also excluded from the benefits analysis.

As discussed above, the Faroe Islands study did not include testing for full-scale IQ. For the Axelrad study, an estimate of a dose-response coefficient for full-scale IQ was estimated using the three subtests. While this extrapolation introduces some uncertainty, information has been presented that demonstrates a high correlation between the subtests and full-scale IQ scores.

While the Seychelles and New Zealand studies use maternal hair mercury as the exposure biomarker, the Faroe Islands study uses cord blood mercury. For purposes of the integrated analysis, it was necessary to express results from all three studies in the same terms. Several studies have examined the relationship between hair mercury and blood mercury, and have reported hair:blood ratios typically in the range of 200 to 300 (see ATSDR 1999, pages 249-252 for a review). However, these studies generally do not use cord blood mercury, which is the exposure metric in the Faroe Islands study. One analysis found that mercury concentrations in cord blood are, on average, 70 percent higher than those in maternal blood (Stern and Smith, 2003). For conversion of Faroe Islands data from cord blood mercury to maternal hair mercury, the Axelrad study used data specific to this population, indicating a median maternal hair:cord blood mercury ratio of 200 (Budtz-Jorgensen et al., 2004a).

One uncertainty concerning the New Zealand study is the strong influence of one child in the study population with a particularly high maternal hair mercury level. Published analyses of the New Zealand study presented results with data for this child both included and excluded (Crump et al., 1998). In keeping with the conclusions of the NRC (2000), the integrated dose-response analysis in the Axelrad study made use of the dose-response coefficients calculated with this child omitted. A sensitivity analysis using the New Zealand coefficient with this child included results in an integrated dose-response coefficient that is reduced in magnitude by 25 percent (-0.125 versus a primary central estimate of -0.18).

Some uncertainty is also associated with the Seychelles study due to the exclusion of some members of the cohort from the data reported by Myers et al. (2003) and used as input to this integrated dose-response analysis. The Seychelles researchers did not include a small number of outliers (defined as observations with model residuals exceeding 3 standard deviation units), and no results are available for the full cohort. However, the authors report that "In all cases, the association between prenatal MeHg exposure and the endpoint was the same, irrespective of whether outliers were included" (Myers et al., 2003).

Finally, the integrated dose-response analysis assumes the exposures assigned to each study subject are accurate representations of true exposure. In reality, there is likely to be some discrepancy between measured and actual exposures, for example, due to variation in hair length. Alternatively, the true exposure of interest may have been during the first trimester of pregnancy, whereas exposures in maternal hair and cord blood measured at birth reflect exposures later in pregnancy. Presence of exposure measurement error could introduce a bias in the results, most likely towards the null (Budtz-Jorgensen et al., 2004b).

5.7.5 Possible Confounding from Long-Chained Polyunsaturated Fatty Acids

Maternal consumption of fish during pregnancy exposes the fetus to long-chain polyunsaturated fatty acids (LCPUFAs), believed to be beneficial for fetal brain development, and to the neurotoxicant MeHg (Helland et al., 2003; Daniels et al., 2004; Dunstan et al., 2006; Judge et al., 2007). Reports from the Seychelles Islands study cohort have suggested a negative impact of MeHg exposure, accompanied by a simultaneous beneficial effect of omega-3 LCPUFAs on children's development (Davidson et al., 2008; Strain et al., 2008). It is unclear whether this result was evidence for independent influences of MeHg and LCPUFAs or effect modification. A recent study by Lynch et al. (2010) used varying coefficient models to characterize the interaction of mercury and nutritional covariates (Hastie and Tibshirani, 1993), including omega-3 LCPUFAs, using data from the Seychelles Islands study.

The Seychelles Islands study cohort of mother-child pairs had fish consumption averaging 9 meals per week. Lynch et al., (2010) assessed maternal nutritional status for five different nutritional covariates known to be present in fish (n-3 LCPUFA, n-6 LCPUFA, iron status, iodine status, and choline) and associated with children's neurological development. The study also included prenatal MeHg exposure (measured in maternal hair).

Lynch et al., (2010) examined two child neurodevelopmental outcomes (Bayley Scales Infant Development-II (BSID-II) Mental Developmental Index (MDI) and Psychomotor Developmental Index (PDI)), each administered at 9 and at 30 months. The varying coefficient models allowed the possible interactions between each nutritional component and MeHg to be modeled as a smoothly varying function of MeHg as an effect modifier. Iron, iodine, choline, and omega-6 LCPUFAs had little or no observable modulation at different MeHg exposures. In contrast the omega-3 LCPUFA docosahexaenoic acid had beneficial effects on the BSID-II PDI that were reduced or absent at higher MeHg exposures. The results from Lynch et al. (2010) suggest a potentially useful modeling method that could shed further light on the issue of interactions between nutritional covariates.

A recent study by Rice et al. (2010) considered possible confounding in a probabilistic assessment of the health benefits of reducing MeHg exposure in the United States. In deciding on a dose-response relationship between MeHg exposure and effects on IQ loss, the authors chose to use the central estimate from the Axelrad study, noting however that Axelrad et al. (2007) did not explicitly consider possible confounding of the MeHg-IQ relationship by the concurrent consumption of LCPUFAs that might enhance cognitive development and bias downward the observed regression coefficient estimates from the Faroe Islands, New Zealand, and Seychelles

Islands studies. Rice et al. (2010) therefore multiplied the central estimate from Axelrad et al. (2010) by an adjustment factor to offset the possible downward bias from inadequate confounder control. A factor of 1.5 was selected "to acknowledge the recent argument of Budtz-Jorgensen et al. (2007) that the parameter estimates from the three epidemiological studies may be biased downward by a factor of approximately 2 because of failure to adequately control for confounding" (Rice et al., 2010).

There remains uncertainty with respect to the nature and magnitude of potential confounding between LCPUFAs and MeHg, and the associated effects on childhood neurodevelopment due to maternal ingestion during pregnancy. Additional research is needed to provide further clarity on this issue, but recent studies such as those referenced above reinforce the view that fish consumption during pregnancy should be approached as a case of multiple exposures to nutrients and to MeHg, with a complex and potentially interactive set of risks and benefits related to infant development. Due to the remaining uncertainty regarding the potential confounding between LCPUFAs and MeHg exposure, we have not incorporated any factors or other quantitative adjustments into this assessment.

5.7.6 References

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5.8 Mercury Benefits Analysis Modeling Methodology

5.8.1 Introduction

This section describes the methodology used to model fishing behavior and associated MeHg exposure levels. The methodology incorporates data, assumptions, and analytical techniques already described in previous sections. Sections 5.7.2 and 5.7.3 below describe elements of the methodology applied to develop a national-scale estimate of benefits associated with avoided IQ loss among freshwater recreational anglers. Section 5.7.4 describes a variation

of the methodology used to estimate risk levels (as measured by IQ loss) among modeled high-risk subpopulations.

5.8.2 Estimation of Exposed Populations and Fishing Behaviors

This section describes the methodology used to estimate the average daily ingestion of mercury (g/day) through noncommercial freshwater fish consumption (HgI) for selected populations of interest. Because the primary measurable health effect of concern—developmental neurological abnormalities in children—occurs as a result of in-utero exposures to mercury, the specific population of interest in this case is prenatally exposed children. To identify and estimate the size of this exposed population, the benefits analysis focuses on pregnant women in freshwater recreational angler households.

Generally speaking, estimating mercury exposures for this exposure pathway and population of interest requires three main components:

N_i = size of the exposed population of interest i (annual number of pregnant women in freshwater angler households during the year),

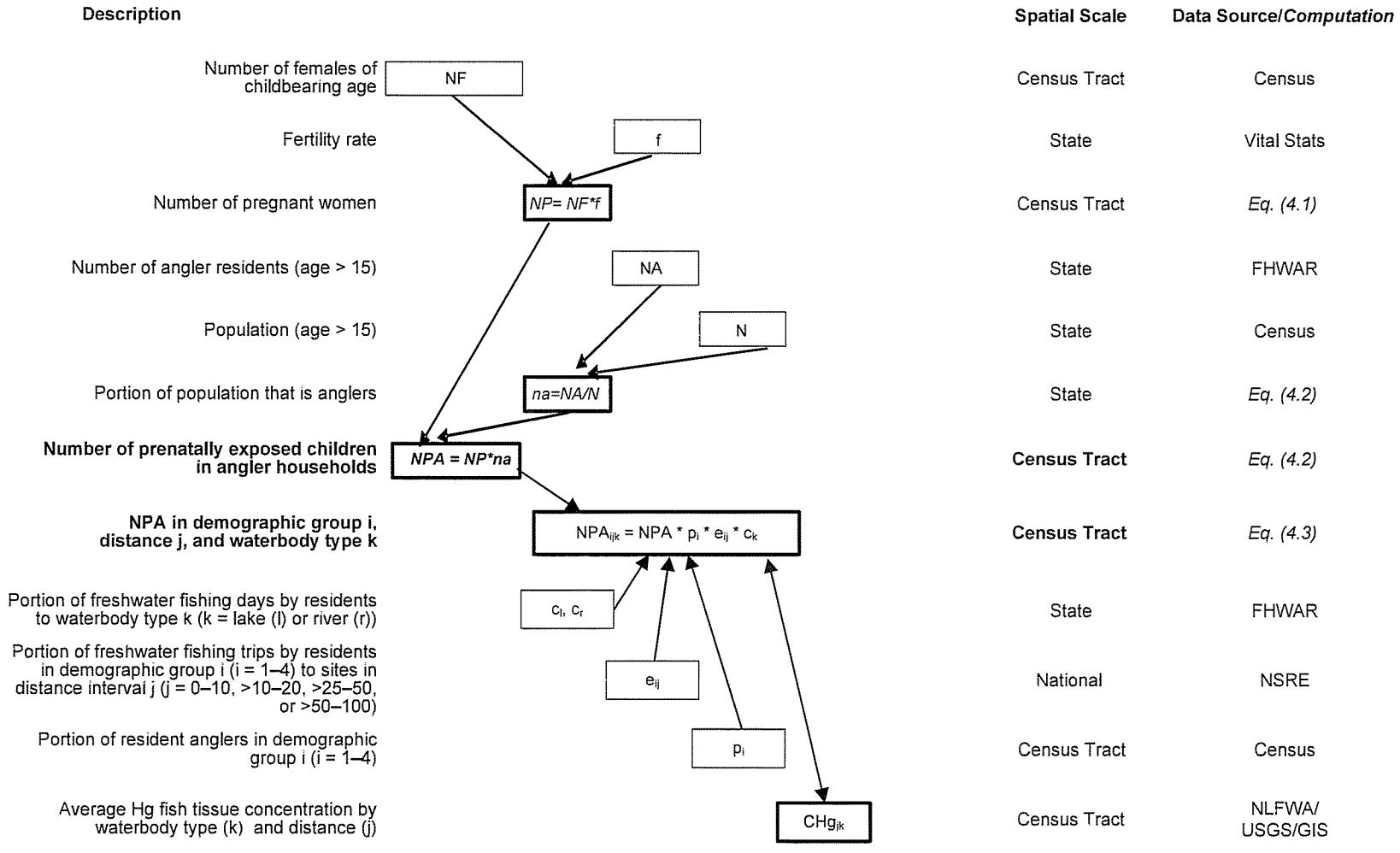
CHg_i = average concentration (ppm) of methyl mercury in noncommercial freshwater fish filets consumed by population i, and

C_i = average daily consumption rate (gm/day) of noncommercial freshwater fish by population i.

The flow diagram in Figure 5-4 illustrates the approach used to estimate the first two components of this equation—N_i and CHg_i. It shows the spatial scale of the data used to estimate these components and describes how these components are interrelated. For the third component—C_i—recommendations from EPA's *Environmental Exposure Factors Handbook* (EPA, 1997) were used to estimate an average consumption rate estimate for recreationally caught freshwater fish.

First, 2000 Census data (U.S. Census Bureau, Census 2000 Summary File 3, Detailed Tables, United States) were used to define the size, age, gender distribution, and income of the populations within each census tract in the 48 contiguous U.S. states.

Figure 5-4. Methodology for Estimating and Linking Exposed Populations and Levels of Mercury Exposure



1. Estimating the number of pregnant women (NP) living in the census tract as

$$NP = NF * f_s, \quad (5.1)$$

where

NF = number of females aged 15 to 44 in the tract (Census 2000) and

f_s = state-level general fertility rate (average number of live births in a year per 1,000 women aged 15 to 44) (2006 Vital Statistics).

2. Estimating the annual number of prenatally exposed children in angler households (NPA) as

$$NPA = NP*(NA_s/N_s), \quad (5.2)$$

where

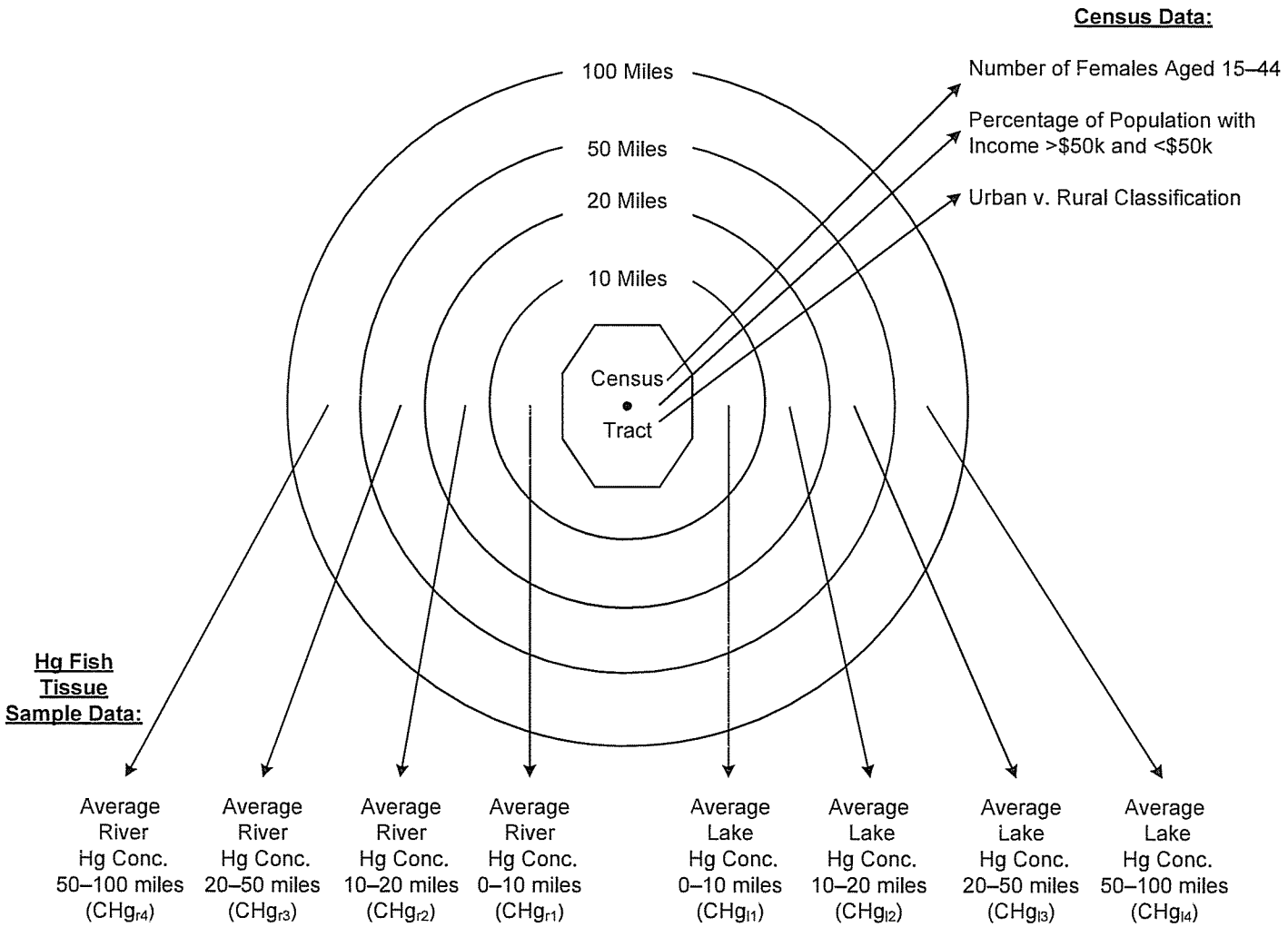
NA_s = state-level number of angler residents (FHWAR) and

N_s = adult population of state s (Census).

Using Eq. (5.2) to estimate NPA implies that (1) the fraction of pregnant women in a state who are in freshwater angler households is equal to the fraction of households in the state that include freshwater anglers (i.e., pregnant women are no more or less likely than the rest of the state population to live in households with freshwater anglers) and (2) the fraction of households in the state that includes freshwater anglers is equal to the fraction of adult residents in the state who are freshwater anglers.

To estimate NPA for years after 2000, it was assumed that state-level fertility rates (f_s) and angler participation rates (NA_s/N_s) would remain constant; however, the number of women of childbearing age in each block (NF) was increased based on county-level population growth projections (Woods and Poole, 2008). In other words, for the period 2000 to 2016, the estimated NPA for each census tract was assumed to increase at the same rate as the projected annual population growth rates for females 15 to 44 in their corresponding counties.

Figure 5-5. Linking Census Tracts to Demographic Data and Mercury Fish Tissue Samples



Fourth, to match exposed populations in each tract with mercury concentrations, we first divided the exposed population into four distinct demographic groups (i = 1 – 4): urban/low income, urban/high income, nonurban/low income, and nonurban/high income. To estimate the portion of households in each demographic group (p_i for i = 1 – 4), tract-level Census data were used to specify (1) the percentage of the population in each tract that resides in an urban area and (2) the percentage with household income less than $50,000 (i.e., the portion in the low-income group).

In addition, it was assumed that

1. each exposed individual in a census tract is associated with freshwater fishing in a single distance interval and a single waterbody type (i.e., all the fish they consume comes from the same distance and type of waterbody) [1], and

2. the exposed populations in each census tract (rather than just the fishing trips) are distributed across the distance intervals and waterbody types according to the estimated proportions (i.e., parameters c, e, and p shown in Figure 5-4).

More specifically, a maximum of 32 separate exposed subpopulations were defined for each census tract:

$$NPA_{ijk} = NPA * p_i * e_{ij} * c_k \text{ (for all i, j, and k)} \quad (5.3)$$

for

i = 1 – 4 demographic subgroup in the census tract,

j = 1 – 4 distance interval, and

k = lake or river.

(See Figure 5-4 for definitions of p_i, e_{ij}, and c_k).

Using this approach, we were able to separately match each subpopulation NPA_{ijk} with the census tract's average mercury concentration for the corresponding distance and waterbody category (CHg_{jk}).

[1] An alternative would be to assume that all anglers in the census tract have the same distribution of trips across distance intervals and water types. This assumption would imply no variation in per-capita mercury exposures within a census tract, but it would not affect the estimates of *total* exposure and *total* IQ losses in the tract.

To approximate the percentage freshwater fishing trips (and exposed individuals) from each census tract matched to each waterbody type (c_l or c_r), we used state-level averages. These averages were calculated for each state, based on the portion of residents' freshwater fishing trips that are to each waterbody type, based on 2001 FHWAR data.

Data from NSRE 1994 were used to approximate the percentage of freshwater fishing trips (and exposed individuals) matched to different distances from anglers' residential location. Four distance intervals were defined as 0–10 miles, >10–20 miles, >20–50 miles, and >50–100 miles. Based on self-reported trip distance information from nearly 2,000 respondents (see Appendix B for details), each of these distance categories was associated with roughly 20% of the reported trips in the NSRE sample. Four distinct demographic groups were also found to have significantly different average travel distances for freshwater fishing in the NSRE sample: high-income urban, high-income rural, low-income urban, and low-income rural. An annual household income threshold of $50,000 (in 2000 dollars) was used to define high and low income, because it is close to the median value for both the NSRE sample and the U.S. population. The portion of trips for each demographic group (i = 1 – 4) to each distance interval (j = 1 – 4) is defined as e_{ij}. The estimated values for e_{ij} are reported in Appendix B.

To estimate average daily mercury ingestion rates for each exposed subpopulation n=ijk, we applied the following equation:

$$HgI_n = CHgFC_n * C_n = (CHg_n * CCF) * C_n \quad (5.4)$$

where

HgI = average daily mercury ingestion rate (μg/day);

CHg = average mercury concentration in uncooked freshwater fish (ppm);

CCF = cooking conversion factor: ratio of mercury concentration in cooked fish to mercury concentration in uncooked fish (= 1.5);

CHgFC = average mercury concentration in cooked freshwater fish (ppm); and

C = average daily self-caught freshwater cooked fish consumption rate (gm/day) = 8 gm/day.

To determine an appropriate daily fish consumption rate (C) for the analysis, EPA conducted an extensive review of existing literature characterizing self-caught freshwater fish

consumption. Based on this review, it was decided that the ingestion rates for recreational freshwater fishers, specified as "recommended" in EPA's *Environmental Exposure Factors Handbook* (EPA, 1997) (mean of 8 gm/day and 95th percentile of 25 gm/day), represented the most appropriate values to use in this analysis. These recommended values were derived based on ingestion rates from four studies conducted in Maine, Michigan, and Lake Ontario (Ebert et al., 1992; Connelly et al., 1996; West et al., 1989; West et al., 1993), which measured annual average daily intake rates for self-caught freshwater fish by all recreational fishers including consumers and non-consumers of fish. The mean values presented in these four studies ranged from 5 to 17 gm/day, while the 95th percent values ranged from 13 to 39 gm/day (Note: the 39 gm/day value actually represents a 96th percent value). The EPA "recommended values" were developed by considering the range and spread of means and 95th percentile values presented in the four studies. EPA recognizes that using mean and 95th percentile consumption rates based on these four studies may not be representative of fishing behavior across the entire 48-state study area and that regional trends in consumption may differ from the values used in this analysis. Moreover, rates of consumption by pregnant women in freshwater angler households may be different from those of the recreational fishers themselves. However, EPA believes that these four studies do represent the best available data for developing recreational fisher ingestion rates in the United States.

Because the consumption rate estimate C is for cooked fish and the mercury concentrations are estimated for uncooked filet, a conversion factor (CCF) was applied to estimate mercury concentrations in cooked fish. Cooking fish tends to reduce the overall weight of fish by approximately one-third (Great Lakes Sport Fish Advisory Task Force, 1993). Because volatilization of mercury is unlikely to occur during cooking, the overall amount of mercury will stay unchanged during cooking, and the concentration of mercury will increase by a factor of roughly 1.5 (Morgan, Berry, and Graves, 1997).

5.8.3 Estimation of Lost Future Earnings

Estimating the IQ decrements in children that result from mothers' ingestion of mercury required two steps. First, based on the estimated average daily maternal ingestion rate, the expected mercury concentration in the hair of exposed pregnant women was estimated as follows:

$$CHgH_n = (0.08)^{-1} * (HgI_n/W), \quad (5.5)$$

where

CHgH = average mercury concentration in maternal hair (ppm) and

W = average body weight for female adults below age 45 (= 64 kg).

This conversion rate between average daily ingestion rate and maternal hair concentration is based on the one compartment model developed by Swartout and Rice (2000). The 2002 EPA Workshop on Methylmercury Neurotoxicity recommended that this one compartment model might be better suited than the PBPK model in modeling dose-response (EPA, 2002). The average body weight estimate (W) was based on EPA's Exposure Factor Handbook (EPA, 1997).

Second, to estimate the expected IQ decrement in offspring resulting from in-utero exposure to mercury through mothers' fish consumption, the following dose-response relationship was applied:

$$dIQ_n = 0.18 * CHgH_n, \quad (5.6)$$

where

dIQ = IQ decrement in exposed mother/child (IQ pts).

The 0.18 dose-response coefficient in this equation is based on the summary findings reported in Axelrad et al. (2007).

The valuation approach used to assess monetary losses due to IQ decrements is based on an approach applied in previous EPA analyses (EPA, 2008). The approach expresses the loss to an affected individual resulting from IQ decrements in terms of foregone future earnings (net of changes in education costs) for that individual. These losses were estimated using the following equation:

$$V_n = VIQ * dIQ_i, \quad (5.7)$$

where

V = present value of net loss per exposed mother/child (2006 dollars) and

VIQ = net loss per change in IQ point.

The net loss per IQ point decrement is estimated based on the following relationship:

$$VIQ = (z * PVY) - (s * PVS), \quad (5.8)$$

where

PVY = median present value of lifetime earnings,

PVS = present value of education costs per additional year of schooling,

z = percentage change in PVY per 1-point change in IQ, and

s = years of additional schooling per 1-point increase in IQ.

The estimate for PVY is derived using earnings and labor force participation rate data from the 2006 Current Population Survey (CPS) and assuming (1) an individual born today would begin working at age 16 and retire at age 67; (2) the growth rate of wages is 1% per year, adjusted for survival probabilities and labor force participation by age; and (3) lifetime earnings are discounted back to the year of birth. Using a 3% discount rate, the resulting present value of median lifetime earnings is $555,427 in 2006 dollars.

Estimates of the average effect of a 1-point increase in IQ on lifetime earnings (z) range from a 1.76% increase (Schwartz, 1994) to a 2.379% increase (Salkever, 1995). The percentage increases in the two studies reflect both the direct impact of IQ on hourly wages and indirect effects on annual earnings as the result of additional schooling and increased labor force participation. The estimate for *s* is based on Schwartz (1994) who reports an increase of 0.131 years of schooling per IQ point.

In addition to this positive net effect on earnings, an increase in IQ is also assumed to have a positive effect on the amount of time spent in school (s) and on associated costs (PVS). The range of estimate for *s* is based on Schwartz (1994) who reports an increase of 0.131 years of schooling per IQ point and Salkever (1995) who reports an increase of 0.1007 years.

The estimate for PVS is derived using an estimate of $16,425 per additional year of schooling in 1992 dollars (EPA, 2005), which is based on U.S. Department of Education data reflecting both direct annual expenditures per student and annual average opportunity cost (i.e., lost income from being in school). We assume these costs are incurred when an individual born today turns 19, based on an average 12.9 years of education among people aged 25 and over in the United

States. Discounting at a 3% rate to the year of birth results in an estimate of $13,453 per additional year of schooling in 2006 dollars.

To incorporate (1) uncertainty regarding the size of z and (2) different assumptions regarding the discount rate, the resulting value estimates for the average net loss per IQ point decrement (VIQ) are expressed as a range. Assuming a 3% discount rate, VIQ ranges from $8,013 (using the Schwartz estimate for z and s) to $11,859 (using the Salkever estimates). With a 7% discount rate assumption, the VIQ estimates range from $893 to $1,958.

5.8.4 Analysis of Potentially High-Risk Subpopulations

The methodology described above is designed to evaluate the aggregate effect of fish tissue mercury concentrations and, correspondingly, the aggregate benefits of reduced concentrations due to proposed emission controls. However, this approach does not provide specific insight into the effects for subpopulations that may be at particularly high risk from mercury exposures because of freshwater fish consumption. In particular, the aggregate analysis applies a uniform average fish consumption rate (C) for the entire exposed population. Although appropriate for an aggregate analysis, this single average rate obscures the large variation in consumption rates that have been observed in studies of specific subpopulations.

To assess effects on potentially high-risk populations, we modified the methodology described above in Section 5.7.2 and focused on six subpopulations for which more specific freshwater fish consumption rate estimates are available:

- low-income African-American recreational/subsistence fishers in the Southeast region[1]
- low-income white recreational/subsistence fishers in the Southeast region
- low-income female recreational/subsistence fishers
- Hispanic subsistence fishers
- Laotian subsistence fishers
- Chippewa/Ojibwe Tribe members in the Great Lakes area

[1] The low-income designation is based on Census 2000 estimates of populations living in poverty. The Southeast for purposes of this analysis comprises Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, and West Virginia.

These specific subpopulations were selected based on published empirical evidence of particularly high self-caught freshwater fish consumption rates among these groups. Evidence for the first three groups is based on a study by Burger (2002), which collected survey data from a random sample of participants in the Palmetto Sportsmen's Classic in Columbia, SC. Out of 458 respondents, 39 were black, 415 were white, and 149 were female. The sample size for the black population is relatively small, which increases uncertainty, particularly in higher percentile consumption rate values provided for this group. In this study, results are also split out for poor respondents (0-20K$ annual income). These consumption rates are relatively high, particularly for the higher percentiles. This observation forms the basis for our decision to assess a number of the subsistence populations only for watersheds located in US Census tracts containing members of source populations below the poverty line for the white and black populations. The black and white fisher populations were extrapolated to cover all watersheds modeled for risk in the Southeastern states. The rationale for this was that fishing activity by these two groups could be generalized in this region of the country. Note, however that these scenarios were only assessed for watersheds in the Southeast located within US Census tracts with at least 25 individuals from that ethnic group below the poverty line. Given the focus of the risk assessment on consumption by women (in considering risk to pregnant women in particular), we extrapolated the female consumption rates to all watersheds in the continental US with at least 25 individuals below the poverty line.

Evidence for the Hispanic and Laotian groups are based on a study by Shilling et al. (2010). This study looks at subsistence fishing activity among ethnic groups associated with more urbanized areas near the Sacramento and San Joaquin rivers in the Central Valley in CA. The authors note that many of these ethnic groups relied on fishing in origin countries and bring that practice here (e.g., Cambodian, Vietnamese and Mexican). The authors also note that fish consumption rates reported here for specific ethnic groups (specifically Southeast Asian) are generally in-line with rates seen in WA and OR studies. The fish consumption rates for Hispanics and Laotians were extrapolated to cover US Census tracts with at least 25 poor members of the ethnic populations.

For the Chippewa population, we use results from a study by Dellinger (2004), which gathered data on self-reported fish consumption rates by Tribes in the Great Lakes area. Because fishing activity is highly variable across Tribes (and closely associated with heritage cultural practices) we have not extrapolated fishing behavior outside of the areas ceded to the Tribes covered in the study (regions in the vicinity of the Great Lakes). The terms "subsistence" and "recreational" fishing are based on the terminology used in these published studies to describe

the population of interest. In general, subsistence fishers are individuals whose primary objective in fishing is to acquire food for household consumption. For recreational fishers, the primary objective is to enjoy the outdoor activity; however, fish consumption is also often an objective.

To assess the distribution of individual risks from mercury exposure in these specific subpopulations, we modified the methodology in the following ways:

1. We limited the analysis to only include census tracts with at least 25 residents (100 residents for the Chippewa group) in the defined demographic group (based on the 2000 Census). Tracts with fewer individuals were assumed to be less representative of the location and conditions of the subpopulation of interest.

2. Rather than using four distance intervals around each census tract, we limited the analysis to one distance interval (0 to 20 miles). This interval was selected to better reflect the likely shorter distances traveled by low-income and/or subsistence fishers who fish with relatively high frequency. As a result, the populations were not subdivided according to income or urban classification; however, the separation between river and lake anglers was preserved using the same methodology described above to define c_r and c_l.

3. Due to data limitations, rather than specifically selecting and estimating populations of pregnant women in angler households for the high-risk demographic subgroup of interest, we used the entire population of the defined demographic subgroup in each selected census tract. This approach was used because *the purpose of this part of the analysis is not to estimate the total size of the exposed population or the aggregate impacts on this demographic subgroup. Instead, the objective is to examine the potential distribution of risks within the group.* Using the entire subgroup population to represent the risk distribution in the exposed population relies on the key assumption that the spatial distribution of the entire subgroup provides a reasonable approximation for the distribution of pregnant women in high-consuming angler households. In other words, it was assumed that the expected proportion of the subgroup's population in each Census tract that consists of pregnant women in fishing households is the same across the selected census tracts. The main limitation of this assumption is that it does not allow or account for spatial variation in freshwater angler participation rates for the subgroups of interest. Unfortunately, data to address these limitations are not readily available.

4. Population size projections for future years (beyond 2000) in each selected tract were based on county-level growth projections for the full population (all ages and both sexes) in the most closely corresponding race category (Woods and Poole, 2008). For example, the Asian and Native American categories were used for the Laotian and Chippewa population projections, respectively.

5. Rather than assuming a single fish consumption rate (C) for all exposed individuals, the analysis assumed and applied a different *distribution* of consumption rates for each subgroup, based on evidence from existing empirical studies (see Table 5-3). Using the consumption rate information reported in Table 5-3, we fit a separate log-normal distribution of consumption rates for each of the six subpopulations. We then applied Equation (5.2) to estimate a specific mercury ingestion rate (HgI) *for each subpopulation member* in the selected census tracts. To specify the average fish tissue mercury concentration (CHg), residents were divided into river and lake fishers according to the state-level percentages (as described in Section 5.7.2) and assigned the corresponding average mercury concentration within the 20-mile interval from the tract centroid. To specify the consumption rate (C) for each individual, we randomly drew a separate value from the specific log-normal consumption rate distribution developed for the subpopulation. Equations (5.3) to (5.8) were then used to estimate the corresponding IQ loss for each individual.

Table 5-3. Reported Distributions of Self-Caught Freshwater Fish Consumption Rates among Selected Potentially High-Risk Subpopulations

Population	Self-Caught Freshwater Fish Consumption Rate (g/day)			Study
	Sample Size	Mean (Median)	90th (95th) Percentile	
Low-income African-American recreational/subsistence fishers in Southeast	39	171(137)	446 (557)	Burger (2002)
Low-income white recreational/subsistence fishers in Southeast	415	38.8 (15.3)	93 (129)	Burger (2002)
Low-income female recreational/subsistence fishers	149	39.1 (11.6)	123 (173)	Burger (2002)
Hispanic subsistence fishers	45	25.8 (19.1)	98[a] (155.9)	Shilling et al. (2010)
Laotian subsistence fishers	54	47.2 (17)	144.8[a] (265.8)	Shilling et al. (2010)
Great Lakes tribal groups	822	60 (113[b])	136.2[a] (213.1)[a]	Dellinger (2004)

[a] Derived values using a log-normal distribution, based on the median and the 95th percentile or standard deviation reported in study.

[b] Standard deviation in parentheses, rather than median.

5.8.5 References

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5.9 Mercury Benefits and Risk Analysis Results

5.9.1 Baseline Incidence

Applying the methodology described in Section 5.7, we first used GIS to link census tract centroids in the continental United States with HUC-12 watersheds containing mercury fish tissue sample data for 1995 to 2007. We found that, out of the 64,500 tracts in the 48-state area, almost all of them are located within 100 miles of at least one HUC-12 with freshwater mercury fish tissue sampling data. Therefore, very few tracts were entirely excluded from the analysis due to a lack of sampling data within 100 miles. Table 5-4 reports the number of tracts linked to HUC-level river or lake mercury concentration estimates within each distance interval. As expected, this number decreases as the size of the distance interval decreases. For example, 33% are within 10 miles of a HUC-12 containing a lake sample, and 52% are within 10 miles of a HUC-12 containing a river sample.

Table 5-4 also reports the average river and lake HUC-level fish tissue mercury concentrations found within each distance interval. Assuming that the 1995 to 2007 samples are representative of baseline conditions in 2005, the distance-specific mean lake concentrations range from 0.26 to 0.3 ppm, and the mean river concentrations vary from 0.25 to 0.27 ppm.

Table 5-4 also reports corresponding river and lake mercury concentration estimates for a 2016 base case scenario. This scenario represents total mercury deposition from all global natural and anthropogenic sources based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule). As described in Section 5.4, CMAQ air quality modeling runs were used to estimate average mercury deposition levels by HUC-12 sub-watershed under both the 2005 base case and the 2016 base case scenarios. For this analysis, it is assumed that HUC-level fish tissue mercury concentrations would change (between the two scenarios) by the same percentage as the change in modeled deposition levels. Overall, the mean concentrations decline by 6% to 9% in the 2005 base case compared with the 2016 base case scenarios.

With these tract-level mercury concentration estimates, we then estimated the size of the exposed populations (NPA) in 2005 and 2016. These estimates are reported in Table 5-5. As described in Section 5.7.2, a separate exposed population (NPA_{jk}) was estimated for each distance interval (j = 1 – 4) and waterbody (k = lake or river) combination at each tract. If mercury concentration data were not available for a specific distance-waterbody combination, then the corresponding exposed population for the tract (NPA_{jk}) was not included in the analysis. Consequently, the exposed population estimates reported in Table 5-5 are best interpreted as lower-bound estimates of the total exposed population. Excluding potentially exposed

populations from the analysis because of missing/unavailable mercury concentration data reduced the total exposed population estimate by roughly 44%. These excluded populations include the portions of the tract-level exposed populations that were matched with fishing trip travel distances that either (1) did not overlap with at least one HUC-12 with sampling data or (2) were greater than 100 miles (see Appendix B). For 2005, there were estimated to be 239,174 prenatally exposed children, and for 2016 the estimate is 244,286 prenatally exposed children.

Table 5-4. Summary of Baseline Mercury Fish Tissue Concentrations

Distance from Tract Centroid	N[a]	2005 Base Case				2016 Base Case			
		Min (ppm)	Mean (ppm)	Max (ppm)	Median (ppm)	Min (ppm)	Mean (ppm)	Max (ppm)	Median (ppm)
Lake Sampling Sites									
0–10 miles	20,998	0.000	0.297	3.561	0.198	0.000	0.276	3.420	0.178
>10–20 miles	35,149	0.000	0.285	3.561	0.209	0.000	0.264	3.420	0.187
>20–50 miles	55,885	0.000	0.289	3.561	0.223	0.000	0.270	3.420	0.202
>50–100 miles	61,820	0.000	0.264	2.333	0.241	0.000	0.247	2.251	0.227
River Sampling Sites									
0–10 miles	33,342	0.006	0.246	4.967	0.185	0.005	0.224	4.924	0.168
>10–20 miles	44,493	0.006	0.269	4.967	0.195	0.005	0.247	4.924	0.174
>20–50 miles	54,970	0.019	0.270	4.480	0.203	0.019	0.251	4.441	0.183
>50–100 miles	62,868	0.023	0.267	4.967	0.214	0.022	0.251	4.924	0.192

[a] Number of tracts (out of 64,419) with at least one HUC-12 with sample data in the distance interval.

Table 5-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case: Number of Prenatally Exposed Children (NPA)		2005 Base Case: Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	2005 Base Case: Average IQ Loss per Exposed Child (dIQ)	2005 Base Case: Total IQ Point Losses	2016 Base Case: Number of Prenatally Exposed Children (NPA)		2016 Base Case: Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	2016 Base Case: Average IQ Loss per Exposed Child (dIQ)	2016 Base Case: Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
Total	**63,978**	**3.74**	**239,174**	**3.04**	**0.11**	**25,544.9**	**3.82**	**244,286**	**2.84**	**0.10**	**24,419.4**
AL	1,081	5.51	5,956	3.28	0.12	685.9	5.53	5,981	3.04	0.11	638.3
AR	623	6.45	4,017	3.80	0.13	537.1	6.55	4,084	3.66	0.13	525.9
AZ	1,097	3.17	3,476	2.21	0.08	269.8	3.75	4,117	2.18	0.08	316.3
CA	6,801	1.19	8,089	6.04	0.21	1,716.4	1.26	8,599	5.74	0.20	1,734.0
CO	1,045	3.53	3,693	1.20	0.04	155.3	3.92	4,101	1.18	0.04	169.8
CT	812	2.47	2,003	4.58	0.16	322.2	2.38	1,929	4.29	0.15	291.3
DC	181	2.23	404	1.67	0.06	23.7	2.03	367	1.35	0.05	17.4
DE	196	1.77	348	1.98	0.07	24.2	1.79	352	1.71	0.06	21.2
FL	3,144	3.28	10,299	5.24	0.18	1,897.5	3.71	11,651	5.17	0.18	2,118.9
GA	1,614	8.38	13,525	3.14	0.11	1,494.8	8.74	14,111	2.88	0.10	1,431.0
IA	791	6.39	5,052	1.21	0.04	215.3	6.18	4,888	1.15	0.04	197.5
ID	280	6.30	1,765	2.43	0.09	150.9	7.13	1,996	2.31	0.08	162.3
IL	2,950	2.33	6,884	1.83	0.06	442.3	2.32	6,831	1.49	0.05	356.9
IN	1,409	5.47	7,711	2.20	0.08	596.7	5.51	7,759	1.90	0.07	519.2
KS	716	2.08	1,490	2.38	0.08	124.8	2.06	1,478	2.34	0.08	121.8
KY	993	4.99	4,954	2.19	0.08	381.9	4.92	4,889	1.90	0.07	326.1
LA	1,103	6.91	7,623	3.82	0.13	1,022.9	6.59	7,269	3.77	0.13	962.6
MA	1,357	1.81	2,456	5.40	0.19	466.0	1.74	2,359	5.04	0.18	417.7
MD	1,210	2.23	2,703	2.16	0.08	204.8	2.35	2,840	1.76	0.06	176.2

(continued)

Table 5-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption (continued)

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case					2016 Base Case				
		Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses	Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
ME	344	4.66	1,602	5.12	0.18	288.3	4.31	1,484	5.05	0.18	263.4
MI	2,701	3.89	10,520	2.72	0.10	1,005.0	3.79	10,234	2.37	0.08	854.0
MN	1,294	11.53	14,915	2.86	0.10	1,501.2	11.71	15,157	2.77	0.10	1,474.7
MO	1,311	3.66	4,796	1.80	0.06	302.7	3.75	4,911	1.70	0.06	294.2
MS	604	9.18	5,546	5.11	0.18	996.2	9.32	5,632	4.98	0.18	986.9
MT	267	3.62	965	2.40	0.08	81.5	3.68	984	2.38	0.08	82.3
NC	1,554	5.13	7,976	3.29	0.12	921.5	5.33	8,280	2.95	0.10	859.1
ND	224	2.89	647	3.43	0.12	78.1	2.79	626	3.41	0.12	74.9
NE	500	3.97	1,984	1.60	0.06	111.9	4.03	2,014	1.56	0.05	110.5
NH	272	3.68	1,001	5.53	0.19	194.5	3.71	1,010	5.39	0.19	191.2
NJ	1,930	1.02	1,965	3.28	0.12	226.5	1.00	1,936	2.98	0.10	202.7
NM	244	1.75	426	1.74	0.06	26.0	1.89	461	1.77	0.06	28.6
NV	471	1.70	803	3.78	0.13	106.8	2.09	985	3.60	0.13	124.8
NY	4,791	1.41	6,770	3.86	0.14	918.4	1.35	6,486	3.54	0.12	807.0
OH	2,923	4.11	12,015	1.61	0.06	678.8	3.93	11,489	1.30	0.05	527.0
OK	987	5.65	5,580	3.07	0.11	602.9	5.73	5,653	3.03	0.11	601.4
OR	754	5.14	3,877	2.80	0.10	382.1	5.43	4,095	2.81	0.10	404.3
PA	3,116	2.40	7,485	2.30	0.08	605.9	2.31	7,194	1.91	0.07	482.2
RI	233	1.55	361	6.01	0.21	76.2	1.53	356	5.15	0.18	64.5
SC	864	7.39	6,388	4.43	0.16	995.4	7.59	6,559	4.08	0.14	941.0

(continued)

Table 5-5. Baseline Levels of Mercury Exposure and IQ Impacts Due to Freshwater Self-Caught Fish Consumption (continued)

State	Number of Census Tracts with Hg Samples w/in 100 Miles	2005 Base Case					2016 Base Case				
		Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses	Number of Prenatally Exposed Children (NPA)		Average Maternal Daily Mercury Ingestion (HgI) (µg/day)	Average IQ Loss per Exposed Child (dIQ)	Total IQ Point Losses
		Mean per Tract	Total in State				Mean per Tract	Total in State			
SD	225	3.29	740	1.77	0.06	45.9	3.20	719	1.72	0.06	43.6
TN	1,253	4.95	6,204	3.01	0.11	656.7	5.06	6,335	2.76	0.10	615.5
TX	4,310	3.97	17,127	2.83	0.10	1,701.2	4.32	18,633	2.67	0.09	1,748.9
UT	482	3.95	1,905	2.05	0.07	137.3	4.68	2,254	2.06	0.07	163.5
VA	1,524	3.66	5,580	2.61	0.09	512.7	3.82	5,820	2.19	0.08	448.7
VT	179	3.50	627	3.85	0.14	84.8	3.37	604	3.70	0.13	78.6
WA	1,315	3.67	4,823	1.69	0.06	287.2	3.90	5,133	1.68	0.06	302.8
WI	1,313	8.03	10,543	2.77	0.10	1,026.2	7.85	10,309	2.59	0.09	938.1
WV	466	6.53	3,042	2.10	0.07	224.3	6.10	2,840	1.66	0.06	166.1
WY	124	4.13	512	1.97	0.07	35.5	3.99	495	1.97	0.07	34.3

For each exposed population, we then estimated their average mercury ingestion rate (HgI) using Equation (5.4) and the IQ loss associated with this exposure level. As reported in Table 5-5, in 2005, the average estimated mercury ingestion rate for the population of exposed pregnant women was 3.04 ug/day. For 2016, the ingestion rate was estimated to be 2.84 ug/day (6.6% lower). The corresponding average IQ loss per prenatally exposed child was 0.11 in 2005 and 0.10 in 2016. Multiplying these average IQ losses by the size of the exposed population, the total loss in IQ points due to mercury exposures through consumption of self-caught freshwater fish was estimated to be 25,545 in 2005. For the 2016 base case, the total decrease in IQ points was estimated to be 24,419 (4.4% lower).

5.9.2 IQ Loss and Economic Valuation Estimates

In addition to the base case scenarios described above, CMAQ air quality modeling runs were used to estimate average mercury deposition levels for three emissions control scenarios:

- **2005 EGU Zero-Out.** This scenario represents total mercury deposition from all global natural and anthropogenic sources *except for U.S. EGUs* based on current-day conditions.

- **2016 EGU Zero-Out.** This scenario represents total mercury deposition from all global natural and anthropogenic sources *except for U.S. EGUs* based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule).

- **2016 Toxics Rule.** This scenario represents total mercury deposition from all global natural and anthropogenic sources based on projected 2016 conditions, including future anticipated regulations (e.g., Transport Rule) *and* the Toxics Rule.

For these three scenarios, it was again assumed that the HUC-level fish tissue mercury concentrations would change (relative to the 2005 base case) by the same percentage as the change in modeled deposition levels.

Mercury exposure and IQ loss estimates were then derived for these three scenarios, using the exposed population estimates for the relevant year (2005 or 2016) and the corresponding mercury concentration estimates for the relevant emission scenario (zero-out or Toxics Rule). In addition, the valuation methodology summarized in Section 5.7.2 (in particular, Equation [5.7]) was applied to estimate the present value of IQ loss estimates for the two base case and three emissions control scenarios.

To assess the aggregate benefits of reductions in EGU emissions, we evaluated five *emission reduction scenarios*.

- 2005 EGU zero-out (relative to 2005 base case)
- 2016 base case (relative to 2005 base case)
- 2016 EGU zero-out (relative to 2016 base case)
- 2016 Toxics Rule (relative to 2005 base case)
- 2016 Toxics Rule (relative to 2016 base case)

The benefits of each emission reduction scenario are calculated as the difference (i.e., decrease) in total present value of IQ losses between the selected emission control scenario and the selected base case scenario.

5.9.3 Primary Results for National Analysis of Exposures from Recreational Freshwater Fish Consumption

Table 5-6 summarizes the aggregate national IQ and present-value loss estimates for the two base case and three emission control scenarios. The highest losses are estimated for the 2005 base case. For the population of prenatally exposed children included in the analysis (almost 240,000, as reported in Table 5-5), mercury exposures under baseline conditions during the year 2005 are estimated to have resulted in more than 25,500 IQ points lost. Assuming a 3% discount rate, the present value of these losses ranges from $204.8 million to $292.5 million.[1] This range of total loss estimates is based on the range of per-IQ-point value (VIQ) estimates summarized in Section 5.7.3. These losses represent expected present value of declines in future net earnings over the entire lifetimes of the children who are prenatally exposed during the year 2005. With a 7% discount rate, the present value range is considerably lower: $22.8 million to $50.0 million.

The lowest losses are estimated to result from the 2016 zero-out scenario, with total IQ losses of less than 24,000 among roughly 244,000 prenatally exposed children and present values of these losses ranging from $190.2 to $281.3 million (3% discount rate).

[1] Monetized benefits estimates are for an immediate change in MeHg levels in fish. If a lag in the response of MeHg levels in fish were assumed, the monetized benefits could be significantly lower, depending on the length of the lag and the discount rate used. As noted in the discussion of the Mercury Maps modeling, the relationship between deposition and fish tissue MeHg is proportional in equilibrium, but the MMaps approach does not provide any information on the time lag of response.

Table 5-6. Summary Estimates of the Aggregate Size and Present Value of IQ Losses Under Alternative Base Case and Emissions Control Scenarios

Scenario	Average IQ Loss per Prenatally Exposed Child (dIQ)	Total IQ Losses from One Year of Exposure	Present Value of Total IQ Losses (2006 dollars)	
			3% Discount Rate	7% Discount Rate
2005 base case	0.1068	25,544.9	$204,690,894 - $302,936,392	$22,811,552 - $50,016,819
2005 EGU zero-out	0.0985	23,561.5	$188,798,519 - $279,416,153	$21,040,444 - $46,133,471
2016 base case	0.1000	24,419.4	$195,672,451 - $289,589,366	$21,806,502 - $47,813,136
2016 EGU zero-out	0.0971	23,722.2	$190,085,858 - $281,321,377	$21,183,910 - $46,448,036
2016 Toxics Rule	0.0979	23,908.6	$191,579,401 - $283,531,775	$21,350,356 - $46,812,987

For the five emission reduction scenarios described above, Table 5-7 reports estimates of aggregate nationwide benefits associated with reductions in mercury exposures and resulting reductions in IQ losses. Most importantly, the benefits of the 2016 Toxics Rule scenario (relative to the 2016 base case) are estimated to range between $4.1 million and $5.8 million (assuming a 3% discount rate), because of an estimated 511 point reduction in IQ losses. These benefits are 73% as large as the benefits of the 2016 zero-out scenario (relative to the same 2016 base case). Relative to the *2005* base case, the benefits of the 2016 Toxics Rule scenario range from $13.1 million to $18.7 million (3% discount). Despite growth in the exposed population from 2005 to 2016, the changes from the 2005 base case to the 2106 base case account for 69% of these benefits, while the changes from the 2016 base case to the 2016 Toxics Rule account for 31%.

Table 5-7. Aggregate Benefit Estimates for Reductions IQ Losses Associated with Alternative Emissions Reduction Scenarios

Emission Reduction Scenario	Decrease in Average IQ Loss per Prenatally Exposed Child (dIQ)[a]	Decrease in Total IQ Losses from One Year of Exposure	Present Value of Decrease in Total IQ Losses (2006 dollars)	
			3% Discount Rate	7% Discount Rate
2005 EGU zero-out (relative to 2005 base case)	0.00829	1983.3	$15,892,375 - $23,520,239	$1,771,108 - $3,883,348
2016 base case (relative to 2005 base case)	0.00684	1125.5	$9,018,443 - $13,347,026	$1,005,051 - $2,203,683
2016 EGU zero-out (relative to 2016 base case)	0.00285	697.2	$5,586,592 - $8,267,989	$622,592 - $1,365,100
2016 Toxics Rule (relative to 2005 base case)	0.00893	1636.3	$13,111,493 - $19,404,617	$1,461,196 - $3,203,832
2016 Toxics Rule (relative to 2016 base case)	0.00209	510.8	$4,093,050 - $6,057,591	$456,145 - $1,000,149

[a] As reported in Table 5-5, the estimated number of prenatally exposed children is 239,174 in 2005 and 244,286 in 2016.

5.9.4 Primary Results for Potentially High-Risk Subpopulations

As described in Section 5.7.4, the methodology used to estimate the *aggregate* benefits of mercury emission reductions was also adapted and applied to assess effects on the distribution of *individual-level* risks among specific potentially high-risk subpopulations in the United States. The analysis of these subpopulations focuses on the distribution of individual mercury exposures risks (i.e., expected IQ loss) within these groups, particularly in the high end of the risk distributions. It also examines the distribution of reductions in IQ loss in these groups as a result of EGU mercury emission reductions.

Low-Income African-American Recreational/Subsistence Fishers in the Southeast Region

The analysis of low-income African-Americans in the Southeast United States focuses on census tracts that have (1) at least one HUC-12 within 20 miles with a mercury fish tissue concentration estimate and (2) at least 25 African-American inhabitants living below the poverty level. Using county-level growth projections, there were an estimated 3.09 million low-income

African Americans in these areas in 2005, and 3.56 million are expected to reside in these areas in 2016.[1] The geographic distribution of the expected 2016 population is shown in Figure 5-6.

The spatial distribution of these population estimates for *all* low-income African Americans in these areas was used to model the distribution of risk among pregnant women in recreational/subsistence fishing households in this group. This approach was used because population estimates for this latter group are not readily available and difficult to generate. The approach assumes that the spatial distribution of these pregnant women is the same as for the total low-income African American population in these areas. Populations were linked at the tract level to average mercury fish tissue concentrations within 20 miles using the methods described in Section 5.7.4. The distribution of mercury ingestion and IQ loss resulting from these fish tissue levels was then simulated using the fish consumption rate distribution for this subpopulation summarized in Table 5-3.

[1] A rough approximation of the size of the annual exposed population – low-income African American pregnant women in angler households in the Southeast in 2016 – can be estimated by multiplying the total population estimate (3.56 million) by (1) the percent of the total U.S. population that is female and age 15-44 (21%; Census 2000) (2) the fertility rate for African American women of childbearing age (6.9%; U.S. Vital Statistics 2005), and (3) the percent of the low-income African American population that are freshwater fishers (6-8%; Henderson 2004). The resulting estimated exposed population is between 9,400 and 12,600 individuals.

Figure 5-6. Modeled African-American Population below the Poverty Level by Census Tract in the Southeast for 2016

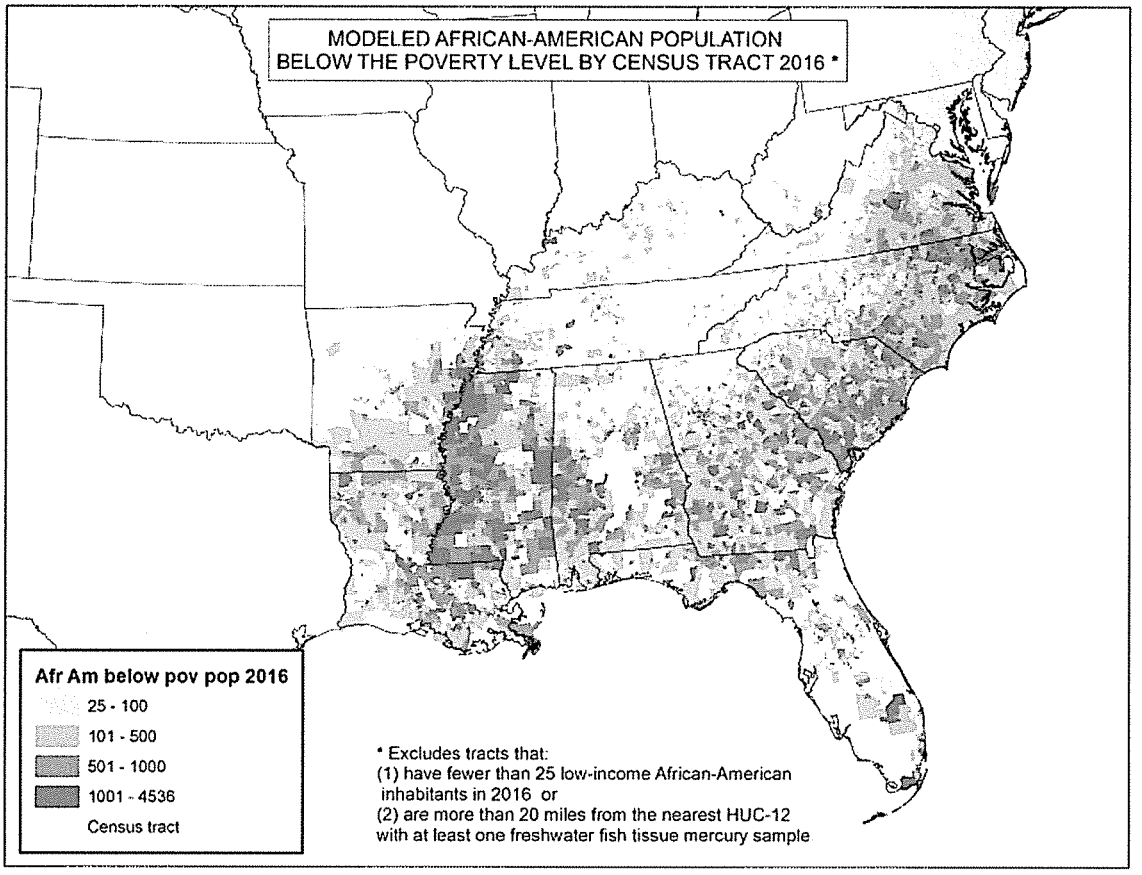


Table 5-8 summarizes the estimated distribution of IQ loss among this subpopulation due to mercury exposure through consumption of self-caught freshwater fish. These distribution estimates are reported for populations in two time periods (2005 and 2016) under base case conditions and under alternative emission control scenarios. In the 2016 base case, the median IQ loss is estimated to be 1.87 points and the 95th percentile is 11.56 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 1.82 points and the 95th percentile to 11.33 points.

Table 5-8. Simulated Distribution of IQ Loss (per Exposed Person) for Low-Income African-American Recreational/Subsistence Fishers in the Southeast Region

Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 base case	0.930	1.980	4.173	8.104	12.023	3.601
2005 EGU zero-out	0.835	1.805	3.854	7.555	11.271	3.342
2016 base case	0.868	1.868	3.974	7.771	11.556	3.437
2016 EGU zero-out	0.832	1.801	3.848	7.551	11.246	3.335
2016 Toxics Rule	0.843	1.820	3.884	7.611	11.328	3.363

Table 5-9 summarizes the estimated distribution of *reductions* in IQ loss associated with alternative emission reduction scenarios.[1] The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.032 points, and the 95th percentile is 0.274 points.

Table 5-9. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Low-Income African-American Recreational/Subsistence Fishers in the Southeast Region

Emission Reduction Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 EGU zero-out (relative to 2005 base case)	0.049	0.123	0.290	0.607	0.935	0.260
2016 EGU zero-out (relative to 2016 base case)	0.019	0.050	0.118	0.240	0.363	0.103
2016 Toxics Rule (relative to 2016 base case)	0.011	0.032	0.082	0.176	0.274	0.074

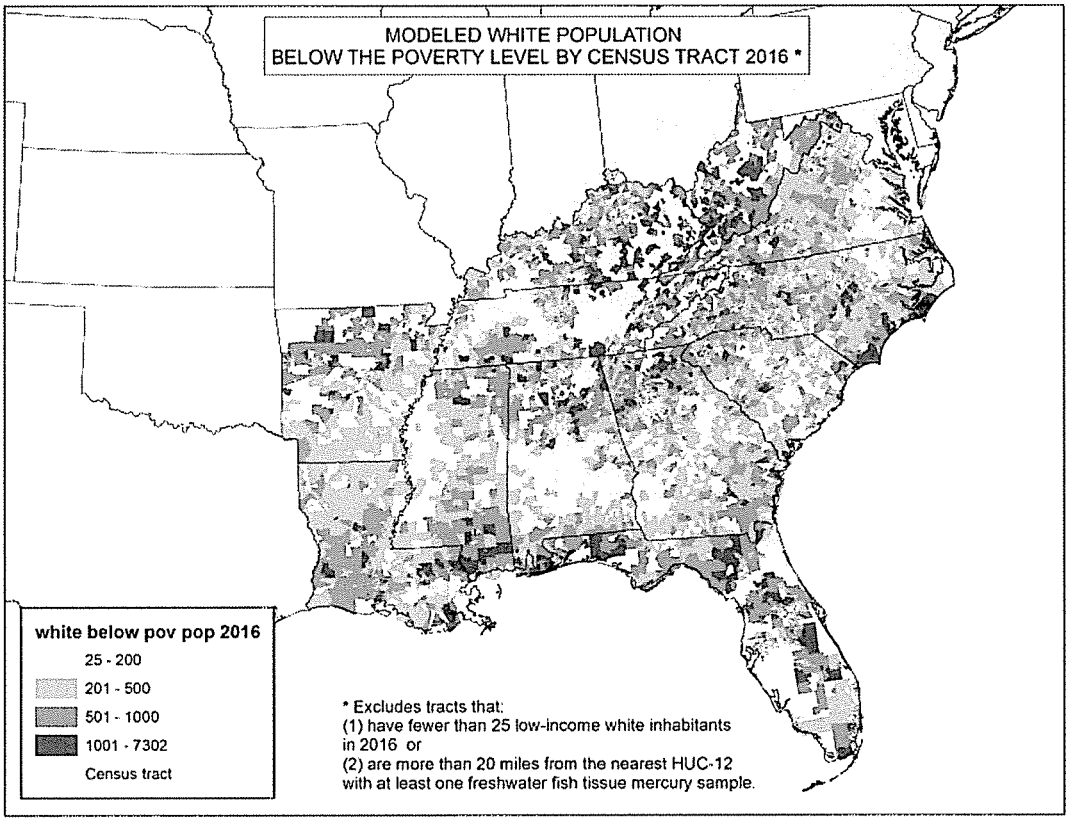
Low-Income White Recreational/Subsistence Fishers in the Southeast Region

The analysis of the low-income white population in the Southeast United States uses the same methodology as for the low-income African-American subpopulation, except that it uses tract-level population estimates for all whites living below the poverty level in the southeast

[1] The scenarios comparing different years (i.e., 2016 base case [relative to 2005 base case] and 2016 TOXICS [compared to 2005 base case]) were not evaluated for the *reduction* in individual exposure analysis because the exposed populations are different in the two periods.

United States, and it uses the different fish consumption rate distribution for this population described in Table 5-3. For this group, the spatial distribution of all low-income whites in these areas was used to simulate the distribution of IQ losses resulting from exposures via pregnant women in recreational/subsistence fishing households. The total low-income white population in the southeastern states was 3.26 million for 2005 and was projected to be 3.58 million in 2016.[1] The geographic distribution of this population for 2016 is shown in Figure 5-7.

Figure 5-7. Modeled White Population below the Poverty Level by Census Tract in the Southeast for 2016



[1] A rough approximation of the size of the annual exposed population – low-income white pregnant women in angler households in the Southeast in 2016-- can be estimated by multiplying the total population estimate (3.58 million) by (1) the percent of the total U.S. population that is female and age 15-44 (21%; Census 2000) (2) the fertility rate for white women of childbearing age (6.6%; U.S. Vital Statistics 2005), and (3) the percent of the low-income white population that are freshwater fishers (10-11%; Henderson 2004). The resulting estimated exposed population is between 15,800 and 17,400 individuals.

Table 5-10 summarizes the estimated distribution of IQ loss among this subpopulation. In the 2016 base case, the median IQ loss is estimated to be 0.188 points, and the 95th percentile is 2.459 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 0.184 points and the 95th percentile to 2.415 points.

Table 5-10. Simulated Distribution of IQ Loss (per Exposed Person) for Low-Income White Recreational/Subsistence Fishers in the Southeast Region

	Percentile					
Scenario	25th	50th	75th	90th	95th	Mean
2005 base case	0.071	0.203	0.580	1.480	2.586	0.670
2005 EGU zero-out	0.063	0.182	0.527	1.362	2.396	0.618
2016 base case	0.065	0.188	0.541	1.396	2.459	0.633
2016 EGU zero-out	0.062	0.182	0.525	1.358	2.396	0.617
2016 Toxics Rule	0.063	0.184	0.530	1.371	2.415	0.622

Table 5-11 summarizes the estimated distribution of reductions in IQ loss associated with alternative emission reduction scenarios. The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.002 points, and the 95th percentile is 0.045 points.

Table 5-11. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Low-Income White Recreational/Subsistence Fishers in the Southeast Region

	Percentile					
Emission Reduction Scenario	25th	50th	75th	90th	95th	Mean
2005 EGU zero-out (relative to 2005 base case)	0.004	0.013	0.042	0.113	0.204	0.051
2016 EGU zero-out (relative to 2016 base case)	0.001	0.004	0.013	0.036	0.066	0.017
2016 Toxics Rule (relative to 2016 base case)	0.001	0.002	0.008	0.024	0.045	0.011

Low-Income Female Recreational/Subsistence Fisher

The analysis of the low-income female population uses tract-level female population and poverty estimates for the entire continental United States, and it uses the specific fish

consumption rate distribution for this population described in Table 5-6. For this group, the total modeled populations used to simulate the distribution of IQ loss were 18.4 million for 2005 and 20.1 million for 2016.[1] The geographic distribution of the population modeled for 2016 is shown in Figure 5-8.

Figure 5-8. Modeled Female Population below the Poverty Level by Census Tract for 2016

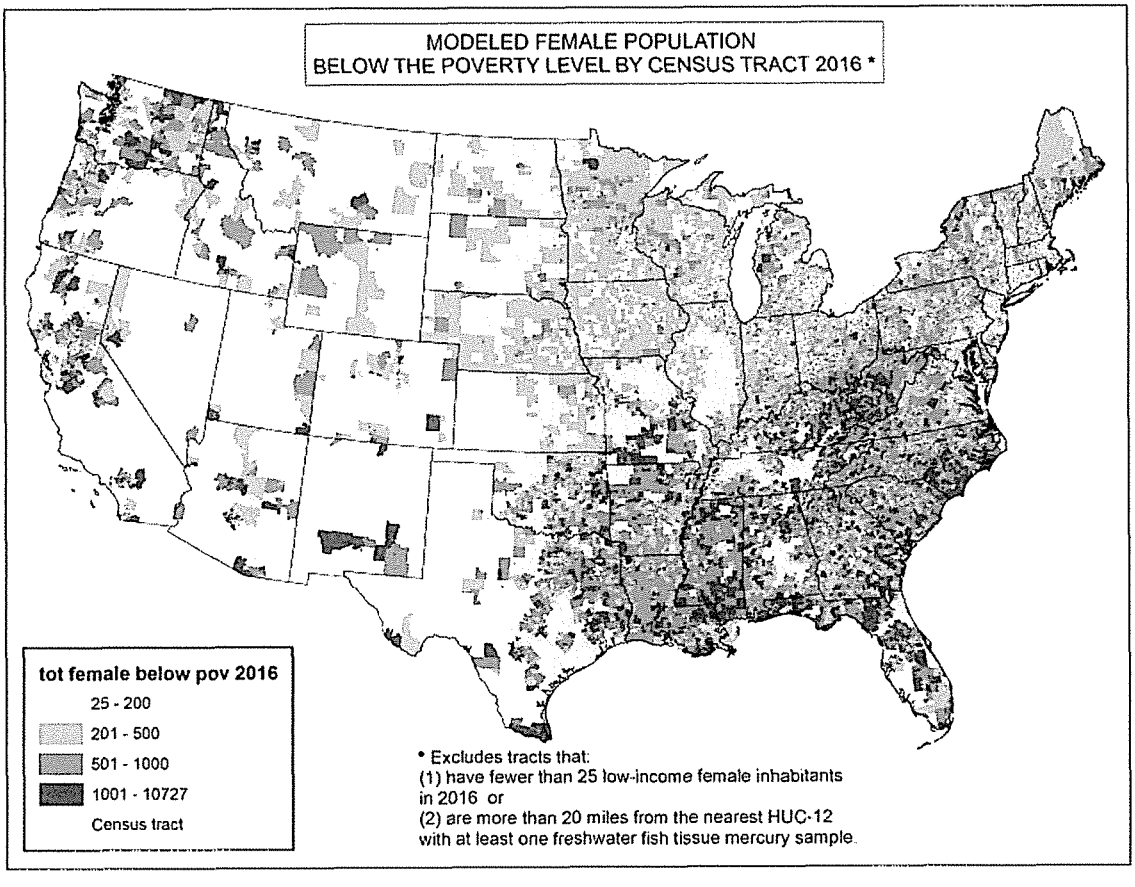


Table 5-12 summarizes the estimated distribution of IQ loss among this subpopulation. In the 2016 base case, the median IQ loss is estimated to be 0.111 points, and the 95th percentile is 3.122 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 0.108 points and the 95th percentile to 3.064 points.

[1] A rough approximation of the size of the annual exposed population – low-income pregnant women in angler households in 2016-- can be estimated by multiplying the total population estimate (20.1 million) by (1) the percent of the total female U.S. population that is age 15-44 (42%; Census 2000) (2) the fertility rate for all women of childbearing age (6.7%; U.S. Vital Statistics 2005), and (3) the percent of the low-income women that are freshwater fishers (7%; Henderson 2004). The resulting estimated exposed population is roughly 62,100 individuals.

Table 5-12. Simulated Distribution of IQ Loss (per Exposed Person) for Low-Income Female Recreational/Subsistence Fishers in the United States

Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 Base Case	0.030	0.122	0.480	1.629	3.373	0.932
2005 EGU Zero-out	0.027	0.109	0.431	1.477	3.073	0.855
2016 Base Case	0.027	0.111	0.439	1.499	3.122	0.868
2016 EGU Zero-Out	0.026	0.107	0.425	1.457	3.038	0.846
2016 Toxics Rule	0.026	0.108	0.429	1.470	3.064	0.853

Table 5-13 summarizes the estimated distribution of reductions in IQ loss associated with alternative emission reduction scenarios. The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.001 points, and the 95th percentile is 0.053 points.

Table 5-13. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Low-Income Female Recreational/Subsistence Fishers in the United States

Emission Reduction Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 EGU zero-out (relative to 2005 base case)	0.001	0.007	0.034	0.127	0.274	0.077
2016 EGU zero-out (relative to 2016 base case)	0.000	0.002	0.010	0.037	0.078	0.021
2016 Toxics Rule (relative to 2016 base case)	0.000	0.001	0.006	0.024	0.053	0.015

Hispanic Subsistence Fisher

The analysis of the Hispanic population uses tract-level population estimates for this group for the entire continental United States, and it uses the specific fish consumption rate distribution for this population described in Table 5-6. For this group, the total modeled populations used to simulate the distribution of IQ loss were 19.6 million for 2005 and 27.2

million for 2016.[1] The geographic distribution of the population modeled for 2016 is shown in Figure 5-9.

Figure 5-9. Modeled Hispanic Population by Census Tract for 2016

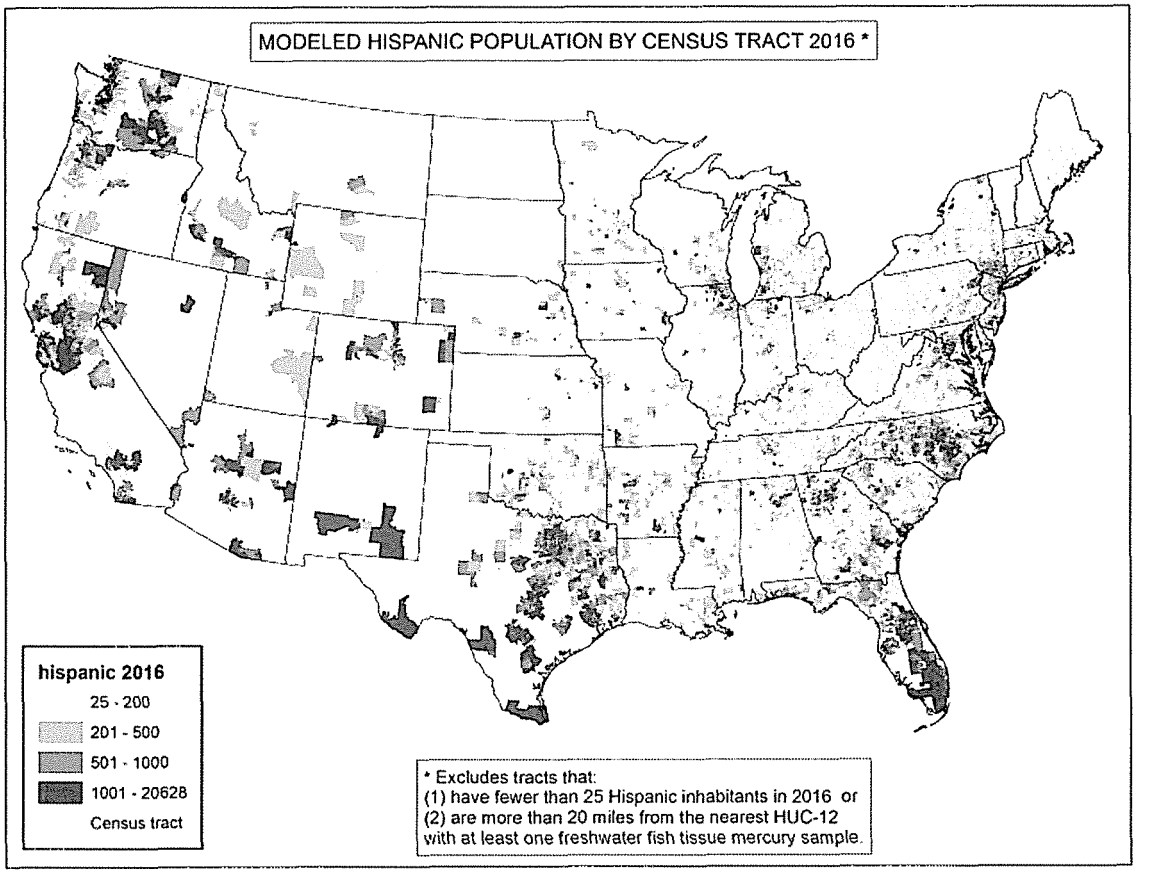


Table 5-14 summarizes the estimated distribution of IQ loss among this subpopulation. In the 2016 base case, the median IQ loss is estimated to be 0.184 points, and the 95th percentile is 2.494 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 0.180 points and the 95th percentile to 2.468 points.

[1] A rough approximation of the size of the annual exposed population – Hispanic pregnant women in angler households in 2016-- can be estimated by multiplying the total population estimate (27.2 million) by (1) the percent of the total U.S. population that is female and age 15-44 (21%; Census 2000) (2) the fertility rate for Hispanic women of childbearing age (9.9%; U.S. Vital Statistics 2005), and (3) the percent of the Hispanic population that are freshwater fishers (5-6%; Henderson 2004). The resulting estimated exposed population is between 59,900 and 71,900 individuals.

Table 5-14. Simulated Distribution of IQ Loss (per Exposed Person) for Hispanic Recreational/Subsistence Fishers in the United States

Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 base case	0.066	0.202	0.588	1.530	2.732	0.724
2005 EGU zero-out	0.060	0.186	0.546	1.435	2.578	0.686
2016 base case	0.059	0.184	0.538	1.402	2.494	0.657
2016 EGU zero-out	0.058	0.179	0.526	1.378	2.456	0.647
2016 Toxics Rule	0.058	0.180	0.530	1.385	2.468	0.650

Table 5-15 summarizes the estimated distribution of reductions in IQ loss associated with alternative emission reduction scenarios. The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.001 points, and the 95th percentile is 0.03 points.

Table 5-15. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Hispanic Recreational/Subsistence Fishers in the United States

Emission Reduction Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 EGU zero-out (relative to 2005 base case)	0.001	0.006	0.027	0.084	0.157	0.038
2016 EGU zero-out (relative to 2016 base case)	0.000	0.002	0.008	0.023	0.042	0.010
2016 Toxics Rule (relative to 2016 base case)	0.000	0.001	0.005	0.016	0.030	0.007

Laotian Subsistence Fishers

The analysis of the Laotian population uses tract-level population estimates for this group for the entire continental United States, and it uses the specific fish consumption rate distribution for this population described in Table 5-6. For this group, the total modeled populations used to simulate the distribution of IQ loss were 80,000 for 2005 and 137,500 for 2016.[1] The geographic distribution of the population modeled for 2016 is shown in Figure 5-10.

[1] A rough approximation of the size of the annual exposed population -- Laotian pregnant women in angler households in 2016-- can be estimated by multiplying the total population estimate (0.14 million) by (1) the percent of the total U.S. population that is female and age 15-44 (21%; Census 2000) (2) the fertility rate for Asian women of childbearing age (6.7%; U.S. Vital Statistics 2005), and (3) the percent of the Laotian

Figure 5-10. Modeled Laotian Population by Census Tract for 2016

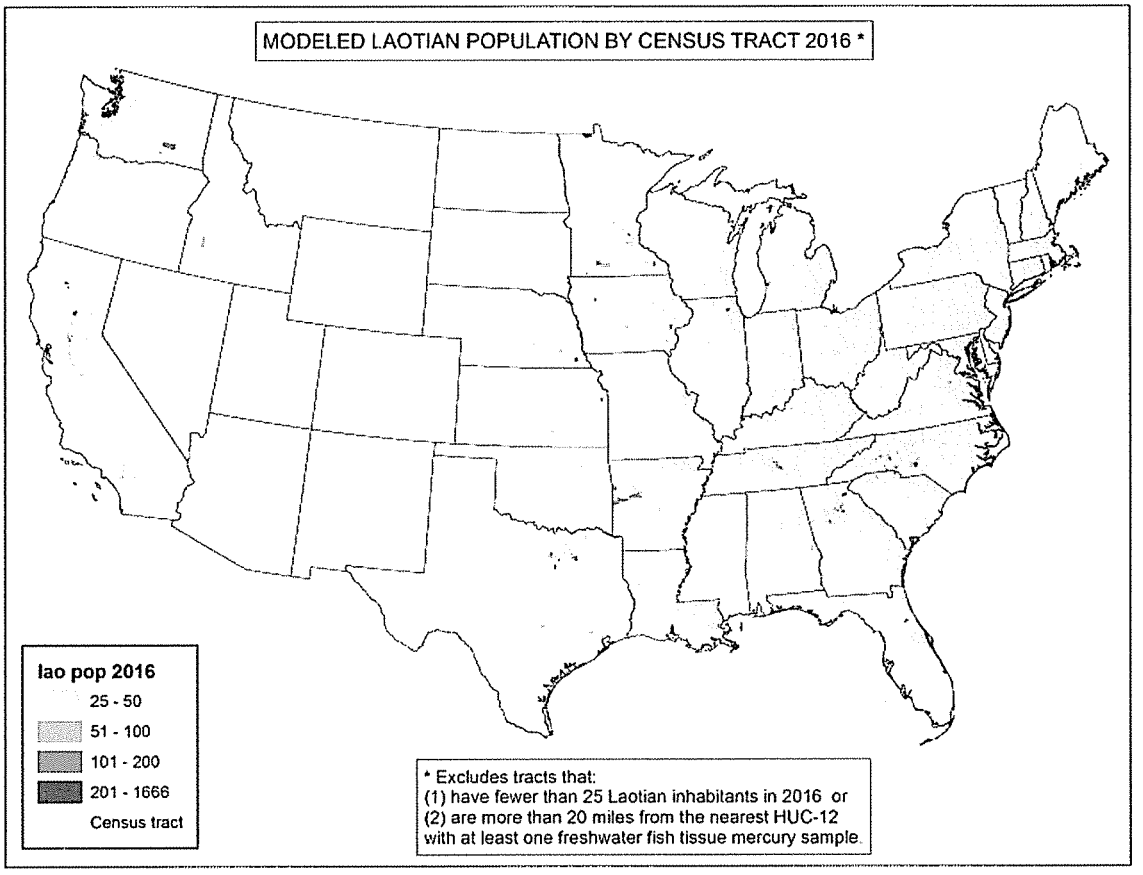


Table 5-16 summarizes the estimated distribution of IQ loss among this subpopulation. In the 2016 base case, the median IQ loss is estimated to be 0.196 points, and the 95th percentile is 4.260 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 0.192 points and the 95th percentile to 4.210 points.

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population that are freshwater fishers (13-50%; Henderson, 2004; Hutchison and Kraft, 1994). The resulting estimated exposed population is between 600 and 3,000 individuals.

Table 5-16. Simulated Distribution of IQ Loss (per Exposed Person) for Laotian Recreational/Subsistence Fishers in the United States

Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 base case	0.061	0.212	0.744	2.330	4.559	1.265
2005 EGU zero-out	0.057	0.198	0.692	2.197	4.335	1.201
2016 base case	0.056	0.196	0.688	2.142	4.260	1.145
2016 EGU zero-out	0.054	0.190	0.671	2.097	4.184	1.126
2016 Toxics Rule	0.055	0.192	0.676	2.111	4.210	1.133

Table 5-17 summarizes the estimated distribution of reductions in IQ loss associated with alternative emission reduction scenarios. The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.001 points, and the 95th percentile is 0.047 points.

Table 5-17. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Laotian Recreational/Subsistence Fishers in the United States

Emission Reduction Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 EGU zero-out (relative to 2005 base case)	0.000	0.003	0.022	0.093	0.209	0.064
2016 EGU Zero-out (relative to 2016 base case)	0.001	0.003	0.011	0.036	0.073	0.019
2016 Toxics Rule (relative to 2016 base case)	0.000	0.001	0.006	0.022	0.047	0.012

Chippewa Tribe Members in the Great Lakes Area

The analysis of the Chippewa population uses tract-level population estimates for this group in Minnesota, Wisconsin, and Michigan, for tracts within 20 miles of the fishing area ceded to the tribe. It uses the specific fish consumption rate distribution for this population described in Table 5-6. For this group, the total modeled populations used to simulate the distribution of IQ loss were 23,900 for 2005 and 29,500 for 2016.[1] The geographic distribution of the population modeled for 2016 is shown in Figure 5-11.

[1] A rough approximation of the size of the annual exposed population – Chippewa pregnant women in angler households in the Great Lakes are 2016-- can be estimated by multiplying the total population estimate (29.5

Figure 5-11. Modeled Chippewa Population by Census Tract in the Great Lakes Area for 2016

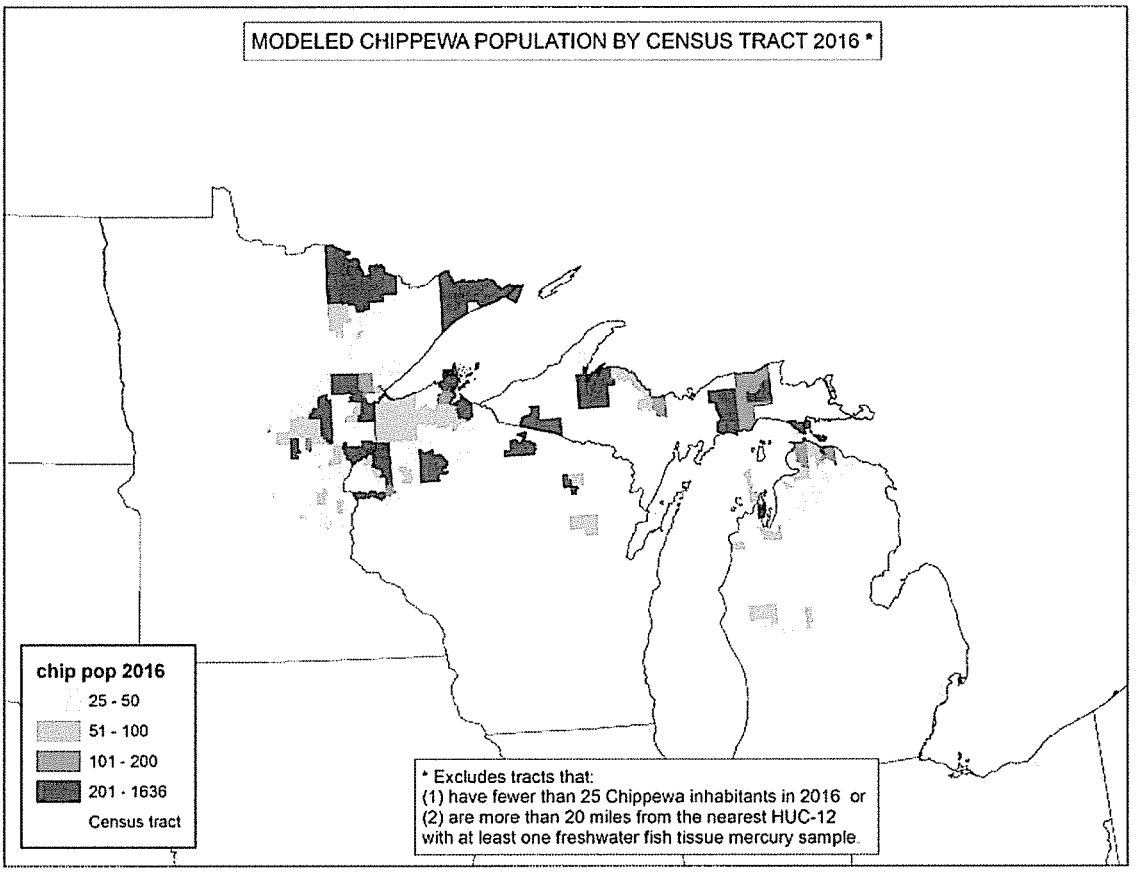

Table 5-18 summarizes the estimated distribution of IQ loss among this subpopulation. In the 2016 base case, the median IQ loss is estimated to be 0.398 points and the 95th percentile is 3.284 points per exposed individual. Under the 2016 Toxics Rule, the median declines to 0.392 points and the 95th percentile to 3.241 points.

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thousand) by (1) the percent of the total U.S. population that is female and age 15-44 (21%; Census 2000) (2) the fertility rate for American Indian women of childbearing age (6%; U.S. Vital Statistics 2005), and (3) the percent of the Chippewa population that are freshwater fishers (26%, assumed to be twice national average). The resulting estimated exposed population is roughly 300 individuals.

Table 5-18. Simulated Distribution of IQ Loss (per exposed person) for Chippewa in the Great Lakes Area

Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 base case	0.175	0.410	0.989	2.125	3.341	0.940
2005 EGU zero-out	0.168	0.397	0.955	2.049	3.237	0.909
2016 base case	0.165	0.398	0.949	2.089	3.284	0.902
2016 EGU zero-out	0.162	0.391	0.933	2.056	3.231	0.887
2016 Toxics Rule	0.163	0.392	0.937	2.062	3.241	0.890

Table 5-18 summarizes the estimated distribution of reductions in IQ loss associated with alternative emission reduction scenarios. The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case) scenario is 0.005 points, and the 95th percentile is 0.041 points.

Table 5-19. Simulated Distribution of Reduction in IQ Loss (per Exposed Person) for Chippewa in the Great Lakes Area

Emission Reduction Scenario	Percentile					Mean
	25th	50th	75th	90th	95th	
2005 EGU zero-out (relative to 2005 base case)	0.005	0.012	0.030	0.068	0.112	0.030
2016 EGU zero-out (relative to 2016 base case)	0.002	0.006	0.014	0.033	0.053	0.014
2016 Toxics Rule (relative to 2016 base case)	0.002	0.005	0.011	0.026	0.041	0.011

Comparison of Risk Distributions across High-Risk Subpopulations

Using the results summarized above, Figure 5-12 compares the simulated IQ loss distributions for the six potential high-risk subpopulations under the 2016 base case scenario. The low-income African-American population in the Southeast United States stands out from the other groups, with relatively high individual risk levels at each percentile. It is estimated that 5% of the prenatally exposed population in this group in 2016 (the 95th and greater percentile) would experience IQ losses of more than 11.55 points because of mercury in freshwater fish under base case conditions. The group with the next highest estimated individual risk levels at the 95th percentile and above is the Laotian subpopulation, with IQ losses of 4.26 points and greater.

Figure 5-12. Comparison of IQ Loss Distributions for Selected High-Risk Populations (2016 Base Case)

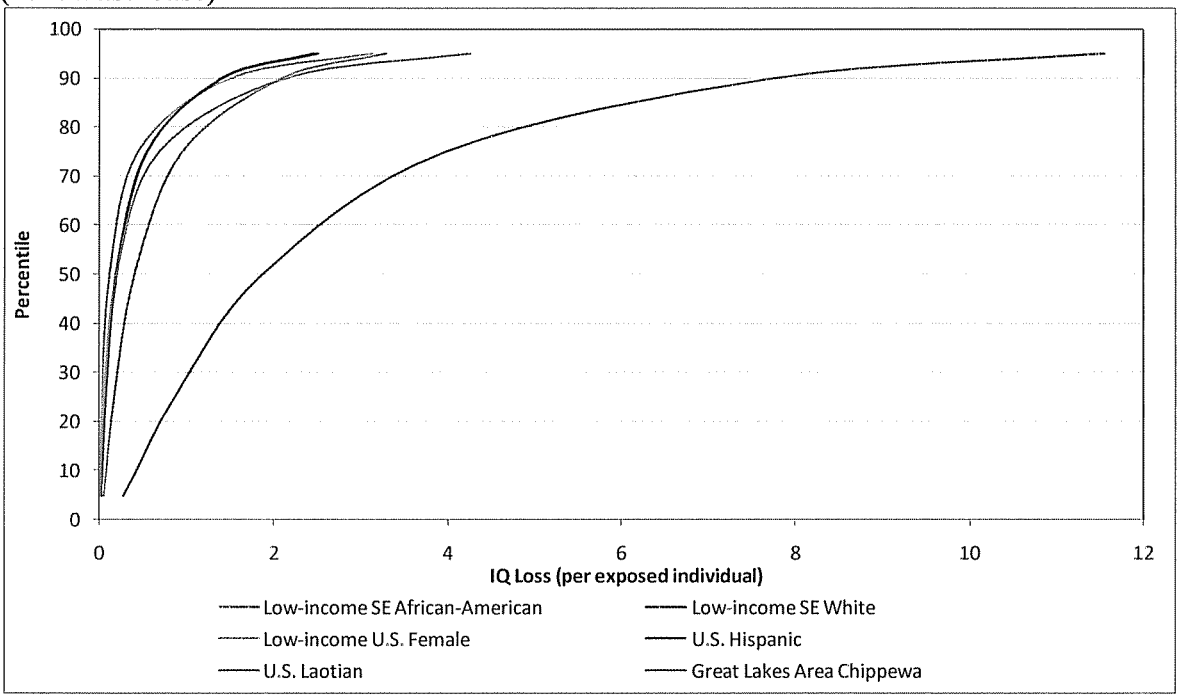
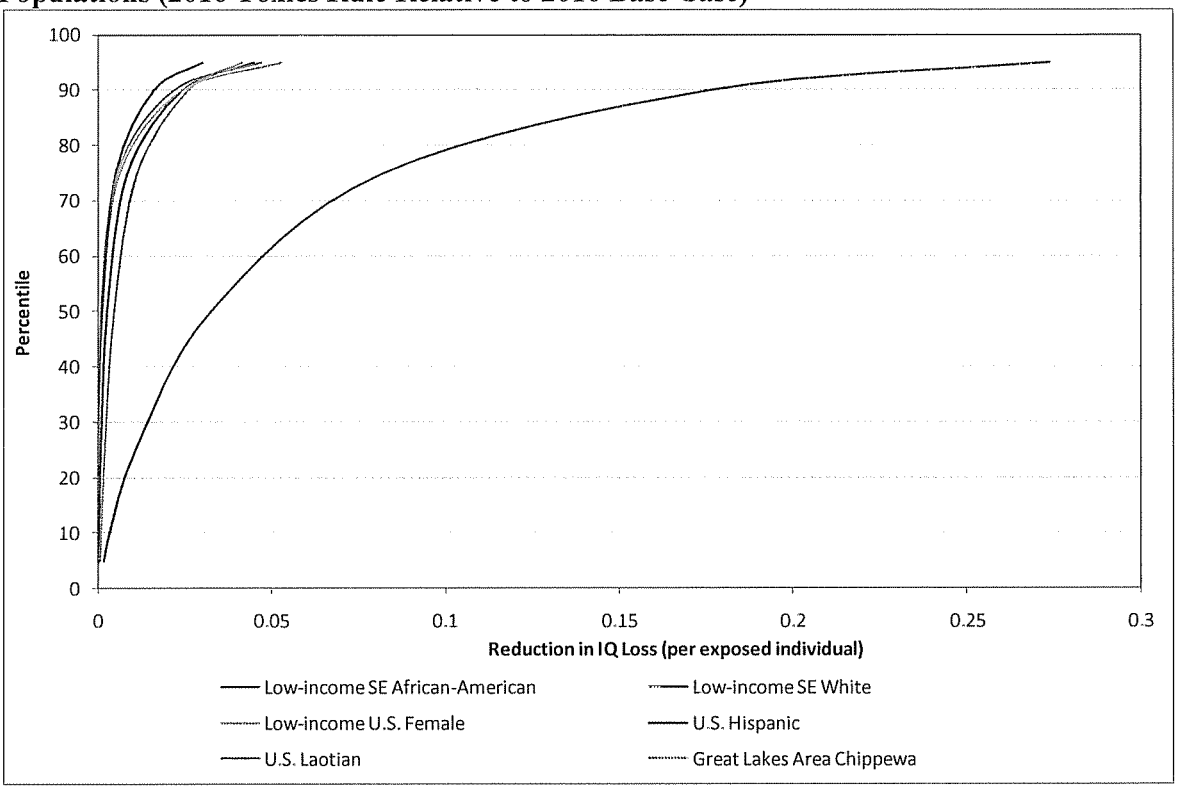


Figure 5-13 provides a similar comparison for the distribution of reductions in IQ loss associated with the 2016 Toxics Rule (relative to the 2016 base case). Again, the low-income African-American subpopulation stands out from the others with the highest reductions at each percentile. It is estimated that 5% of the prenatally exposed population in this group in 2016 would have expected reductions in IQ losses of more than 0.274 points because of reductions in mercury emissions. At the 95th percentile, all of the other subpopulations have expected reductions in IQ loss between 0.03 and 0.053 IQ points.

Figure 5-13. Comparison of Reduction in IQ Loss Distributions for Selected High-Risk Populations (2016 Toxics Rule Relative to 2016 Base Case)



5.9.5 Discussion of Assumptions, Limitations, and Uncertainties

Uncertainty regarding the model results and estimates reported in Section 5.8 can arise from several sources. Some of the uncertainty can be attributed to model uncertainty. For example, to estimate exposures a number of different modeling approaches have been selected and combined. The separate model components are summarized in Figure 5-4 and equations (5.) to (5.8), each of which simplifies potentially complex processes. The results, therefore, depend importantly on how these models are selected, specified, and combined.

Another important source of uncertainty can be characterized as input or parameter uncertainties. Each of the modeling components discussed in this report requires summary data and estimates of key model parameters. For example, estimating IQ losses associated with consumption of freshwater fish requires estimates of the size of the exposed population of interest, the average mercury concentrations in consumed fish, the freshwater fish consumption rate for the exposed population, and the concentration-response relationship between mercury ingestion and IQ loss. All of these inputs are measured with some degree of uncertainty and can affect, to differing degrees, the confidence range of our summary results. The discussion below

identifies and highlights some of the key model parameters, characterizes the source and extent of uncertainties associated with them, and characterizes the potential effects of these uncertainties on the model results.

To organize this discussion, we discuss different components of the modeling framework separately. This section first discusses issues related to estimating the mercury concentrations and then those related to estimating the exposed population. After that, it discusses issues related to matching these two components and then concludes by discussing the estimation of mercury ingestion through fish consumption.

Mercury Concentration Estimates

As described in Section 5.2.2, the mercury concentration estimates for the analysis come from several different sources, including fish tissue sample data from the National Listing of Fish Advisories (NLFA) and several other state- and national-level sources. These estimates were then used to approximate mercury concentrations across the study area. Some of the key assumptions, limitations, and uncertainties associated with these estimates are the following:

- The fish tissue sampling data from various sources are subject to measurement and reporting error and variability. The NLFA is the largest and most detailed source of data on mercury in fish; however, even this system was not centrally designed (e.g., by EPA) using a common set of sampling and analytical methods. Rather, states collected the data primarily to support the development of advisories, and the data are submitted voluntarily to EPA. Each state uses different methods and criteria for sampling and allocates different levels of resources to their monitoring programs. In addition, there are uncertainties regarding the precise locations (lat/long coordinates) of some of the samples. The heterogeneity and potential errors across state sampling programs can bias the results in any direction and contribute to uncertainty.

- The fish tissue sampling data were assigned as either lake or river samples, based on the site name and/or the location coordinates mapped to the nearest type of waterbody. This process also involves measurement error and may have resulted in misclassifications for some of the samples. These errors are not expected to bias results, but they contribute to uncertainty.

- The mercury concentration estimates used in the model were based on simple temporal and spatial averages of reported fish tissue samples. This approach assumes that the mercury samples are representative of "local" conditions (i.e., within the

same HUC-12) in similar waterbodies (i.e., rivers or lakes). However, even though states use a variety of approaches to monitor and sample fish tissue contaminants, in some cases, the sampling sites are selected to target areas with high levels of angler activity and/or a high level of pollution potential. To the extent that sample selection procedures favor areas with relatively high mercury, the spatial extrapolation methods used in this report will tend to overstate exposures. These approaches also implicitly assume that mercury concentration estimates are strongly spatially correlated, such that closer sampling sites (i.e., from the same HUC or distance interval) provide more information about mercury concentrations than more distant sites. To the extent that spatial correlation is weaker than assumed, this will increase the degree of uncertainty in the modeling results.

- To generate average mercury fish tissue concentration estimates, all available samples from the three main data sources (1995-2009) and from freshwater fish larger the 7 inches were included in the analysis. Smaller fish were excluded to better approximate concentrations in the types of fish that are more likely to be consumed, and samples from years before 1995 were excluded to better represent more recent conditions. Even with these sample selection procedures, average concentration estimates from the retained samples may still under or overestimate actual concentrations in currently consumed fish.

Exposed Population Estimates

The methods described in Section 5.7 to estimate the total exposed population of interest in 2005 and 2016 involve the following key assumptions, limitations, and uncertainties:

- The approach relies on data from the FHWAR to estimate state-level freshwater angler activity levels, including freshwater fishing participation rates and lake-to-river trip ratios. Each of these data elements is measured with some error in the FHWAR, but they are based on a relatively large sample. More importantly the state-level averages are applied to each modeled census tract in the state; therefore, the model fails to capture within-state variation in these factors, which contributes to uncertainty in the model estimates.

- The analysis also uses state-level fertility rate data to approximate the rate of pregnancy among women of childbearing age in angler households for a smaller geographic area. The state-level fertility rates from the National Vital Statistics are estimated with relatively little error; however, applying these rates to specific

census tracts (and specifically to women in angler households) does involve considerably more uncertainty.

- The approach assumes that, in each census tract, the percentage of women who live in freshwater angler households (i.e., households with at least one freshwater angler) is equal to the percentage of the state adult population that fishes. Applying the state-level participation rate to approximate the conditions at a block level creates uncertainty. More importantly, however, using individual-based fishing participation rates to approximate household rates is likely to underestimate the percentage of women living in freshwater angler households.[1] Unfortunately, data on household participation levels in freshwater fishing are not readily available.

- Census tract populations are only included in the model if they are matched to distance intervals and waterbody types that have spatial overlap with at least one HUC-12 sub-watershed containing a mercury concentrations estimate for that waterbody type. By design, this approach undercounts the exposed population (by roughly 40 to 45%) and, therefore, leads to underestimates of national aggregate baseline exposures and risks and underestimates of the risk reductions and benefits resulting from mercury emission reductions.

- All of the tract-level population estimates are based on Census 2000 data, which are projected forward to 2005 and 2016 using county-level growth projections for the subpopulations of interest from Woods and Poole (2008). Therefore, the 2005 and 2016 population estimates incorporate uncertainty from both the growth projections themselves and from transferring the county-level growth estimates to the tract level.

The purpose of the analysis of potentially high risk subpopulations is not to estimate the size of the exposed population but rather to characterize the distribution of individual-level risks in the subpopulations of interest. Nevertheless, the size and spatial distribution of the total population in each group was used as a proxy for characterizing the spatial distribution of pregnant women in freshwater fishing households in each group.

[1] For example, hypothetically if one out of every three members in each household fished, the population rate would be 33%, but the household rate would be 100%.

The main assumption underlying this approach is that the expected proportion of the subgroup's population in each Census tract that consists of pregnant women in fishing households is the same across the selected census tracts. The main limitation of this assumption is that it does not allow or account for spatial variation in (1) the percentage of the subpopulation that are women of childbearing age, (2) the percentage of these women that are pregnant (i.e., fertility rate) and (3) the freshwater angler participation rates for the subgroups of interest. Unfortunately, spatially varying data for the last component (fishing participation rates among the subpopulations of interest) are not readily available. This assumption is not expected to bias the results but it does contribute to uncertainty in the estimated distributions of individual-level risks.

Matching of Exposed Populations to Mercury Concentrations

The methods described in Section 5.7 to match the exposed population estimates with the corresponding mercury concentration estimates involve the following key assumptions, limitations, and uncertainties:

- For the aggregate benefits analysis, tract-level exposed populations are assigned to waterbody types based on state-level ratios of lake-to-river fishing days (from the FHWAR). They are further assigned to distance intervals based on observed travel distance patterns in national fishing data (NSRE, 1994). Both of these assignment methods involve uncertainty, but particularly the second method because it is based on much more aggregate data and on a much smaller and more dated sample of anglers. This approach does not take into account the physical characteristics of the area in which the population is located. In particular, the allocation of exposures to lakes or rivers at different distances from each census tract does not take into account the presence or number of these waterbodies in each distance interval. Using these state and national level estimates to represent conditions at a local (i.e., census tract) level increases uncertainty in the model results, but it is not expected to bias the results in either direction.

- For the analysis of potentially high-risk populations, these methods and assumptions were slightly modified. In particular, because these analyses focus on low-income and/or subsistence fishing populations, all trips were assumed to occur within 20 miles of the census tract. Unfortunately, it is difficult to evaluate the accuracy of this restriction due to limited data on travel distances for the subgroups of interest.

One potentially important factor that is not included for matching populations and mercury concentrations is the effect of fish consumption advisories on fishing behavior. Evidence summarized in Jakus, McGuinness, and Krupnick (2002) suggests that awareness of advisories by anglers is relatively low (less than 50%), and even those who are aware do not always alter their fishing behavior. Nonetheless, anglers are less likely to fish in areas with advisories. Unfortunately, we were not able to reliably quantify the reduction and redistribution of fishing trips in either model to account for fish advisories. By excluding these effects, the model estimates are likely to overstate mercury exposures.

Fish Consumption Estimates

One of the most influential variables in both modeling approaches is the rate of self-caught freshwater fish consumption. The following key assumptions, limitation, and uncertainties are associated with the methods used:

- For the aggregate analysis we have assumed 8 g/day for the general population in freshwater angler households (based on recommendations in EPA's EFH). Unfortunately, data are not available to reliably vary this rate with respect to characteristics of the population across the entire study area. Uncertainty regarding the true average fish consumption rate has a direct effect on uncertainty for the aggregate exposure and benefit estimates. Because a single consumption rate is applied uniformly across the entire exposed population and because it is a multiplicative factor in the model, the two uncertainties are directly proportional to one another. The recommended 8 g/day rate is based on four studies with mean estimates ranging from 5 g/day (37% less than 8) to 17 g/day (113% more than 8). If it is assumed that this range of estimates represents the uncertainty in the *mean* freshwater fish consumption rate for the study population, then the resulting uncertainty range for the estimated *mean* mercury ingestion level (and resulting IQ loss) will also be between –37% and +113% of the mean mercury ingestion level.

- To analyze the distributions of individual-level risks in potentially high risk subpopulations, we applied empirical distributions of fish consumption rates for specific subpopulations. One of the main limitations of this approach is that these empirical distributions are based on relatively small and localized samples. In particular, the estimated distribution of consumption rates for low-income African American subsistence/recreational fishers in the Southeastern U.S. (see Table 5.3) is based on a very small sample (N=39) drawn from one location (Columbia, SC). The sample sizes

for the other groups, particularly the Hispanic (N= 45) and Laotian (N=54) populations are also small; therefore, there is considerable uncertainty regarding how well these empirical consumption rate distributions reflect actual rates of consumption in the subpopulations of interest.

Another related and potentially influential variable in the modeling approach is the assumed conversion factor for mercury concentrations between uncooked and cooked fish. Studies have found that cooking fish tends to reduce the overall weight of fish by approximately one-third (Great Lakes Sport Fish Advisory Task Force, 1993) without affecting the overall amount of mercury. But these conversion rates depend on cooking practices and types of fish. Uncertainty regarding this conversion factor also has a proportionate effect on the modeling results.

Measurement and Valuation of IQ Related Effects

The models for estimating and valuing IQ effects involve three main steps. The first step is translating maternal mercury ingestion rates to mercury levels in hair. The second step is translating differences in hair mercury concentrations during pregnancy to IQ changes in offspring. The third step is translating IQ losses into expected reductions in lifetime earnings. As discussed below, each of these steps also involves the following assumptions, limitations, and uncertainties:.

- The conversion of mercury ingestion rate to mercury concentration in hair is based on uncertainty analysis of a toxicokinetic model for estimating reference dose (Swartout and Rice, 2000). The conversion factor was estimated by considering the variability and uncertainty in various inputs used in deriving the dose including body weight, hair-to-blood mercury ratio, half-life of MeHg in blood, and others. Therefore, there is uncertainty regarding the conversion factor between hair mercury concentration and mercury ingestion rate. Although, the median conversion factor (0.08 μg/kg-day/hair-ppm) is used, the 90% confidence interval is from 0.037 to 0.16 μg/kg day/hair-ppm. Any change in the conversion factor will proportionately affect the benefits results because of the linearity of the model.

- The dose-response model used to estimate neurological effects on children because of maternal mercury body burden is susceptible to various uncertainties. In particular, there are three main concerns. First, there are other cognitive end-points that have stronger association with MeHg than IQ point losses. Therefore, using IQ points as a primary end point in the benefits assessment may underestimate the impacts. Second,

blood-to-hair ratio for mercury is uncertain, which can cause the results from analyses based on mercury concentration in blood to be uncertain. Third, uncertainty is associated with the epidemiological studies used in deriving the dose-response models.

- With regard to the relationship between prenatal methylmercury exposure and childhood IQ loss, we expect greater uncertainty in associated estimates of IQ loss as exposure levels increase beyond those observed in the primary studies (i.e., Faroe Islands, New Zealand, Seychelles Islands studies) used to derive the dose-response function. In particular, high-end total exposure estimates for some of the subsistence-level fishing subpopulations included in this assessment likely exceed levels observed in the three primary studies.

- To parameterize the dose-response relationship between maternal hair concentrations and IQ loss for this analysis, we applied the results of an integrative study by Axelrad et al. (2007). The implications of applying this study include the following:

    - This approach may confound potentially positive cognitive effects of fish consumption and, more specifically, omega-3 fatty acids. Results from Rice (2010) offer a reasonable, but highly uncertain, estimate for offsetting the possible downward bias resulting from the positive confounding effects of fatty acids. Rice's high coefficient reflects the central estimate of Axelrad but adjusted upwards by a factor of 1.5 to "acknowledge the recent argument of Budtz-Jorgensen (2007) that the parameter estimates from these three epidemiological studies (Faroe Islands, Seychelles Islands, New Zealand) may be biased downward by a factor of approximately 2 because of failure to adequately control for confounding." A third study, Oken (2008), analyzes a cohort in Massachusetts and also seems to support a higher "Axelrad-plus" coefficient range due to evidence of fatty acid confounding (i.e., positive cognitive effects of fatty acids in fish may have previously led to underestimates of mercury-attributable IQ loss). This study offers further qualitative support for a higher-end estimate but is limited by the fact that it did not control for the children's home environment, which is generally a significant factor in early cognitive development.

    - The dose-response coefficient from the Axelrad et al. study is sensitive to the exclusion of one outlier data point from the Seychelles study. Including the outlier would reduce the effect size by about 25 percent. If this outlier actually reflects

the true response for a subset of the populations, then risks (as modeled) could be biased high specifically for this subpopulation

- Because the dose-response coefficient is applied uniformly across the entire exposed population and is a multiplicative factor in the model, the uncertainty in this parameter has a directly proportional effect on the reported risk and benefit estimates. In other words, adjusting the absolute value of the dose-response coefficient upward by a factor of 1.5 (i.e., based on Rice, 2010) would yield reductions in IQ losses and benefits from mercury emission reductions that are also greater by a factor of 1.5.

- The valuation of IQ losses is based on a unit-value approach developed by EPA, which estimates that the average effect of a 1-point reduction in IQ is to reduce the present value of net future earnings. Three key assumptions of this unit-value approach are that (1) there is a linear relationship between IQ changes and net earnings losses, (2) the unit value applies to even very small changes in IQ, and (3) the unit value will remain constant (in real present value terms) for several years into the future. Each of these assumptions contributes to uncertainty in the result. In particular the unit value estimate is itself subject to two main sources of uncertainty.

  - The first source is directly related to uncertainties regarding the average reductions in future earnings and years in school as a result of IQ changes. The average percentage change estimates are subject to statistical error, modeling uncertainties, and variability across the population. To address these uncertainties we have included in the analysis and reported results a range of values for this parameter, based on statistical analyses by Salkever (1995) and Schwartz (1994).

  - The second main source of uncertainty is the estimates of average lifetime earnings and costs of schooling. Both of these estimates are derived from national statistics from the early 1990s, but they are also subject to statistical error, modeling uncertainties, and variability across the population. It is also worth noting that the lost future earnings estimates do not include present value estimates for nonwage/nonsalary earnings (i.e., fringe benefits) and household (nonmarket) production. Based on the results of Grosse et al. (2009), including these factors would increase the present value of median earnings (both explicit and implicit) by a factor of roughly 1.9. However, it is not known whether IQ changes have a similar effect on these other (implicit) earnings.

Unquantified Benefits

In addition to the uncertainties discussed above associated with the benefit analysis of reducing exposures to MeHg from recreational freshwater angling, we are unable to quantify several additional benefits, which adds to the uncertainties in the final estimate of benefits.

Table 5-20 displays the health and ecosystem effects associated with MeHg exposure that are discussed in Section 5.22 for which we are currently unable to quantify. We note that specifically with regard to health effects, the NRC (2000) provided the following observation: "Neurodevelopmental effects are the most extensively studied sensitive end point for MeHg exposure, but there remains some uncertainty about the possibility of other health effects at low levels of exposure. In particular, there are indications of immune and cardiovascular effects, as well as neurological effects emerging later in life, that have not been adequately studied."

Table 5-20. Unquantified Health and Ecosystem Effects Associated with Exposure to Mercury

Category of Health or Ecosystem Effect	Potential Health or Ecosystem Outcomes
Neurologic Effects	Impaired cognitive development Problems with language Abnormal social development
Cardiovascular Effects*	Potential for fatal and non-fatal AMI (heart attacks)
Genotoxic Effects*	Association with genetic effects
Immunotoxic Effects*	Possible autoimmunity effects in antibodies
Ecological Effects*	Neurological effects in wildlife (birds, fish, and mammals) that is similar to humans

*These are potential effects and are not quantified because the literature is either contradictory or incomplete.

In addition to the health and ecosystem effects that we are not able to quantify, we are currently unable to quantify exposures to other segments of the U.S. population including consumption of commercial seafood and freshwater fish (produced domestically as well as imported from foreign sources) and consumption of recreationally caught seafood from estuaries, coastal waters, and the deep ocean. These consumption pathways impact additional recreational anglers who are not modeled in our benefits analysis as well as the general U.S. population. Reductions in domestic fish tissue concentrations can also impact the health of foreign consumers (consuming U.S. exports). Because of technical/theoretical limitations in the science, EPA is unable to quantify the benefits associated with several of these fish consumption pathways. For example, reductions in U.S. power plant emissions will result in a lowering of the global burden of elemental mercury, which will likely produce some degree of reduction in

mercury concentrations for fish sourced from the open ocean and freshwater and estuarine waterbodies in foreign countries. In the case of mercury reductions for fish in the open ocean, complexities associated with modeling the linkage between changes in air deposition of mercury and reductions in biomagnification and bioaccumulation up the food chain (including open ocean dilution and the extensive migration patterns of certain high-consumption fish such as tuna) prevent the modeling of fish obtained from the open ocean. In the case of commercial fish obtained from foreign freshwater and estuarine waterbodies, although technical challenges are associated with modeling long-range transport of elemental mercury and the subsequent impacts to fish in these distant locations, additional complexities such as accurately modeling patterns of harvesting and their linkages to commercial consumption in the United States prevent inclusion of foreign-sourced freshwater and estuarine fish in the primary benefits analysis.

Finally, with regard to commercially-produced freshwater fish sourced in the United States (i.e., fish from catfish, bass, and trout farms), we are unable to accurately quantify effects from this consumption pathway because many of the fish farms operating in the United States use feed that is not part of the aquatic food web of the waterbody containing the fish farm (e.g., use of agricultural-based supplemental feed). In addition, many of the farms involve artificial "constructed" waterbody environments that are atypical of aquatic environments found in the regions where those farms are located, thereby limiting the applicability of Mercury Maps' assumption in linking changes to mercury deposition to changes in mercury fish tissue concentrations (e.g., waterbodies may have restricted or absent watersheds and modified aquatic chemistry, which can effect methylation rates and impact time scales for reaching steady-state mercury fish tissue concentrations following reductions in mercury deposition). Some research indicates that the recycling of water at fish farms can magnify the mercury concentration because the system does not remove mercury as it is recycled, while newly deposited mercury is added to the system. Thus, additional research on aquaculture farms is necessary before a benefits analysis can be conducted.

Exclusion of these commercial pathways means that this benefits analysis, although covering an important source of exposure to domestic mercury emissions (recreational freshwater anglers), excludes a large and potentially important group of individuals. Recreational freshwater consumption accounts for approximately 10 to 17% of total U.S. fish consumption, and 90% is derived from commercial sources (domestic seafood, aquaculture, and imports) (EPA, 2005).

In conclusion, several unquantified benefits associated with this analysis add to the overall uncertainty in estimating total benefits. To the extent that the proposed rule will reduce

mercury deposition from power plants over estuarine areas, coastal, and open ocean waters, there would be a subsequent reduction in mercury fish tissue concentrations in these different waterbodies and an associated benefit from avoided decrements in IQ and other known health and ecosystem effects.

5.9.6 Overall Conclusions

Total Baseline Incidence of IQ Loss: Self-Caught Fish Consumption among Recreational Freshwater Anglers

- Out of 64,500 census tracts in the continental U.S., 63,978 are located within 100 miles of at least one HUC-12 watershed with freshwater mercury fish tissue sampling data, and therefore were included in the modeling of IQ loss among recreational freshwater anglers.

- Approximately 240,000 prenatally exposed children were modeled, with an average IQ loss of 0.11 and 0.10 IQ points, respectively, from self-caught freshwater fish consumption for the 2005 and 2016 base case scenarios.

- The highest estimated state-specific average IQ loss among children of freshwater recreational anglers is 0.21 IQ points under the 2005 base case scenario, in both California and Rhode Island.

- Total estimated IQ loss from self-caught freshwater fish consumption among children of recreational anglers is estimated at 25,555 and 24,419 IQ points, respectively, for the 2005 and 2016 base case scenarios.

- The present economic value of baseline IQ loss for 2005 ranges from $204.7 million to $302.9 million, assuming a 3% discount rate, and from $22.8 million to $50 million, assuming a 7% discount rate.

- The present economic value of baseline IQ loss for 2016 ranges from $195.7 million to $289.6 million, assuming a 3% discount rate, and from $21.8 million to $47.8 million, assuming a 7% discount rate.

Avoided IQ Loss and Economic Benefits due to Regulatory Action: Self-Caught Fish Consumption among Recreational Freshwater Anglers

- Eliminating all mercury air emissions from U.S. EGUs in 2016 would result in an estimated 0.00893 fewer IQ points lost per prenatally exposed child from self-caught freshwater fish consumption, as compared with the 2005 base case scenario.

- The present economic value of avoided IQ loss from eliminating all mercury air emissions from U.S. EGUs in 2016 is estimated at a range of $5.6 million to $8.3 million, assuming a 3% discount rate, and $0.6 million to $1.4 million, assuming a 7% discount rate.

- Reduced mercury air emissions due to implementation of the Toxics Rule in 2016 would result in an estimated 0.00209 fewer IQ points lost per prenatally exposed child from self-caught freshwater fish consumption, as compared with the 2016 base case scenario.

- The present economic value of avoided IQ loss from reduced mercury air emissions due to implementation of the Toxics Rule in 2016 is estimated at a range of $4.1 million to $6.1 million, assuming a 3% discount rate, and $0.5 million to $1 million, assuming a 7% discount rate.

Risk (IQ loss) Associated with Self-Caught Freshwater Fish Consumption among Selected High-Risk Subpopulations[1]

- Low-Income African-American Subsistence-Level Fishers in the Southeast
    - In the 2016 base case scenario, the median IQ loss is estimated to be 1.87 IQ points and the 95th percentile is 11.56 IQ points per exposed individual.
    - The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.032 IQ points, and the 95th percentile is 0.274 IQ points.
- Low-Income White Subsistence-Level Fishers in the Southeast
    - In the 2016 base case scenario, the median IQ loss is estimated to be 0.19 IQ points and the 95th percentile is 2.46 IQ points per exposed individual.
    - The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.002 IQ points, and the 95th percentile is 0.045 IQ points.
- Low-Income Female Subsistence-Level Fishers
    - In the 2016 base case scenario, the median IQ loss is estimated to be 0.11 IQ points and the 95th percentile is 3.12 IQ points per exposed individual.
    - The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.001 IQ points, and the 95th percentile is 0.053 IQ points.
- Hispanic Subsistence-Level Fisher

[1] We do note that overall confidence in IQ loss estimates above approximately 7 points decreases because we begin to apply the underlying IQ loss function at exposure levels (ppm hair levels) above those reflected in epidemiological studies used to derive those functions. The 39.1 ppm was the highest measured ppm level in the Faroes Island study, while ~86 was the highest value in the New Zealand study (USEPA, 2005) (a 7 IQ points loss is approximately associated with a 40 ppm hair level given the concentration-response function we are using).

- In the 2016 base case scenario, the median IQ loss is estimated to be 0.18 IQ points and the 95th percentile is 2.94 IQ points per exposed individual.
- The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.001 IQ points, and the 95th percentile is 0.030 IQ points.

- Laotian Subsistence-Level Fishers
  - In the 2016 base case scenario, the median IQ loss is estimated to be 0.20 IQ points and the 95th percentile is 4.26 IQ points per exposed individual.
  - The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.001 IQ points, and the 95th percentile is 0.047 IQ points.

- Chippewa Tribal Members in the Great Lakes Area
  - In the 2016 base case scenario, the median IQ loss is estimated to be 0.40 IQ points and the 95th percentile is 3.28 IQ points per exposed individual.
  - The median reduction in IQ loss resulting from the 2016 Toxics Rule (relative to the 2016 base case scenario) is 0.005 IQ points, and the 95th percentile is 0.041 IQ points.

5.10 Benefits Associated with Reductions in Other HAP than Mercury

5.10.1 Hazards

Emissions data collected during development of this proposed rule show that HCl emissions represent the predominant HAP emitted by industrial boilers. Coal- and oil-fired EGUs emit lesser amounts of HF, chlorine, metals (As, Cd, Cr, Hg, Mn, Ni, and Pb), and organic HAP emissions. Although numerous organic HAP may be emitted from coal- and oil-fired EGUs, only a few account for essentially all the mass of organic HAP emissions. These organic HAP are formaldehyde, benzene, and acetaldehyde.

Exposure to high levels of these HAP is associated with a variety of adverse health effects. These adverse health effects include chronic health disorders (e.g., irritation of the lung, skin, and mucus membranes, effects on the central nervous system, and damage to the kidneys), and acute health disorders (e.g., lung irritation and congestion, alimentary effects such as nausea and vomiting, and effects on the kidney and central nervous system). We have classified three of the HAP as human carcinogens and five as probable human carcinogens. The following sections briefly discuss the main health effects information we have regarding the key HAPs emitted by EGUs.

Acetaldehyde

Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen, based on nasal tumors in rats, and is considered toxic by the inhalation, oral, and intravenous routes.[1] Acetaldehyde is reasonably anticipated to be a human carcinogen by the U.S. Department of Health and Human Services (DHHS) in the 11th Report on Carcinogens and is classified as possibly carcinogenic to humans (Group 2B) by the IARC.[2,3] The primary noncancer effects of exposure to acetaldehyde vapors include irritation of the eyes, skin, and respiratory tract.[4]

Arsenic

Arsenic, a naturally occurring element, is found throughout the environment and is considered toxic through the oral, inhalation and dermal routes. Acute (short-term) high-level inhalation exposure to As dust or fumes has resulted in gastrointestinal effects (nausea, diarrhea, abdominal pain, and gastrointestinal hemorrhage); central and peripheral nervous system disorders have occurred in workers acutely exposed to inorganic As. Chronic (long-term) inhalation exposure to inorganic As in humans is associated with irritation of the skin and mucous membranes. Chronic inhalation can also lead to conjunctivitis, irritation of the throat and respiratory tract and perforation of the nasal septum.[5] Chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage in humans. Inorganic As exposure in humans, by the inhalation route, has been shown to be strongly associated with lung cancer, while ingestion of inorganic As in humans has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. EPA has classified inorganic As a Group A, human carcinogen.[6]

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[1] U.S. Environmental Protection Agency (U.S. EPA). 1991. Integrated Risk Information System File of Acetaldehyde. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at http://www.epa.gov/iris/subst/0290.htm.

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

[3] International Agency for Research on Cancer (IARC). 1999. Re-evaluation of some organic chemicals, hydrazine, and hydrogen peroxide. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemical to Humans, Vol 71. Lyon, France.

[4] U.S. Environmental Protection Agency (U.S. EPA). *1991. Integrated Risk Information System File of* Acetaldehyde. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at http://www.epa.gov/iris/subst/0290.htm.

[5] Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Arsenic. Atlanta, GA: U.S. Department of Health and Human Services. Available on the Internet at < http://www.atsdr.cdc.gov/mhmi/mmg168.html#bookmark02>

[6] U.S. Environmental Protection Agency (U.S. EPA). 1998. Integrated Risk Information System File for Arsenic. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at: *http://www.epa.gov/iris/subst/0278.htm.*

Benzene

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.[1,2,3] EPA states in its IRIS database that data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. The IARC has determined that benzene is a human carcinogen and the DHHS has characterized benzene as a known human carcinogen.[4,5]

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.[6,7]

Cadmium

Breathing air with lower levels of Cd over long periods of time (for years) results in a build-up of Cd in the kidney, and if sufficiently high, may result in kidney disease. Lung cancer has been found in some studies of workers exposed to Cd in the air and studies of rats that inhaled Cd. The U.S. DHHS has determined that Cd and Cd compounds are known human carcinogens. The IARC has determined that Cd is carcinogenic to humans. EPA has determined that Cd is a probable human carcinogen.[8]

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[1] U.S. Environmental Protection Agency (U.S. EPA). 2000. Integrated Risk Information System File for Benzene. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at: http://www.epa.gov/iris/subst/0276.htm.

[2] 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.

[3] 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.

[4] 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.

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

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

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

[8] Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Public Health Statement for Cadmium. CAS# 1306-19-0. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at <http://www.atsdr.cdc.gov/PHS/PHS.asp?id=46&tid=15>.

Chlorine

The acute (short term) toxic effects of Cl_2 are primarily due to its corrosive properties. Chlorine is a strong oxidant that upon contact with water moist tissue (e.g., eyes, skin, and upper respiratory tract) can produce major tissue damage.[1] Chronic inhalation exposure to low concentrations of Cl_2 (1 to 10 parts per million, ppm) may cause eye and nasal irritation, sore throat, and coughing. Chronic exposure to Cl_2, usually in the workplace, has been reported to cause corrosion of the teeth. Inhalation of higher concentrations of Cl_2 gas (greater than 15 ppm) can rapidly lead to respiratory distress with airway constriction and accumulation of fluid in the lungs (pulmonary edema). Exposed individuals may have immediate onset of rapid breathing, blue discoloration of the skin, wheezing, rales or hemoptysis (coughing up blood or blood-stain sputum). Intoxication with high concentrations of Cl_2 may induce lung collapse. Exposure to Cl_2 can lead to reactive airways dysfunction syndrome (RADS), a chemical irritant-induced type of asthma. Dermal exposure to Cl_2 may cause irritation, burns, inflammation and blisters. EPA has not classified Cl_2 with respect to carcinogenicity.

Chromium

Chromium may be emitted in two forms, trivalent Cr (Cr^{+3}) or hexavalent Cr (Cr^{+6}). The respiratory tract is the major target organ for Cr^{+6} toxicity, for acute and chronic inhalation exposures. Shortness of breath, coughing, and wheezing have been reported from acute exposure to Cr^{+6}, while perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, and other respiratory effects have been noted from chronic exposures. Limited human studies suggest that Cr^{+6} inhalation exposure may be associated with complications during pregnancy and childbirth, but there are no supporting data from animal studies reporting reproductive effects from inhalation exposure to Cr^{+6}. Human and animal studies have clearly established the carcinogenic potential of Cr^{+6} by the inhalation route, resulting in an increased risk of lung cancer. EPA has classified Cr^{+6} as a Group A, human carcinogen. Trivalent Cr is less toxic than Cr^{+6}. The respiratory tract is also the major target organ for Cr^{+3} toxicity, similar to Cr^{+6}. EPA has not classified Cr^{+3} with respect to carcinogenicity.

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[1] Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Chlorine. Atlanta, GA: U.S. Department of Health and Human Services. http://www.atsdr.cdc.gov/mmg/mmg.asp?id=198&tid=36.

Formaldehyde

Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[1] EPA is currently reviewing recently published epidemiological data. 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."[2] 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.[3,4]

Hydrogen Chloride

Hydrogen chloride is a corrosive gas that can cause irritation of 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.[5] 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. Exposure to HCl can lead to RADS,

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[1] U.S. EPA. 1987. Assessment of Health Risks to Garment Workers and Certain Home Residents from Exposure to Formaldehyde, Office of Pesticides and Toxic Substances, April 1987.

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

3 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

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

[5] Agency for Toxic Substances and Disease Registry (ATSDR). Medical Management Guidelines for Hydrogen Chloride. Atlanta, GA: U.S. Department of Health and Human Services. Available online at http://www.atsdr.cdc.gov/mmg/mmg.asp?id=758&tid=147#bookmark02.

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 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.[1]

Hydrogen Fluoride

Acute (short-term) inhalation exposure to gaseous HF can cause severe respiratory damage in humans, including severe irritation and pulmonary edema. Chronic (long-term) oral exposure to fluoride at low levels has a beneficial effect of dental cavity prevention and may also be useful for the treatment of osteoporosis. Exposure to higher levels of fluoride may cause dental fluorosis. One study reported menstrual irregularities in women occupationally exposed to fluoride via inhalation. The EPA has not classified HF for carcinogenicity[2].

Lead

The main target for Pb toxicity is the nervous system, both in adults and children. Long-term exposure of adults to Pb at work has resulted in decreased performance in some tests that measure functions of the nervous system. Lead exposure may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people. Lead exposure may also cause anemia.

Children are more sensitive to the health effects of Pb than adults. No safe blood Pb level in children has been determined. At lower levels of exposure, Pb can affect a child's mental and physical growth. Fetuses exposed to Pb in the womb may be born prematurely and have lower weights at birth. Exposure in the womb, in infancy, or in early childhood also may slow mental development and cause lower intelligence later in childhood. There is evidence that these effects may persist beyond childhood.[3]

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[1] U.S. Environmental Protection Agency (U.S. EPA). 1995. Integrated Risk Information System File of Hydrogen Chloride. Research and Development, National Center for Environmental Assessment, Washington, DC. This material is available electronically at http://www.epa.gov/iris/subst/0396.htm.

[2] U.S. Environmental Protection Agency. Health Issue Assessment: Summary Review of Health Effects Associated with Hydrogen Fluoride and Related Compounds. EPA/600/8-89/002F. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, Cincinnati, OH. 1989.

[3] Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Public Health Statement for Lead. CAS#: 7439-92-1. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available on the Internet at < http://www.atsdr.cdc.gov/ToxProfiles/phs13.html>.

There are insufficient data from epidemiologic studies alone to conclude that Pb causes cancer (is carcinogenic) in humans. The DHHS has determined that Pb and Pb compounds are reasonably anticipated to be human carcinogens based on limited evidence from studies in humans and sufficient evidence from animal studies, and the EPA has determined that Pb is a probable human carcinogen.

xi. Manganese

Health effects in humans have been associated with both deficiencies and excess intakes of Mn. Chronic exposure to high levels of Mn by inhalation in humans results primarily in central nervous system effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically-exposed workers. Manganism, characterized by feelings of weakness and lethargy, tremors, a masklike face, and psychological disturbances, may result from chronic exposure to higher levels. Impotence and loss of libido have been noted in male workers afflicted with manganism attributed to inhalation exposures. The EPA has classified Mn in Group D, not classifiable as to carcinogenicity in humans[1].

Nickel

Respiratory effects have been reported in humans from inhalation exposure to Ni. No information is available regarding the reproductive or developmental effects of Ni in humans, but animal studies have reported such effects. Human and animal studies have reported an increased risk of lung and nasal cancers from exposure to Ni refinery dusts and nickel subsulfide. The EPA has classified nickel subsulfide as a human carcinogen and nickel carbonyl as a probable human carcinogen[2,3]. The IARC has classified Ni compounds as carcinogenic to humans[4].

Selenium

Acute exposure to elemental Se, hydrogen selenide, and selenium dioxide (SeO_2) by inhalation results primarily in respiratory effects, such as irritation of the mucous membranes, pulmonary edema, severe bronchitis, and bronchial pneumonia. One Se compound, selenium sulfide, is carcinogenic in animals exposed orally. EPA has classified elemental Se as a Group

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[1] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Manganese. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[2] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Nickel Subsulfide. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[3] U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) on Nickel Carbonyl. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.

[4] Nickel (IARC Summary & Evaluation , Volume 49, 1990), http://www.inchem.org/documents/iarc/vol49/nickel.html

D, not classifiable as to human carcinogenicity, and selenium sulfide as a Group B2, probable human carcinogen.

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APPENDIX B.
ANALYSIS OF TRIP TRAVEL DISTANCE FOR RECREATIONAL FRESHWATER ANGLERS

As described in Section 5.7.7, the method used to estimate exposures to mercury in freshwater fish requires information about how far individuals typically travel for freshwater fishing. This appendix describes the data and methods used to analyze travel distance patterns by freshwater anglers, and it reports the results that were used to estimate exposures.

B.1 Data

To conduct an analysis of trip travel distance for freshwater anglers, we used data from the NSRE 1994. As described previously, this 16,000-person survey elicited information on water-based recreation activities—specifically boating, fishing, swimming, and wildlife viewing—during the previous year. Respondents were asked about *their most recent trip* taken in each of the four categories. Of particular interest to this analysis is data concerning fishing trip characteristics for all respondents who fished in freshwater bodies during the previous year. Of the 3,220 respondents who had reported fishing, 2,482 visited either a lake, pond, river, or stream on their most recent trip.

The fishing module elicited location information about most recent fishing trip taken during the preceding 12 months. This trip was recorded as either a single- or multiday trip to a specific water body ("site") identified by the respondent. Subsequently, a series of questions were asked to gather location data on the specific site visited, including the site name, the state in which the site was located, and the name of the city or town nearest the site. To identify potential determinants of travel distance for a freshwater fishing trip, we analyzed the 2,384 available responses to the following survey question: "What was the one way travel distance, in miles from your home, to your destination on *site*?" Table B-1 presents summary statistics for travel distance, which are reported separately for single-day, multiday, and aggregated trips. As would be expected, median travel distance varied according to trip type, from 20 miles for a single-day trip to almost 140 miles for a multiday trip. Across both trip types, the average travel distance was slightly less than 100 miles.

Table B-1. Reported Trip Travel Distance for Freshwater Anglers (miles)

	N	Mina	P5	P25	P50	Mean	P75	P95	Max
All trip types	2384	0	2	10	20	91.9	45	125	3000
Single-day trips only	1791	0	2	10	20	41	45	125	1100
Multiday trips only	586	3	18	70	138	248.2	300	850	3000

[a] Seven respondents reported traveling 0 miles for their most recent trip; all were described as single-day trips.

Note: Ninety-eight respondents who visited freshwater bodies on their most recent fishing trip did not report the travel distance.

B.2 Analysis of Travel Distance Data

The influence of multiple demographic characteristics on travel distance was tested using multivariate regression analysis. Table B-2 reports descriptive statistics for the anglers included in this analysis. As indicated by the table, over 90 percent of the sample is white; males comprise a higher percentage of the sample (62 percent) than females. More than half the sample had completed at least some college and three-fourths of the sample reported being employed. The survey asked respondents to classify their place of residence as either rural, suburban, or urban. Approximately 40 percent described their area as rural, 37 percent as suburban, and 23 percent as urban. Respondents were assigned to a U.S. Census geographic region by matching their zip code to a corresponding state. The states were then aggregated to the appropriate Census region (http://www.census.gov/geo/www/us_regdiv.pdf). The majority of respondents resided in the South and Midwest, followed by the West and Northeast.

Table B-2. Demographic Characteristics of Freshwater Anglers[a]

	N	Frequency	
Gender	2267	62%	Male
Race	2250	91%	White
		4%	Black
		2%	Hispanic
		2%	Other
Education	2262	11%	Less than high school degree
		34%	High school degree/equivalent
		55%	Some college or more
Work status	2263	75%	Employed
Geography	2237	23%	Urban
		37%	Suburban
		41%	Rural
Region	2205	13%	Northeast
		33%	South
		31%	Midwest
		23%	West

[a] In total, 2,384 respondents reported information on trip travel distance to a freshwater destination.

Note: Values may not add to 100 percent due to rounding.

Table B-3 presents additional characteristics on the demographic distribution of the sample. The average age of respondents was 38 years, while household size averaged approximately three members, with less than one person under the age of six. Respondents' average weekly leisure time was 28 hours. However, this varied significantly across the sample, from zero to 168 hours. In the survey, family income is reported as a categorical variable, with respondents selecting the income range that reflected family income in the previous year. The midpoint of this range was taken to produce a continuous income variable. Subsequently, this value was converted to (2000$) using the consumer price index. Median (mean) income was estimated to be $57,325 ($66,496) annually.

Table B-3. Demographic Characteristics of Freshwater Anglers

	N	Mean	SD	Min	Max
Age	2245	38.4	14.5	16	92
Household size	2255	3.1	1.5	1	10
'rs	2270	0.3	0.7	1	5
yrs	2254	2.2	0.9	0	7
Weekly leisure time (hrs)	2025	27.7	23.9	0	168
Family income (2000$)	1851	66496	57324	8938	208547

Multivariate regression analysis was used to identify determinants of travel distance to freshwater fishing sites. The dependent variable in this analysis was the miles traveled to the most recent freshwater fishing site. The explanatory variables included several demographic and geographic characteristics of the respondents.

Separate regressions were conducted for the full sample (1), single-day trips only (2), and multiday trips only (3). The results are reported in Table B-4. Family income was estimated to have a positive and highly significant effect in all three models. Dummy variables for urban and suburban location were also found to have positive and highly significant effects in all models. These results suggest that wealthier anglers and those living in or near metropolitan areas tend to travel further to fishing sites, relative to less-wealthy anglers and those living in rural areas. In models (1) and (2) dummy variables for the Midwest and West regions also had positive and highly significant effects on trip travel distance, relative to the South region. The Northeast region did not have a statistically significant effect on distance traveled. Education was estimated to be positively and significantly related to distance traveled in the first and second models. (Note that the respondent's level of education, recorded in the survey as a categorical variable, was recoded as a continuous variable for the regression analysis.) Neither age, race, nor gender had significant effects (at a 5 percent level) on travel distance in any of the models.

B.3 Summary Results Applied in the Population Centroid Approach

Given the high significance of geographic area and family income across the regressions, nonparametric results (frequency distributions) were generated for four mutually exclusive subgroups of respondents and five travel distance categories. The results are reported in Table B-5. Respondents were categorized into the four following groups:

- G1: family income ≥$50,000 (in 2000 dollars) and urban or suburban resident
  - (N = 452 for single-day trips)
  - (N = 649 for single- and multiday trips)
- G2: family income ≤$50,000 and urban or suburban resident
  - (N = 329 for single-day trips)
  - (N = 417 for single- and multiday trips)
- G3: family income ≥$50,000 and rural resident
  - (N = 295 for single-day trips
  - (N = 376 for single- and multiday trips)
- G4: family income ≤$50,000 and rural resident
  - (N = 309 for single-day trips)
  - (N = 386 for single- and multiday trips)

Table B-4. OLS Regression Results for Determinants of Reported Trip Travel Distance (miles)

Variable Description	(1) Full Sample (both single- and multiday trips) Coefficient	t-stat	(2) Single-Day Trips Only Coefficient	t-stat	(3) Multiday Trips Only Coefficient	t-stat
CONSTANT	0.6966	1.54	1.7954	3.89**	2.2493	3.26**
AGE	0.0044	1.83*	0.0011	0.44	0.001	0.28
GENDER	0.0572	0.83	0.0173	0.25	0.1446	1.39
EDUC	0.1729	2.48**	0.1552	2.21**	0.128	1.22
MINORITY	–0.0437	–0.36	0.0228	0.19	–0.1391	–0.76
FAMILY INCOME (log)	0.187	4.41**	0.0827	1.92*	0.1759	2.78**
URBAN	0.3491	3.95**	0.2799	3.12**	0.2121	1.62*
SUBURBAN	0.3422	4.48**	0.193	2.50**	0.4298	3.67**
NEAST	–0.0387	–0.36	–0.2549	–2.42**	0.1525	0.89
MIDWEST	0.3856	4.65**	0.1	1.21	0.4923	3.63**
WEST	0.6103	6.73**	0.3374	3.59**	0.3239	2.32**
	R^2 = 0.077 N = 1,798		R^2 = 0.041 N = 1,360		R^2 = 0.112 N = 434	

** = significant at 5 percent level.

* = significant at 10 percent level.

Table B-5. Travel Distance Frequencies by Demographic Group (Percentage in each distance category)

Travel Distance (mi)	(G1) High-Income and Urban/Suburban Resident	(G2) Low-Income and Urban/Suburban Resident	(G3) High-Income and Rural Resident	(G4) Low-Income and Rural Resident
Single-day trips only (N = 1,385)				
	(N = 452)	(N = 329)	(N = 295)	(N = 309)
Distance ≤10 mi	23%	32%	31%	34%
>10 mi to 20 mi	18%	23%	22%	24%
>20 mi to 50 mi	31%	20%	28%	26%
>50 mi to 100 mi	17%	19%	14%	11%
Distance >100 mi	11%	6%	5%	5%
Full sample (both single- and multiday trips) (N = 1,828)				
	(N = 649)	(N = 417)	(N = 376)	(N = 386)
Distance ≤10 mi	16%	26%	24%	29%
>10 mi to 20 mi	13%	18%	18%	21%
>20 mi to 50 mi	24%	18%	25%	25%
>50 mi to 100 mi	19%	19%	16%	14%
Distance >100 mi	27%	18%	17%	11%

These categories were selected because they match categories that can be easily identified in Census data and because they split the sample into roughly similar group sizes. Travel distance was categorized into ranges reported in the first column of Table B-5. The results are consistent with those generated from the regression analysis. Among respondents on single-day trips, the number that traveled longer distances (greater than 100 miles) increased from the low-income rural cohort (5 percent) to the higher-income urban/suburban cohort (11 percent). The

same pattern holds for those taking either a single- or multiday trip. The number traveling longer distances more than doubled, from 11 percent among low-income rural respondents to 27 percent among high-income urban/suburban respondents. These results indicate higher-income urban/suburban anglers travel greater distances to freshwater destinations than lower-income urban/suburban anglers and rural anglers.

As described in Section 5.7, the trip frequency estimates reported in Table B-5 for the full sample were used in the population centroid approach to weight exposures to mercury in fish according to distance from the Census tract centroid, income levels in the tract, and whether the tract is predominantly rural or urban/suburban.

Chapter 6
CO-BENEFITS ANALYSIS AND RESULTS

Synopsis

This chapter contains a subset of the criteria-pollutant related health and welfare expected to occur as a co-benefit of the proposed Toxics Rule in 2016. This rule is expected to yield significant reductions in SO_2 and NO_x from EGUs, which in turn would lower overall ambient levels of $PM_{2.5}$ across much of the eastern U.S. In this chapter we quantify the health and welfare co-benefits resulting from these air quality improvements.

We estimate the monetized co-benefits of the proposed remedy to be $59 billion to $140 billion at a 3% discount rate and $53 billion to $120 billion at a 7% discount rate in 2016. All estimates are in 2007$. This co-benefits analysis accounts for both decreases and increases in emissions across the country resulting from aspects of the proposed provisions of the rule from reductions in SO_2, NOx and directly emitted $PM_{2.5}$. These estimates omit the benefits from several important categories, including ecosystem benefits and the direct health benefits from reducing exposure to tropospheric Ozone, NO_2 and SO_2 due to time constraints.

6.1 Overview

This chapter contains a subset of the estimated health and welfare co-benefits of the proposed Toxics Rule in 2016. The Toxics Rule is expected to yield significant net reductions in SO_2 and NO_x from EGUs, which in turn would lower overall ambient levels of $PM_{2.5}$ and ozone across much of the eastern U.S. The analysis in this chapter aims to characterize the benefits of these air quality changes by answering two key questions:

1. What are the health and welfare effects of changes in ambient particulate matter ($PM_{2.5}$) resulting from reductions in precursors including NO_x SO_2 and directly-emitted $PM_{2.5}$?
2. What is the economic value of these effects?

In this analysis we consider an array of health and welfare impacts attributable to changes in $PM_{2.5}$ air quality. The 2009 $PM_{2.5}$ Integrated Science Assessment (U.S. EPA, 2009d) identify the human health effects associated with these ambient pollutants, which include premature mortality and a variety of morbidity effects associated with acute and chronic exposures. PM welfare effects include visibility impairment and materials damage. NO_x welfare effects include aquatic and terrestrial acidification and nutrient enrichment (U.S. EPA, 2008f). SO_2 welfare effects include aquatic and terrestrial acidification and increased mercury methylation (U.S. EPA, 2008f). Though models exist for quantifying these ecosystem impacts, time and resource constraints precluded us from quantifying most of those effects in this analysis.

Table 6-1 summarizes the total monetized co-benefits of the proposed rule in 2016. This table reflects the economic value of the change in $PM_{2.5}$-related human health impacts and the monetized value of CO_2 reductions occurring as a result of the proposed Toxics Rule.

Table 6-2 summarizes the human health and welfare benefits categories contained within the primary benefits estimate, those categories that were unquantified due to limited data or time.

Table 6-1. Estimated monetized co-benefits of the proposed Toxics Rule(billions of 2007$)[a]

Benefits Estimate	*Eastern U.S.* [B]	*Western U.S.*	*Total*
Pope et al. (2002) $PM_{2.5}$ mortality estimate			
Using a 3% discount rate	$55 +B ($4.4—$170)	$1 +B ($0. 1—$3.1)	$57+B ($4.5—$170)
Using a 7% discount rate	$51 +B ($4.1—$160)	$0.9 +B ($0.1—$2.8)	$52 +B ($4.1—$160)
Laden et al. (2006) $PM_{2.5}$ mortality estimate			
Using a 3% discount rate	$140 +B ($12—$390)	$2.5 +B ($0.2—$7.2)	$140 +B ($12—$400)
Using a 7% discount rate	$120 +B ($11—$360)	$2.2 +B ($0.2—$6.6)	$120 +B ($11—$360)

[A] For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. A detailed listing of unquantified health and welfare effects is provided in Table 6-2. Estimates here are subject to uncertainties discussed further in the body of the document. Estimates rounded to two significant figures. Value of total co-benefits includes CO_2-related benefits discounted at 3%.
[B] Includes Texas and those states to the north and east.

The co-benefits analysis in this chapter relies on an array of data inputs—including air quality modeling, health impact functions and valuation functions among others—which are themselves subject to uncertainty and may also in turn contribute to the overall uncertainty in this analysis. As a means of characterizing this uncertainty we employ two primary techniques. First, we use Monte Carlo methods for characterizing random sampling error associated with the concentration response functions from epidemiological studies and economic valuation functions. Second, because this characterization of random statistical error may omit important sources of uncertainty we also employ the results of an expert elicitation on the relationship between premature mortality and ambient $PM_{2.5}$ concentration (Roman et al., 2008); this provides additional insight into the likelihood of different outcomes and about the state of

knowledge regarding the benefits estimates. Both approaches have different strengths and weaknesses, which are fully described in Chapter 5 of the PM NAAQS RIA (U.S. EPA, 2006).

Given that reductions in premature mortality dominate the size of the overall monetized co-benefits, more focus on uncertainty in mortality-related benefits gives us greater confidence in our uncertainty characterization surrounding total PM2.5-related co-benefits. Certain EPA RIA's including the 2008 Ozone NAAQS RIA (U.S. EPA, 2008a) contained a suite of sensitivity analyses, only some of which we include here due in part to time constraints. In particular, these analyses characterized the sensitivity of the monetized benefits to the specification of alternate cessation lags and income growth adjustment factors. The estimated co-benefits increased or decreased in proportion to the specification of alternate income growth adjustments and cessation lags, making it possible for readers to infer the sensitivity of the results in this RIA to these parameters by referring to the PM NAAQS RIA (2006d) and Ozone NAAQS RIA (2008a).

For example, the use of an alternate lag structure would change the $PM_{2.5}$-related mortality benefits discounted at 3% discounted by between 10.4% and –27%; when discounted at 7%, these benefits change by between 31% and -49%. When applying higher and lower income growth adjustments, the monetary value of $PM_{2.5}$ -related premature changes between 30% and -10%; the value of chronic endpoints change between 5% and -2% and the value of acute endpoints change between 6% and -7%.

Consistent with the proposed Transport Rule (U.S. EPA, 2010), we bin the estimated number of avoided $PM_{2.5}$-related premature mortalities resulting from the implementation of the Toxics Rule according to the projected 2016 baseline $PM_{2.5}$ air quality levels (Figures 6-19 to 6-21). This presentation is consistent with our approach to applying $PM_{2.5}$ mortality risk coefficients that have not been adjusted to incorporate an assumed threshold. The avoided PM-related impacts we estimate in this analysis occur predominantly among populations exposed at or above the lowest measured air quality level (LML) of each epidemiological study, increasing our confidence in the PM mortality analysis. Approximately 30% of the avoided impacts occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study); about 85% occur at or above an annual mean $PM_{2.5}$ level of 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study). 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. However, the analysis below confirms that the great majority of the impacts occur at or above each study's LML.

Table 6-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed Toxics Rule

Pollutant/ Effect	*Quantified and monetized in base estimate*	*Unquantified*
PM: health[a]	Premature mortality based on cohort study estimates[b] and 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, pre-term birth and other reproductive outcomes 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[D]	Household soiling 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]
Ozone: health		Premature mortality based on short-term study estimates Hospital admissions: respiratory Emergency room visits for asthma Minor restricted activity days School loss days Chronic respiratory damage Premature aging of the lungs Non-asthma respiratory emergency room visits UVb exposure (+/-)[c]
Ozone: welfare		Decreased outdoor worker productivity Yields for: --Commercial forests --Fruits and vegetables, and --Other commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-)[c] Climate impacts
NO_2: health		Respiratory hospital admissions Respiratory emergency department visits Asthma exacerbation Acute respiratory symptoms Premature mortality Pulmonary function

Pollutant/ Effect	*Quantified and monetized in base estimate*	*Unquantified*
NO_X: welfare		Commercial fishing and forestry from acidic deposition effects Commercial fishing, agriculture and forestry from nutrient deposition effects Recreation in terrestrial and estuarine ecosystems from nutrient deposition effects Other ecosystem services and existence values for currently healthy ecosystems Coastal eutrophication from nitrogen deposition effects
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

[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.

[D] Visibility-related benefits quantified in air quality modeled scenario, but not the revised scenario. The total benefits reported in Table 6-1 do not reflect visibility benefits.

6.2 Benefits Analysis Methods

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 (specific effects that can be associated with changes in air quality) 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. In some

cases, the changes in environmental quality can be directly valued, as is the case for changes in visibility. In other cases, such as for changes in ozone and PM, a health and welfare impact analysis must first be conducted to convert air quality changes into effects that can be assigned dollar values.

For the purposes of this RIA, the health impacts analysis (HIA) is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to ozone and PM. There may be other, indirect health impacts associated with implementing emissions controls, such as occupational health impacts for coal miners.

The welfare impacts analysis is limited to changes in the environment that have a direct impact on human welfare. For this analysis, we are limited by the available data to examine impacts of changes in visibility in Class 1 areas. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, for example, changes in deposition of nitrogen and 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. (2000) 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.

6.2.1 Health Impact Assessment

The Health Impact Assessment (HIA) quantifies the changes in the incidence of adverse health impacts resulting from changes in human exposure to $PM_{2.5}$ and ozone air quality. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants (Levy et al. 2009). PC-based tools such as the environmental Benefits Mapping and Analysis Program (BenMAP) can systematize health impact analyses by applying a database of key input parameters, including health impact functions and population projections. 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). EPA and others

have relied upon this method to predict future changes in health impacts expected to result from the implementation of regulations affecting air quality (U.S. EPA, 2008a).

The HIA approach used in this analysis involves three basic steps: (1) utilizing CAMx-generated projections of $PM_{2.5}$ and ozone 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.

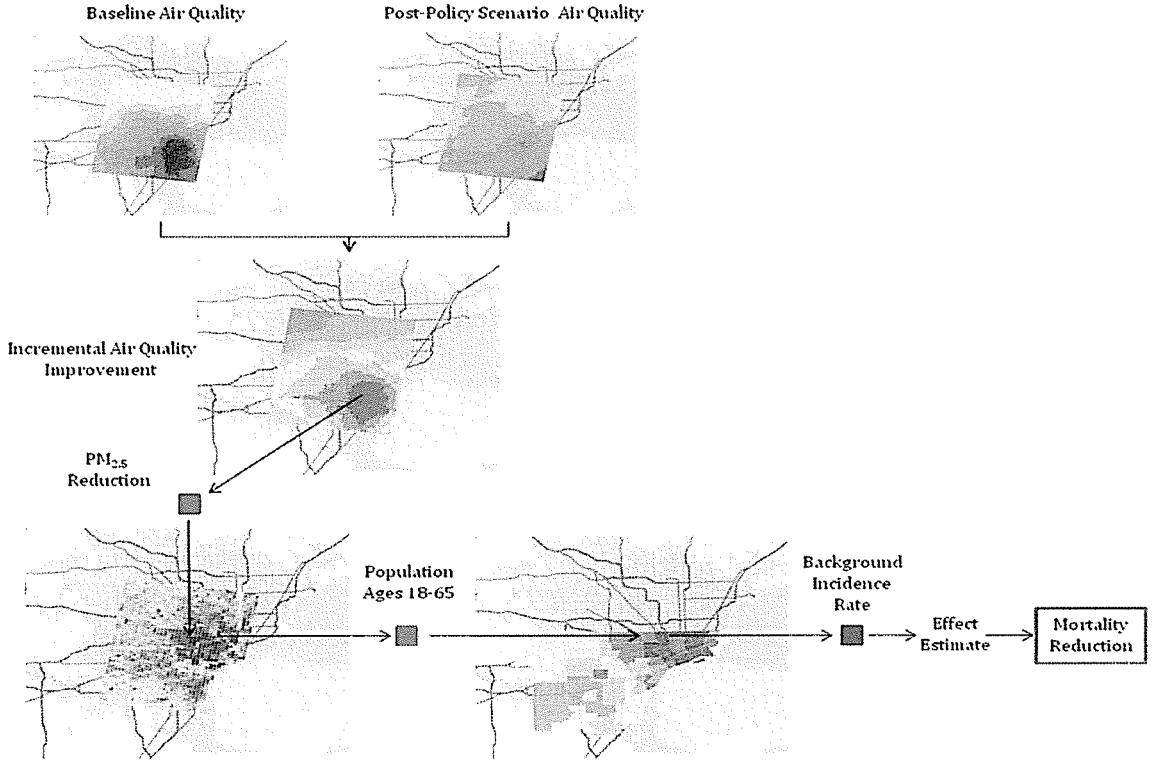
A typical health impact function might look as follows:

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

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. Tools such as BenMAP can systematize the HIA calculation process, allowing users to draw upon a library of existing air quality monitoring data, population data and health impact functions.

Figure 6-1 provides a simplified overview of this approach.

Figure 6-1. Illustration of BenMAP Approach



6.2.2 Economic Valuation of Health Impacts

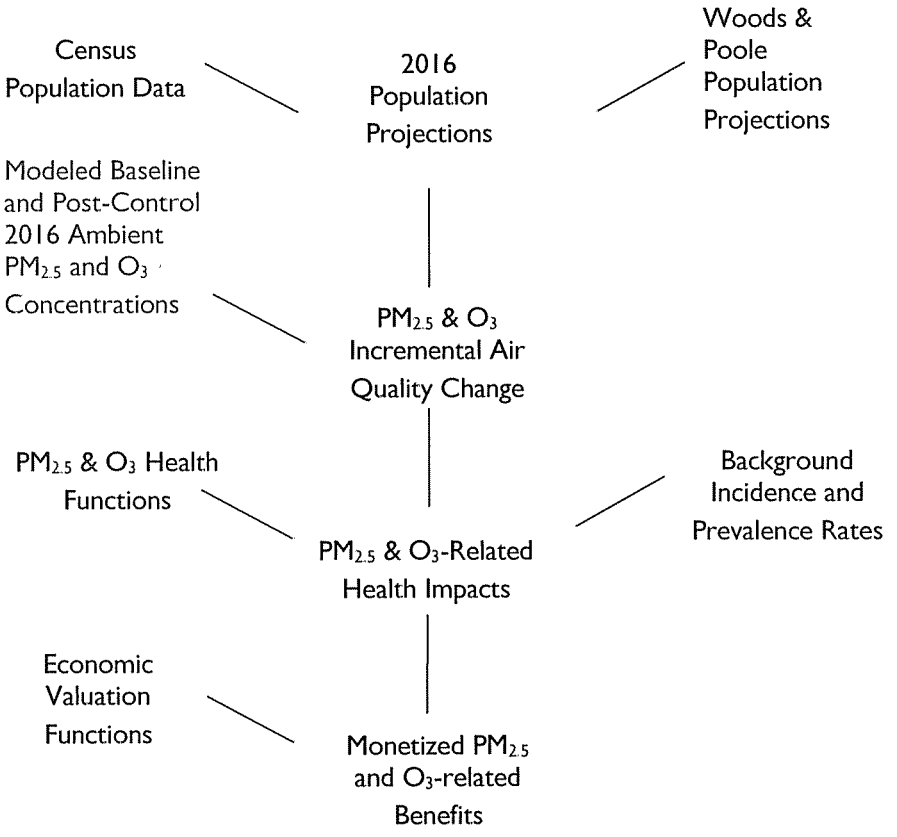
After quantifying the change in adverse health impacts, the final step is to estimate the economic value of these avoided impacts. The appropriate economic value for a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a small amount for a large population. The appropriate economic measure is therefore *ex ante* Willingness to Pay (WTP) for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by

epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These cost of illness (COI) estimates generally (although not in every case) understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect.

We use the BenMAP model (Abt Associates, 2008) to estimate the health impacts and monetized health benefits for the proposed remedy. Figure 6-2 below shows the data inputs and outputs for the BenMAP model.

Figure 6-2. Data Inputs and Outputs for the BenMAP Model



6.2.3 Adjusting the Results of the $PM_{2.5}$ Benefits Analysis to Account for the Emission Reductions in the Proposed Rule

As described in chapter 3 of this RIA, EPA modified the proposed rule requirements after the completion of the air quality modeling for this rule. These changes to the rule affected both the overall level and distribution of $PM_{2.5}$ precursor emissions across the U.S., which in turn affect the level $PM_{2.5}$ co-benefits. Time constraints prevented the Agency from modeling the air quality changes resulting from this updated emissions scenario. In the absence of updated air quality modeling, we adjusted our benefits estimates to reflect these emission changes by applying benefit per-ton estimates.

Benefit per-ton (BPT) estimates quantify the health impacts and monetized human health benefits of an incremental change in air pollution precursor emissions. In circumstances where we are unable to perform air quality modeling because of resource or time constraints, this

approach can provide a reasonable estimate of the benefits of emission reduction scenarios. EPA has used the benefit per-ton technique in previous RIAs, including the recent Ozone NAAQS RIA (U.S. EPA, 2008), the NO_2 NAAQS RIA (U.S. EPA, 2010b) and the proposed Transport rule (U.S. EPA, 2010c).

For this co-benefits analysis we created per-ton estimates of $PM_{2.5}$-related incidence- and monetized benefits based on the benefits of the air quality modeled scenario. Our approach here is methodologically consistent with the technique reported in Fann, Fulcher & Hubbell (2009), but adjusted for this analysis to better match the spatial distribution of air quality changes expected under the Toxics Rule. To derive the BPT estimates for this analysis, we:

1. *Quantified the human and monetized health benefits of changes in each PM species.* We first estimated the health impacts and monetized benefits of reductions in directly emitted $PM_{2.5}$, particulate sulfate and particulate nitrate.[1] We found that, reductions in NOx and SOx led to significant decreases in particulate sulfate and small increases in particulate nitrate, indicating that nitrate replacement limited the nitrate reductions from NOx decreases. Reductions in directly emitted $PM_{2.5}$ were fairly modest, providing a very small change in $PM_{2.5}$. We elected not to generate a NOx benefit per ton for three reasons: (a) reductions in NO_x emissions for this rule were relatively small; (b) previous EPA modeling indicates that $PM_{2.5}$ formation is less sensitive to NO_x emission reductions on a per-$\mu g/m^3$ basis (Fann, Fulcher and Hubbell, 2009); and (c) particulate nitrate formation is governed by complex non-linear chemistry that is difficult to characterize using benefit per-ton estimates.[2]
2. *Divided the health impacts and monetized benefits by the emission reduction.* For the reasons described above, we quantified a SO2 benefit per ton estimate alone. By dividing the particulate sulfate-related benefits in the eastern and western U.S. by the total SO_2-related emission reductions in these two areas, we generated an array of eastern and western benefit per ton estimates.

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[1] Consistent with advice from the Health Effects Subcommittee of the Science Advisory Board, we assume that each PM species is equally toxic. We quantify the change in incidence for each PM specie by applying risk coefficients based on undifferentiated $PM_{2.5}$ mass.

[2] The Toxics Rule reduces both SO_2 and NOx emissions. In general SO_2 is a precursor to particulate sulfate and NOx is a precursor to particulate nitrate. However, there are also several interactions between the $PM_{2.5}$ precursors which cannot be easily quantified. For example, under conditions in which SO_2 levels are reduced by a substantial margin, "nitrate replacement" may occur. This occurs when particulate ammonium sulfate concentrations are reduced, thereby freeing up excess gaseous ammonia. The excess ammonia is then available to react with gaseous nitric acid to form particulate nitrate when meteorological conditions are conducive (cold temperatures and high humidity). The impact of nitrate replacement is also affected by concurrent NOx reductions. NOx reductions can lead to decreases in nitrate, which competes with the process of nitrate replacement. NOx reductions can also lead to reductions in photochemical by-products which can reduce both particulate sulfate and secondary organic carbon PM concentrations.

The resulting BPT estimates were then multiplied by the projected SO_2 emission reductions for the proposed Toxics rule scenario to produce an estimate of the PM—related health impacts and monetized co-benefits. There is no analogous approach for estimating a BPT for visibility, and so the benefits of the alternative remedies omit this important monetized benefit, however, in the model scenario (Appendix C), visibility benefits added $2.2B to the monetized benefits.

An implicit assumption in our approach is that the size and distribution of SO_2 emissions, and the relative levels of NOx and SO_2 emissions, are fairly similar in the modeled and revised policy cases. While the modeled and revised policy case achieve roughly similar levels of SO_2 reductions (2.35 million tons versus 2.06 million tons, respectively), the modeled case concentrates SO_2 reductions primarily among a few Midwestern and southeastern states, while the revised case distributes SO_2 reductions more evenly across both the Midwest, southeast and west. Likewise, the modeled case generates the largest NOx reductions in the Midwest, while the revised case shifts these reductions to western states including Montana, Colorado and Utah. The shifting distribution of NO_x and SO_2 reductions between the modeled and revised cases are likely to affect the overall size of the benefits, a factor that we incompletely account for in our benefit per-ton estimates.

We did not develop an ozone benefit per ton estimate for two reasons. First, the overall level of ozone-related benefits in the modeled case is relatively small compared to those associated with $PM_{2.5}$ reductions (see appendix C), due in part to the fairly modest NO_x emission reductions. Second, the complex non-linear chemistry of ozone formation introduces uncertainty to the development and application of a benefit per ton estimate. Taken together, these factors argued against developing an ozone benefit per ton estimate for this RIA, especially given the shift in the geographic pattern of NOx reductions.

6.3 Uncertainty Characterization

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty and this analysis is no exception. As outlined both in this and preceding chapters, many inputs were used to derive the estimate of benefits for the proposed remedy, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the benefits analysis, may have a disproportionately large

impact on estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to large impacts on total benefits.

The National Research Council (NRC) (2002, 2008) highlighted the need for EPA to conduct rigorous quantitative analysis of uncertainty in its benefits estimates and to present these estimates to decision makers in ways that foster an appropriate appreciation of their inherent uncertainty. In general, the NRC concluded that EPA's general methodology for calculating the benefits of reducing air pollution is reasonable and informative in spite of inherent uncertainties. Since the publication of these reports, EPA's Office of Air and Radiation (OAR) continues to make progress toward the goal of characterizing the aggregate impact of uncertainty in key modeling elements on both health incidence and benefits estimates in two key ways: Monte Carlo analysis and expert-derived concentration-response functions. In this analysis, we use both of these two methods to assess uncertainty quantitatively, as well as provide a qualitative assessment for those aspects that we are unable to address quantitatively.

First, we used Monte Carlo methods for characterizing random sampling error associated with the concentration response functions from epidemiological studies and random effects modeling to characterize both sampling error and variability across the 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 premature mortality. 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.

Second, because characterization of random statistical error omits important sources of uncertainty (e.g., in the functional form of the model—e.g., whether or not a threshold may exist), we also incorporate the results of an expert elicitation on the relationship between premature mortality and ambient $PM_{2.5}$ concentration (Roman et al., 2008). Use of the expert elicitation and incorporation of the standard errors approaches provide insights into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates. However, there are significant unquantified uncertainties present in upstream inputs including emission and air quality. Both approaches have different strengths and weaknesses, which are fully described in Chapter 5 of the PM NAAQS RIA (U.S. EPA, 2006).

In benefit analyses of air pollution regulations conducted to date, the estimated impact of reductions in premature mortality has accounted for 85% to 95% of total monetized benefits. Therefore, it is particularly important to attempt to characterize the uncertainties associated with reductions in premature mortality. The health impact functions used to estimate avoided premature deaths associated with reductions in ozone have associated standard errors that represent the statistical errors around the effect estimates in the underlying epidemiological studies. In our results, we report credible intervals based on these standard errors, reflecting the uncertainty in the estimated change in incidence of avoided premature deaths. We also provide multiple estimates, to reflect model uncertainty between alternative study designs.

For premature mortality associated with exposure to PM, we follow the same approach used in the RIA for 2006 PM NAAQS (U.S. EPA, 2006), presenting two empirical estimates of premature deaths avoided, and a set of twelve estimates based on results of the expert elicitation study. Even these multiple characterizations, including confidence intervals, omit the contribution to overall uncertainty of uncertainty in air quality changes, baseline incidence rates, populations exposed and transferability of the effect estimate to diverse locations. Furthermore, the approach presented here does not yet include methods for addressing correlation between input parameters and the identification of reasonable upper and lower bounds for input distributions characterizing uncertainty in additional model elements. As a result, the reported confidence intervals and range of estimates give an incomplete picture about the overall uncertainty in the estimates. This information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

Some key sources of uncertainty in each stage of the PM health impact assessment are the following:

- gaps in scientific data and inquiry;
- variability in estimated relationships, such as epidemiological effect estimates, introduced through differences in study design and statistical modeling;
- errors in measurement and projection for variables such as population growth rates;
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions; and
- biases due to omissions or other research limitations.

In Table 6-3 we summarize some of the key uncertainties in the benefits analysis.

Table 6-3. Primary Sources of Uncertainty in the Benefits Analysis

1. *Uncertainties Associated with Impact Functions* - The value of the ozone or PM effect estimate in each impact function. - Application of a single impact function to pollutant changes and populations in all locations. - Similarity of future-year impact functions to current impact functions. - Correct functional form of each impact function. - Extrapolation of effect estimates beyond the range of ozone or PM concentrations observed in the source epidemiological study. - Application of impact functions only to those subpopulations matching the original study population.
2. *Uncertainties Associated with CAMx-Modeled Ozone and PM Concentrations* - Responsiveness of the models to changes in precursor emissions from the control policy. - Projections of future levels of precursor emissions, especially ammonia and crustal materials. - Lack of ozone and $PM_{2.5}$ monitors in all rural areas requires extrapolation of observed ozone data from urban to rural areas.
3. *Uncertainties Associated with PM Mortality Risk* - Limited scientific literature supporting a direct biological mechanism for observed epidemiological evidence. - Direct causal agents within the complex mixture of PM have not been identified. - The extent to which adverse health effects are associated with low-level exposures that occur many times in the year versus peak exposures. - The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. - Reliability of the $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. *Uncertainties Associated with Possible Lagged Effects* - The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels that would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. *Uncertainties Associated with Baseline Incidence Rates* - Some baseline incidence rates are not location specific (e.g., those taken from studies) and therefore may not accurately represent the actual location-specific rates. - Current baseline incidence rates may not approximate well baseline incidence rates in 2014. - Projected population and demographics may not represent well future-year population and demographics.
6. *Uncertainties Associated with Economic Valuation* - Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. - Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates because of differences in income or other factors.
7. *Uncertainties Associated with Aggregation of Monetized Benefits* - Health and welfare benefits estimates are limited to the available impact functions. Thus, unquantified or unmonetized benefits are not included.

6.4 Benefits Analysis Data Inputs

In Figure 6-2, we summarized the key data inputs to the health impact and economic valuation estimate. Below we summarize the data sources for each of these inputs, including demographic projections, effect coefficients, incidence rates and economic valuation. Our approach here is generally consistent with the proposed Transport Rule (U.S. EPA, 2010c).

6.4.1 Demographic Data

Quantified and monetized human health impacts depend on the demographic characteristics of the population, including age, location, and income. We use projections based on economic forecasting models developed by Woods and Poole, Inc (Woods and Poole, 2008). The Woods and Poole (WP) database contains county-level projections of population by age, sex, and race out to 2030. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates (Hollman et al., 2000). According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process:

1. First, national-level variables such as income, employment, and populations are forecasted.
2. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an "export-base" approach, which relies on linking industrial-sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector.
3. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort-component method based on fertility and mortality in each area.
4. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2016 based on historical rates of mortality, fertility, and migration.

6.4.2 Effect Coefficients

The first step in selecting effect coefficients is to identify the health endpoints to be quantified. We base our selection of health endpoints on consistency with EPA's Integrated

Science Assessments (which replace the Criteria Document), with input and advice from the EPA Science Advisory Board - Health Effects Subcommittee (SAB-HES), a scientific review panel specifically established to provide advice on the use of the scientific literature in developing benefits analyses for air pollution regulations (http://www.epa.gov/sab/). In general, we follow a weight of evidence approach, based on the biological plausibility of effects, availability of concentration-response functions from well conducted peer-reviewed epidemiological studies, cohesiveness of results across studies, and a focus on endpoints reflecting public health impacts (like hospital admissions) rather than physiological responses (such as changes in clinical measures like Forced Expiratory Volume (FEV1)).

There are several types of data that can support the determination of types and magnitude of health effects associated with air pollution exposures. These sources of data include toxicological studies (including animal and cellular studies), human clinical trials, and observational epidemiology studies. All of these data sources provide important contributions to the weight of evidence surrounding a particular health impact. However, only epidemiology studies provide direct concentration-response relationships which can be used to evaluate population-level impacts of reductions in ambient pollution levels in a health impact assessment.

For the data-derived estimates, we relied on the published scientific literature to ascertain the relationship between PM and adverse human health effects. We evaluated epidemiological studies using the selection criteria summarized in Table 6-4. These criteria include consideration of whether the study was peer-reviewed, the match between the pollutant studied and the pollutant of interest, the study design and location, and characteristics of the study population, among other considerations. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. In general, the use of results from more than a single study can provide a more robust estimate of the relationship between a pollutant and a given health effect. However, there are often differences between studies examining the same endpoint, making it difficult to pool the results in a consistent manner. For example, studies may examine different pollutants or different age groups. For this reason, we consider very carefully the set of studies available examining each endpoint and select a consistent subset that provides a good balance of population coverage and match with the pollutant of interest. In many cases, either because of a lack of multiple studies, consistency problems, or clear superiority in the quality or comprehensiveness of one study over others, a single published study is selected as the basis of the effect estimate.

Table 6-4. Criteria Used when Selecting C-R Functions

Consideration	*Comments*
Peer-Reviewed Research	Peer-reviewed research is preferred to research that has not undergone the peer-review process.
Study Type	Among studies that consider chronic exposure (e.g., over a year or longer), prospective cohort studies are preferred over ecological studies because they control for important individual-level confounding variables that cannot be controlled for in ecological studies.
Study Period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and lifestyle over time. However, when there are only a few studies available, studies from all years will be included.
Population Attributes	The most technically appropriate measures of benefits would be based on impact functions that cover the entire sensitive population but allow for heterogeneity across age or other relevant demographic factors. In the absence of effect estimates specific to age, sex, preexisting condition status, or other relevant factors, it may be appropriate to select effect estimates that cover the broadest population to match with the desired outcome of the analysis, which is total national-level health impacts. When available, multi-city studies are preferred to single city studies because they provide a more generalizable representation of the C-R function.
Study Size	Studies examining a relatively large sample are preferred because they generally have more power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population or through repeated observations on a smaller population (e.g., through a symptom diary recorded for a panel of asthmatic children).
Study Location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior, and lifestyle.
Pollutants Included in Model	When modeling the effects of ozone and PM (or other pollutant combinations) jointly, it is important to use properly specified impact functions that include both pollutants. Using single-pollutant models in cases where both pollutants are expected to affect a health outcome can lead to double-counting when pollutants are correlated.
Measure of PM	For this analysis, impact functions based on $PM_{2.5}$ are preferred to PM_{10} because of the focus on reducing emissions of $PM_{2.5}$ precursors, and because air quality modeling was conducted for this size fraction of PM. Where $PM_{2.5}$ functions are not available, PM_{10} functions are used as surrogates, recognizing that there will be potential downward (upward) biases if the fine fraction of PM_{10} is more (less) toxic than the coarse fraction.
Economically Valuable Health Effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping Endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double-counting of benefits.

When several effect estimates for a pollutant and a given health endpoint have been selected, they are quantitatively combined or pooled to derive a more robust estimate of the relationship. The BenMAP Technical Appendices provides details of the procedures used to combine multiple impact functions (Abt Associates, 2008). In general, we used fixed or random effects models to pool estimates from different studies of the same endpoint. Fixed effects pooling simply weights each study's estimate by the inverse variance, giving more weight to studies with greater statistical power (lower variance). Random effects pooling accounts for both within-study variance and between-study variability, due, for example, to differences in population susceptibility. We used the fixed effects model as our null hypothesis and then determined whether the data suggest that we should reject this null hypothesis, in which case we would use the random effects model. Pooled impact functions are used to estimate hospital admissions and asthma exacerbations. For more details on methods used to pool incidence estimates, see the BenMAP Manual Appendices (Abt Associates, 2008), which are available with the BenMAP software at http://www.epa.gov/benmap.html.

Effect estimates selected for a given health endpoint were applied consistently across all locations nationwide. This applies to both impact functions defined by a single effect estimate and those defined by a pooling of multiple effect estimates. Although the effect estimate may, in fact, vary from one location to another (e.g., because of differences in population susceptibilities or differences in the composition of PM), location-specific effect estimates are generally not available.

The specific studies from which effect estimates for the primary analysis are drawn are included in Table 6-5. We highlight in blue those studies that have been added since the 2005 CAIR benefits analysis and incorporated into the central benefits estimate. In all cases where effect estimates are drawn directly from epidemiological studies, standard errors are used as a partial representation of the uncertainty in the size of the effect estimate. Below we provide the basis for selecting these studies.

Table 6-5. Health Endpoints and Epidemiological Studies Used to Quantify Health Impacts[a]

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Premature mortality—cohort study, all-cause	$PM_{2.5}$ (annual avg)	Pope et al. (2002) Laden et al. (2006)	>29 years >25 years
Premature mortality, total exposures	$PM_{2.5}$ (annual avg)	Expert Elicitation (Roman et al., 2008)	>24 years
Premature mortality—all-cause	$PM_{2.5}$ (annual avg)	Woodruff et al. (2006)	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	$PM_{2.5}$ (annual avg)	Abbey et al. (1995)	>26 years
Nonfatal heart attacks	$PM_{2.5}$ (24-hour avg)	Peters et al. (2001)	Adults (>18 years)
Hospital Admissions			
Respiratory	$PM_{2.5}$ (24-hour avg)	*Pooled estimate:* Moolgavkar (2003)—ICD 490–496 (COPD) Ito (2003)—ICD 490–496 (COPD)	>64 years
	$PM_{2.5}$ (24-hour avg)	Moolgavkar (2000)—ICD 490–496 (COPD)	20–64 years
	$PM_{2.5}$ (24-hour avg)	Ito (2003)—ICD 480–486 (pneumonia)	>64 years
	$PM_{2.5}$ (24-hour avg)	Sheppard (2003)—ICD 493 (asthma)	<65 years
Cardiovascular	$PM_{2.5}$ (24-hour avg)	*Pooled estimate:* Moolgavkar (2003)—ICD 390–429 (all cardiovascular) Ito (2003)—ICD 410–414, 427–428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	$PM_{2.5}$ (24-hour avg)	Moolgavkar (2000)—ICD 390–429 (all cardiovascular)	20–64 years
Asthma-related ER visits	$PM_{2.5}$ (24-hour avg)	Norris et al. (1999)	0–18 years

Endpoint	Pollutant	Study	Study Population
Other Health Endpoints			
Acute bronchitis	$PM_{2.5}$ (annual avg)	Dockery et al. (1996)	8–12 years
Upper respiratory symptoms	PM_{10} (24-hour avg)	Pope et al. (1991)	Asthmatics, 9–11 years
Lower respiratory symptoms	$PM_{2.5}$ (24-hour avg)	Schwartz and Neas (2000)	7–14 years
Asthma exacerbations	$PM_{2.5}$ (24-hour avg)	Pooled estimate: Ostro et al. (2001) (cough, wheeze and shortness of breath) Vedal et al. (1998) (cough)	6–18 years[b]
Work loss days	$PM_{2.5}$ (24-hour avg)	Ostro (1987)	18–65 years
Minor Restricted Activity Days (MRADs)	$PM_{2.5}$ (24-hour avg)	Ostro and Rothschild (1989)	18–65 years

[a] Studies or air quality metrics highlighted in blue represent updates incorporated since the 2005 CAIR RIA

[b] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the Science Advisory Board Health Effects Subcommittee (SAB-HES), we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. Science Advisory Board. 2004. Advisory 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. EPA-SAB-COUNCIL-ADV-04-004. See also National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

[c] Gilliland et al. (2001) studied children aged 9 and 10. Chen et al. (2000) studied children 6 to 11. Based on recent advice from the National Research Council and the EPA SAB-HES, we have calculated reductions in school absences for all school-aged children based on the biological similarity between children aged 5 to 17.

6.4.2.1 $PM_{2.5}$ Premature Mortality Effect Coefficients

Both long- and short-term exposures to ambient levels of $PM_{2.5}$ air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis.

Although a number of uncertainties remain to be addressed by continued research (NRC, 2002), a substantial body of published scientific literature documents the correlation between elevated $PM_{2.5}$ concentrations and increased mortality rates (U.S. EPA, 2009d). Time-series methods have been used to relate short-term (often day-to-day) changes in $PM_{2.5}$ concentrations and changes in daily mortality rates up to several days after a period of elevated $PM_{2.5}$ concentrations. Cohort methods have been used to examine the potential relationship between

community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between $PM_{2.5}$ and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they account for the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). This section discusses some of the issues surrounding the estimation of $PM_{2.5}$-related premature mortality. To demonstrate the sensitivity of the benefits estimates to the specific sources of information regarding the impact of $PM_{2.5}$ exposures on the risk of premature death, we are providing estimates in our results tables based on studies derived from the epidemiological literature and from the EPA sponsored expert elicitation. The epidemiological studies from which these estimates are drawn are described below. The expert elicitation project and the derivation of effect estimates from the expert elicitation results are described in the 2006 $PM_{2.5}$ NAAQS RIA and Roman et al. (2008). In the interest of brevity we do not repeat those details here. However, Figure 6-13 summarizes the estimated $PM_{2.5}$-related premature mortalities avoided using risk estimates drawn from the expert elicitation.

Over a dozen epidemiological studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977). Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). However, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert et al., 1989). These early "ecological cross-sectional" studies (Lave and Seskin, 1977; Ozkaynak and Thurston, 1987) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet.

Over the last 17 years, several studies using "prospective cohort" designs have been published that appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual level information with respect to health status and residence. The most extensive analyses have been based on data from two prospective cohort groups, often referred to as the Harvard "Six-Cities Study" (Dockery et al., 1993; Laden et al., 2006) and the "American Cancer Society or ACS study" (Pope et al., 1995; Pope et al., 2002; Pope et al., 2004, Krewski et al. 2009); these studies have found consistent relationships between fine particle indicators and

premature mortality across multiple locations in the United States. A third major data set comes from the California-based 7th Day Adventist Study (e.g., Abbey et al., 1999), which reported associations between long-term PM exposure and mortality in men. Results from this cohort, however, have been inconsistent, and the air quality results are not geographically representative of most of the United States, and the lifestyle of the population is not reflective of much of the U.S. population. Analysis is also available for a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000; Lipfert et al., 2003, 2006). The characteristics of this group differ from the cohorts in the Six-Cities, ACS, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment.

Given their consistent results and broad geographic coverage, and importance in informing the NAAQS development process, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that the initial published studies (Pope et al., 1995 and Dockery et al., 1993) were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by the Health Effect Institute (HEI) (Krewski et al., 2000). The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded the conclusions of the original investigators. While the HEI reexamination lends credibility to the original studies, it also highlights sensitivities concerning the relative impact of various pollutants, such as SO_2, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling. Further confirmation and extension of the findings of the 1993 Six City Study and the 1995 ACS study were recently completed using more recent air quality and a longer follow-up period for the ACS cohort was published over the past several years (Pope et al., 2002, 2004; Laden et al., 2006, Krewski et al. 2009). The follow up to the Harvard Six City Study both confirmed the effect size from the first analysis and provided additional confirmation that reductions in $PM_{2.5}$ are likely to result in reductions in the risk of premature death. This additional evidence stems from the observed reductions in $PM_{2.5}$ in each city during the extended follow-up period. Laden et al. (2006) found that mortality rates consistently went down at a rate proportionate to the observed reductions in $PM_{2.5}$.

A number of additional analyses have been conducted on the ACS cohort data (Jarrett et al., 2009; Pope et al., 2009). These studies have continued to find a strong significant relationship between $PM_{2.5}$ and mortality outcomes and life expectancy. Specifically, much of the recent research has suggested a stronger relationship between cardiovascular mortality and lung cancer mortality with $PM_{2.5}$, and a less significant relationship between respiratory-related mortality and $PM_{2.5}$. The extended analyses of the ACS cohort data (Krewski et al. 2009) provides additional refinements to the analysis of PM-related mortality by (a) extend the follow-up period by 2 years to the year 2000, for a total of 18 years; (b) incorporate ecological., or neighborhood-level co-variates so as to better estimate personal exposure; (c) perform an extensive spatial analysis using land use regression modeling. These additional refinements may make this analysis well-suited for the assessment of PM-related mortality for EPA benefits analyses.

In developing and improving the methods for estimating and valuing the potential reductions in mortality risk over the years, EPA consulted with the SAB-HES. That panel recommended using long-term prospective cohort studies in estimating mortality risk reduction (U.S. EPA-SAB, 1999b). This recommendation has been confirmed by a report from the National Research Council, which stated that "it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure" (NRC, 2002, p. 108). More specifically, the SAB recommended emphasis on the ACS study because it includes a much larger sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six Cities Study) than other studies of its kind. Because of the refinements in the extended follow-up analysis, the SAB-HES recommended using the Pope et al. (2002) study as the basis for the primary mortality estimate for adults and suggests that alternate estimates of mortality generated using other cohort and time-series studies could be included as part of the sensitivity analysis (U.S. EPA-SAB, 2004b). The PM NAAQS Risk and Exposure Assessment (U.S. EPA, 2010) utilized risk coefficients drawn from the Krewski et al. (2009) study. In a December of 2009 consultation with the SAB-HES, the Agency proposed utilizing the Krewski et al. (2009) extended analysis of the ACS cohort data. The panel is scheduled to issue an advisory in early 2010.

As noted above, since 2004 SAB review, an extended follow-up of the Harvard Six cities study has been published (Laden et al., 2006) and in recent RIAs (see for example the SO_2 NAAQS, PM NAAQS, CAIR and Nonroad Diesel RIAs), we have included this estimate of mortality impacts based on application of the C-R function derived from this study. We use this specific estimate to represent the Six Cities study because it both reflects among the most up-to-

date science and was cited by many of the experts in their elicitation responses. It is clear from the expert elicitation that the results published in Laden et al. (2006) are potentially influential, and in fact the expert elicitation results encompass within their range the estimates from both the Pope et al. (2002) and Laden et al. (2006) studies (see Figure 6-13 below). 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.

6.4.2.2 Chronic Bronchitis

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because the Toxics Rule is expected to reduce $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

Nonfatal Myocardial Infarctions (Heart Attacks)

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including those for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for nonfatal heart attacks. The estimate used in the Toxics Rule analysis is based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997; Tsuji et al., 1996). As such,

significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions

Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room are treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital.

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM with other types of hospital admissions. The only type of emergency room visits that have been consistently linked to ozone and PM in the United States are asthma-related visits.

To estimate avoided incidences of cardiovascular hospital admissions associated with $PM_{2.5}$, we used studies by Moolgavkar (2003) and Ito (2003). Additional published studies show a statistically significant relationship between PM_{10} and cardiovascular hospital admissions. However, given that the control options we are analyzing are expected to reduce primarily $PM_{2.5}$, we focus on the two studies that examine $PM_{2.5}$. Both of these studies provide an effect estimate for populations over 65, allowing us to pool the impact functions for this age group. Only Moolgavkar (2000) provided a separate effect estimate for populations 20 to 64.[1] Total cardiovascular hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

[1]Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Given the very small (<5 percent) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect this choice to introduce much bias.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, additional published studies show a statistically significant relationship between PM_{10} and respiratory hospital admissions. We used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito (2003) estimated pneumonia and only for the population 65 and older. In addition, Sheppard (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidences of PM-related respiratory-related hospital admissions are the sum of COPD, pneumonia, and asthma admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999). As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of $PM_{2.5}$ on asthma emergency room visits in populations under 65, although there may still be significant impacts in the adult population under 65.

To estimate avoided incidences of respiratory hospital admissions associated with ozone, we used a number of studies examining hospital admissions for a range of respiratory illnesses, including pneumonia and COPD. Two age groups, adults over 65 and children under 2, were examined. For adults over 65, Schwartz (1995) provides effect estimates for two different cities relating ozone and hospital admissions for all respiratory causes (defined as ICD codes 460–519). Impact functions based on these studies were pooled first before being pooled with other studies. Two studies (Moolgavkar et al., 1997; Schwartz, 1994a) examine ozone and pneumonia hospital admissions in Minneapolis. One additional study (Schwartz, 1994b) examines ozone and pneumonia hospital admissions in Detroit. The impact functions for Minneapolis were pooled together first, and the resulting impact function was then pooled with the impact function for Detroit. This avoids assigning too much weight to the information coming from one city. For COPD hospital admissions, two studies are available: Moolgavkar et al. (1997), conducted in Minneapolis, and Schwartz (1994b), conducted in Detroit. These two studies were pooled

together. To estimate total respiratory hospital admissions for adults over 65, COPD admissions were added to pneumonia admissions, and the result was pooled with the Schwartz (1995) estimate of total respiratory admissions. Burnett et al. (2001) is the only study providing an effect estimate for respiratory hospital admissions in children under 2.

We used two studies as the source of the concentration-response functions we used to estimate the effects of ozone exposure on asthma-related emergency room (ER) visits: Peel et al. (2005) and Wilson et al. (2005). We estimated the change in ER visits using the effect estimate(s) from each study and then pooled the results using the random effects pooling technique (see Abt, 2005). The Peel et al. study (2005) estimated asthma-related ER visits for all ages in Atlanta, using air quality data from 1993 to 2000. Using Poisson generalized estimating equations, the authors found a marginal association between the maximum daily 8-hour average ozone level and ER visits for asthma over a 3-day moving average (lags of 0, 1, and 2 days) in a single pollutant model. Wilson et al. (2005) examined the relationship between ER visits for respiratory illnesses and asthma and air pollution for all people residing in Portland, Maine from 1998–2000 and Manchester, New Hampshire from 1996–2000. For all models used in the analysis, the authors restricted the ozone data incorporated into the model to the months ozone levels are usually measured, the spring-summer months (April through September). Using the generalized additive model, Wilson et al. (2005) found a significant association between the maximum daily 8-hour average ozone level and ER visits for asthma in Portland, but found no significant association for Manchester. Similar to the approach used to generate effect estimates for hospital admissions, we used random effects pooling to combine the results across the individual study estimates for ER visits for asthma. The Peel et al. (2005) and Wilson et al. (2005) Manchester estimates were not significant at the 95 percent level, and thus, the confidence interval for the pooled incidence estimate based on these studies includes negative values. This is an artifact of the statistical power of the studies, and the negative values in the tails of the estimated effect distributions do not represent improvements in health as ozone concentrations are increased. Instead, these should be viewed as a measure of uncertainty due to limitations in the statistical power of the study. We included both hospital admissions and ER visits as separate endpoints associated with ozone exposure because our estimates of hospital admission costs do not include the costs of ER visits and most asthma ER visits do not result in a hospital admission.

Acute Health Events and School/Work Loss Days

In addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of

ozone and PM. The sources for the effect estimates used to quantify these effects are described below.

Around 4 percent of U.S. children between the ages of 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002c). Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,[1] with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of 5 and 17 were estimated using an effect estimate developed from Dockery et al. (1996).

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 were estimated using an effect estimate from Schwartz and Neas (2000).

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Days of work lost due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987). Children may also be absent from school because of respiratory or other diseases caused by exposure to air pollution. Most studies examining school absence rates have found little or no association with $PM_{2.5}$, but several studies have found a significant association between ozone levels and school absence rates. We used two recent studies, Gilliland et al. (2001) and Chen et al. (2000), to estimate changes in absences (school loss days) due to changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined absence on a given day. We converted the Gilliland estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimated an average duration of school absence of 1.6 days by dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence rate from Gilliland et al. (2001).

[1]See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

This provides estimates from Chen et al. (2000) and Gilliland et al. (2001), which can be pooled to provide an overall estimate.

MRAD result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work most of the day will instead spend the day at a desk doing paper and phone work because of difficulty breathing or chest pain. The effect of $PM_{2.5}$ and ozone on MRAD was estimated using an effect estimate derived from Ostro and Rothschild (1989).

For the Toxics Rule, we have followed the SAB-HES recommendations regarding asthma exacerbations in developing the primary estimate. To prevent double-counting, we focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation.[1] Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998) that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between

[1] Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double counting of benefits. Concerns over double counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double counting, especially if they evaluate similar endpoints. The SAB-HES, in commenting on the analytical blueprint for 812, acknowledged these challenges in evaluating asthmatic symptoms and appropriately adding them into the primary analysis (SAB-HES, 2004). However, despite these challenges, the SAB-HES recommends the addition of asthma-related symptoms (i.e., asthma exacerbations) to the primary analysis, provided that the studies use the panel study approach and that they have comparable design and baseline frequencies in both asthma prevalence and exacerbation rates. Note also, that the SAB-HES, while supporting the incorporation of asthma exacerbation estimates, does not believe that the association between ambient air pollution, including ozone and PM, and the new onset of asthma is sufficiently strong to support inclusion of this asthma-related endpoint in the primary estimate.

$PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM_{10} and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

6.4.3 Baseline Incidence Estimates

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the *relative risk* of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 10 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per million people, that number must be multiplied by the millions of people in the total population.

Table 6-6 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and

prevalence data, we used age-specific rates where available. We applied concentration-response functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits. In most cases, we used a single national incidence rate, due to a lack of more spatially disaggregated data. Whenever possible, the national rates used are national averages, because these data are most applicable to a national assessment of benefits. For some studies, however, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Regional incidence rates are available for hospital admissions, and county-level data are available for premature mortality. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth (Abt Associates, 2008); this represents a change from the 2005 CAIR analysis, which used static rates.

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 6-7 lists the prevalence rates used to determine the applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years. EPA is aware that more current estimates of asthma prevalence are available from the American Lung Association. However, we applied these older rates to maintain methodological consistency with the proposed Transport Rule. We anticipate incorporating more recent prevalence rates for the RIA accompanying the rule; other things being equal, the newer rates would result in a larger overall estimate of asthma-related impacts.

Table 6-6. Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population

		Rates	
Endpoint	*Parameter*	*Value*	*Source*[a]
Mortality	Daily or annual mortality rate projected to 2015	Age-, cause-, and county-specific rate	CDC Wonder (1996–1998) U.S. Census bureau
Hospitalizations	Daily hospitalization rate	Age-, region-, and cause-specific rate	1999 NHDS public use data files[b]
Asthma ER Visits	Daily asthma ER visit rate	Age- and region- specific visit rate	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]
Chronic Bronchitis	Annual prevalence rate per person		1999 NHIS (American Lung Association, 2002b, Table 4)
	• Aged 18–44	0.0367	
	• Aged 45–64	0.0505	
	• Aged 65 and older	0.0587	
	Annual incidence rate per person	0.00378	Abbey et al. (1993, Table 3)

Endpoint	*Parameter*	*Rates*	
		Value	*Source*[a]
Nonfatal Myocardial Infarction (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+		1999 NHDS public use data files[b]; adjusted by 0.93 for probability of surviving after 28 days (Rosamond et al., 1999)
	• Northeast	0.0000159	
	• Midwest	0.0000135	
	• South	0.0000111	
	• West	0.0000100	
Asthma Exacerbations	Incidence (and prevalence) among asthmatic African-American children		Ostro et al. (2001)
	• daily wheeze	0.076 (0.173)	
	• daily cough	0.067 (0.145)	
	• daily dyspnea	0.037 (0.074)	
	Prevalence among asthmatic children		Vedal et al. (1998)
	• daily wheeze	0.038	
	• daily cough	0.086	
	• daily dyspnea	0.045	
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002c, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[d]	0.0012	Schwartz et al. (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18–65)		1996 HIS (Adams, Hendershot, and Marano, 1999, Table 41); U.S. Bureau of the Census (2000)
	• Aged 18–24	0.00540	
	• Aged 25–44	0.00678	
	• Aged 45–64	0.00492	
Minor Restricted-Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

[a] The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.

[b] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/.

[c] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/.

[d] Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

Table 6-7. Asthma Prevalence Rates Used for this Analysis

	Asthma Prevalence Rates	
Population Group	*Value*	*Source*
All Ages	0.0386	American Lung Association (2002a, Table 7)—based on 1999 HIS
< 18	0.0527	American Lung Association (2002a, Table 7)—based on 1999 HIS
5–17	0.0567	American Lung Association (2002a, Table 7)—based on 1999 HIS
18–44	0.0371	American Lung Association (2002a, Table 7)—based on 1999 HIS
45–64	0.0333	American Lung Association (2002a, Table 7)—based on 1999 HIS
65+	0.0221	American Lung Association (2002a, Table 7)—based on 1999 HIS
Male, 27+	0.021	2000 HIS public use data files[a]
African American, 5 to 17	0.0726	American Lung Association (2002a, Table 9)—based on 1999 HIS
African American, <18	0.0735	American Lung Association (2002a, Table 9)—based on 1999 HIS

[a] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/.

6.4.4 Economic Valuation Estimates

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects for a large population. Therefore, the appropriate economic measure is WTP for changes in risk of a health effect rather than WTP for a health effect that would occur with certainty (Freeman, 1993). Epidemiological studies generally provide estimates of the relative risks of a particular health effect that is avoided because of a reduction in air pollution. We converted those to units of avoided statistical incidence for ease of presentation. We calculated the value of avoided statistical incidences by dividing individual WTP for a risk reduction by the related observed change in risk.

WTP estimates generally are not available for some health effects, such as hospital admissions. In these cases, we used the cost of treating or mitigating the effect as a primary estimate. These cost-of-illness (COI) estimates generally understate the true value of reducing the risk of a health effect, because they reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering (Harrington and Portney, 1987; Berger, 1987). We provide unit values for health endpoints (along with information on the distribution of the unit value) in Table 6-8. All values are in constant year 2006 dollars, adjusted for growth in real income out to 2016 using projections provided by Standard and Poor's. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real income increases. Many of the valuation studies used in this analysis were conducted in the late 1980s

and early 1990s. Because real income has grown since the studies were conducted, people's willingness to pay for reductions in the risk of premature death and disease likely has grown as well. We did not adjust cost of illness-based values because they are based on current costs. Similarly, we did not adjust the value of school absences, because that value is based on current wage rates. For these two reasons, these cost of illness estimates may underestimate the economic value of avoided health impacts in 2016. The discussion below provides additional details on ozone and $PM_{2.5}$-related related endpoints.

Mortality Valuation

Following the advice of the EEAC of the SAB, EPA currently uses the VSL approach in calculating the primary estimate of mortality benefits, because we believe this calculation provides the most reasonable single estimate of an individual's willingness to trade off money for reductions in mortality risk (U.S. EPA-SAB, 2000). The VSL approach is a summary measure for the value of small changes in mortality risk experienced by a large number of people. For a period of time (2004-2008), the Office of Air and Radiation (OAR) valued mortality risk reductions using a value of 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 $5.5 million (2000$) 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)[1] 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 1991. The mean VSL across these studies is $6.3 million (2000$).[2] 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. The Agency anticipates presenting results from this effort to the SAB-EEAC in Spring 2010 and that draft guidance will be available shortly thereafter.

As indicated in the previous section on quantification of premature mortality benefits, we assumed for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3% discount rate to the value of premature mortality occurring in future years.[3]

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis community. EPA strives to use the best economic science in its analyses. Given the mixed theoretical finding and empirical evidence regarding adjustments to VSL for risk and population characteristics, we use a single VSL for all reductions in mortality risk.

Although there are several differences between the labor market studies EPA uses to derive a VSL estimate and the PM air pollution context addressed here, those differences in the

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[1] In the (draft) update of the Economic Guidelines (U.S. EPA, 2008d), 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.

[2] In this analysis, we adjust the VSL to account for a different currency year (2006$) and to account for income growth to 2014. After applying these adjustments to the $6.3 million value, the VSL is $7.8M.

[3] The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3% discount rate for its base estimate in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7% rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA's *Guidelines for Preparing Economic Analyses* (EPA, 2010).

affected populations and the nature of the risks imply both upward and downward adjustments. Table 6-11 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the $6.3 million value while acknowledging the significant limitations and uncertainties in the available literature.

Table 6-8. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	*Expected Direction of Bias*
Age	Uncertain, perhaps overestimate
Life Expectancy/Health Status	Uncertain, perhaps overestimate
Attitudes Toward Risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate

The SAB-EEAC has reviewed many potential VSL adjustments and the state of the economics literature. The SAB-EEAC advised EPA to "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk" (U.S. EPA, 2000a). In developing our primary estimate of the benefits of premature mortality reductions, we have followed this advice and discounted over the lag period between exposure and premature mortality.

Uncertainties Specific to Premature Mortality Valuation. The economic benefits associated with reductions in the risk of premature mortality are the largest category of monetized benefits of the Toxics Rule. In addition, in prior analyses, EPA has identified valuation of mortality-related benefits as the largest contributor to the range of uncertainty in monetized benefits (U.S. EPA, 1999b).[1] Because of the uncertainty in estimates of the value of reducing premature mortality risk, it is important to adequately characterize and understand the various types of economic approaches available for valuing reductions in mortality risk. Such an

[1] This conclusion was based on an assessment of uncertainty based on statistical error in epidemiological effect estimates and economic valuation estimates. Additional sources of model error such as those examined in the PM mortality expert elicitation may result in different conclusions about the relative contribution of sources of uncertainty.

assessment also requires an understanding of how alternative valuation approaches reflect that some individuals may be more susceptible to air pollution-induced mortality or reflect differences in the nature of the risk presented by air pollution relative to the risks studied in the relevant economics literature.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's WTP to improve one's own chances of survival plus WTP to improve other individuals' survival rates. The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

Although a survival curve approach provides a theoretically preferred method for valuing the benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value reductions in premature mortality risk using the VSL approach.

Other uncertainties specific to premature mortality valuation include the following:

- *Across-study variation:* There is considerable uncertainty as to whether the available literature on VSL provides adequate estimates of the VSL for risk reductions from air pollution reduction. Although there is considerable variation in the analytical designs and data used in the existing literature, the majority of the studies involve the value of risks to a middle-aged working population. Most of the studies examine differences in wages of risky occupations, using a hedonic wage approach. Certain characteristics of both the population affected and the mortality risk facing that

population are believed to affect the average WTP to reduce the risk. The appropriateness of a distribution of WTP based on the current VSL literature for valuing the mortality-related benefits of reductions in air pollution concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on the extent to which the risks being valued are similar and the extent to which the subjects in the studies are similar to the population affected by changes in pollution concentrations.

- *Level of risk reduction:* The transferability of estimates of the VSL from the wage-risk studies to the context of the PM NAAQS analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study provides a result that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is 10 times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the VSL does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al., 1998).

- *Voluntariness of risks evaluated:* Although job-related mortality risks may differ in several ways from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. Some evidence suggests that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

- *Sudden versus protracted death:* A final important difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events, whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of

sudden death. To the extent that the mortality risks addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.

- *Self-selection and skill in avoiding risk:* Recent research (Shogren and Stamland, 2002) suggests that VSL estimates based on hedonic wage studies may overstate the average value of a risk reduction. This is based on the fact that the risk-wage trade-off revealed in hedonic studies reflects the preferences of the marginal worker (i.e., that worker who demands the highest compensation for his risk reduction). This worker must have either a higher workplace risk than the average worker, a lower risk tolerance than the average worker, or both. However, the risk estimate used in hedonic studies is generally based on average risk, so the VSL may be upwardly biased because the wage differential and risk measures do not match.

- *Baseline risk and age:* Recent research (Smith, Pattanayak, and Van Houtven, 2006) finds that because individuals reevaluate their baseline risk of death as they age, the marginal value of risk reductions does not decline with age as predicted by some lifetime consumption models. This research supports findings in recent stated preference studies that suggest only small reductions in the value of mortality risk reductions with increasing age.

Chronic Bronchitis Valuation

The best available estimate of WTP to avoid a case of CB comes from Viscusi, Magat, and Huber (1991). The Viscusi, Magat, and Huber study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi, Magat, and Huber (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the Benefits Technical Support Document (TSD) for the Nonroad Diesel rulemaking (Abt Associates, 2003).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: the WTP to avoid a case of severe CB, as described by Viscusi, Magat, and Huber; the severity level of an average pollution-related case of CB (relative to that

of the case described by Viscusi, Magat, and Huber); and the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution, which is about $340,000 (2006$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

Nonfatal Myocardial Infarctions Valuation

The Agency has recently incorporated into its analyses the impact of air pollution on the expected number of nonfatal heart attacks, although it has examined the impact of reductions in other related cardiovascular endpoints. We were not able to identify a suitable WTP value for reductions in the risk of nonfatal heart attacks. Instead, we use a COI unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3% discount rate, we estimated a present discounted value in lost earnings (in 2006$) over 5 years due to a myocardial infarction of $8,774 for someone between the ages of 25 and 44, $12,932 for someone between the ages of 45 and 54, and $74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2006$) using a 7% discount rate are $7,855, $11,578, and $66,920, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. As such, we do not include lost earnings in the cost estimates for these age groups.

We found three possible sources in the literature of estimates of the direct medical costs of myocardial infarction:

- Wittels et al. (1990) estimated expected total medical costs of myocardial infarction over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is $144,111 in year 2006$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or

medical procedures being used. The authors note that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay for myocardial infarction may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in the year-2000 Agency for Healthcare Research and Quality (AHRQ) HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our nonfatal myocardial infarction cases), whose length of stay was therefore substantially shorter than it would be if they had not died.

- Eisenstein et al. (2001) estimated 10-year costs of $44,663 in 1997$, or $64,003 in 2006$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal myocardial infarction of $15,540 (in 1995$) and $1,051 annually thereafter. Converting to year 2006$, that would be $30,102 for a 5-year period (without discounting) or $38,113 for a 10-year period.

In summary, the three different studies provided significantly different values (see Table 6-9).

Table 6-9. Alternative Direct Medical Cost of Illness Estimates for Nonfatal Heart Attacks

Study	*Direct Medical Costs (2006$)*	*Over an x-Year Period, for x =*
Wittels et al. (1990)	$144,111[a]	5
Russell et al. (1998)	$30,102[b]	5
Eisenstein et al. (2001)	$64,003[b]	10
Russell et al. (1998)	$38,113[b]	10

[a] Wittels et al. (1990) did not appear to discount costs incurred in future years.

[b] Using a 3% discount rate. Discounted values as reported in the study.

As noted above, the estimates from these three studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we

used estimates for medical costs that similarly cover a 5-year period (i.e., estimates from Wittels et al. (1990) and Russell et al. (1998). We used a simple average of the two 5-year estimates, or $65,902, and added it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 6-10.

Table 6-10. Estimated Costs Over a 5-Year Period (in 2006$) of a Nonfatal Myocardial Infarction

Age Group	*Opportunity Cost*	*Medical Cost*[a]	*Total Cost*
0–24	$0	$84,955	$84,955
25–44	$10,757[b]	$84,955	$95,713
45–54	$15,855[b]	$84,955	$100,811
55–65	$91,647[b]	$84,955	$176,602
> 65	$0	$84,955	$84,955

[a] An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998).

[b] From Cropper and Krupnick (1990), using a 3% discount rate.

6.4.5 Hospital Admissions Valuation

In the absence of estimates of societal WTP to avoid hospital visits/admissions for specific illnesses, estimates of total cost of illness (total medical costs plus the value of lost productivity) typically are used as conservative, or lower bound, estimates. These estimates are biased downward, because they do not include the willingness-to-pay value of avoiding pain and suffering.

The International Classification of Diseases (ICD-9, 1979) code-specific COI estimates used in this analysis consist of estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). We based all estimates of hospital charges and length of stays on statistics provided by the Agency for Healthcare Research and Quality (AHRQ 2000). We estimated the opportunity cost of a day spent in the hospital as the value of the lost daily wage, regardless of whether the hospitalized individual is in the workforce. To estimate the lost daily wage, we divided the 1990 median weekly wage by five and inflated the result to year 2006$ using the CPI-U "all items." The resulting estimate is $127.93. The total cost-of-illness estimate for an ICD code-specific hospital stay lasting n days, then, was the mean hospital charge plus $127.93 multiplied by n.

Table 6-11. Unit Values for Economic Valuation of Health Endpoints (2006$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Distributions of Estimates
	2000 Income Level	2016 Income Level	
Premature Mortality (Value of a Statistical Life)	$6,300,000	$8,600,000	EPA currently recommends a central VSL of $6.3m (2000$) based on a Weibull distribution fitted to 26 published VSL estimates (5 contingent valuation and 21 labor market studies). The underlying studies, the distribution parameters, and other useful information are available in Appendix B of EPA's current Guidelines for Preparing Economic Analyses (U.S. EPA, 2000).
Chronic Bronchitis (CB)	$340,000	$470,000	The WTP to avoid a case of pollution-related CB is calculated as where x is the severity of an average CB case, WTP_{13} is the WTP for a severe case of CB, and $ is the parameter relating WTP to severity, based on the regression results reported in Krupnick and Cropper (1992). The distribution of WTP for an average severity-level case of CB was generated by Monte Carlo methods, drawing from each of three distributions: (1) WTP to avoid a severe case of CB is assigned a 1/9 probability of being each of the first nine deciles of the distribution of WTP responses in Viscusi et al. (1991); (2) the severity of a pollution-related case of CB (relative to the case described in the Viscusi study) is assumed to have a triangular distribution, with the most likely value at severity level 6.5 and endpoints at 1.0 and 12.0; and (3) the constant in the elasticity of WTP with respect to severity is normally distributed with mean = 0.18 and standard deviation = 0.0669 (from Krupnick and Cropper [1992]). This process and the rationale for choosing it is described in detail in the Costs and Benefits of the Clean Air Act, 1990 to 2010 (U.S. EPA, 1999b).
Nonfatal Myocardial Infarction (heart attack)			No distributional information available. Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990). Direct medical costs are based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990).
3% discount rate			
Age 0–24	$79,685	$79,685	
Age 25–44	$88,975	$88,975	
Age 45–54	$93,897	$93,897	
Age 55–65	$167,532	$167,532	
Age 66 and over	$79,685	$79,685	
7% discount rate			Lost earnings:

Health Endpoint	Central Estimate of Value Per Statistical Incidence: 2000 Income Level	Central Estimate of Value Per Statistical Incidence: 2016 Income Level	Derivation of Distributions of Estimates
Age 0–24			Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings:
Age 25–44			
Age 45–54	$77,769	$77,769	
Age 55–65	$87,126	$87,126	age of onset: at 3% at 7'
Age 66 and over	$91,559	$91,559	25–44 $8,774 $7,855
	$157,477	$157,477	45–54 $12,932 11,578
	$77,769	$77,769	55–65 $74,746 66,920
			Direct medical expenses: An average of: 1. Wittels et al. (1990) ($102,658—no discounting) 2. Russell et al. (1998), 5-year period ($22,331 at 3% discount rate; $21,113 at 7% discount rate)
Hospital Admissions			
Chronic Obstructive Pulmonary Disease (COPD)	$16,606	$16,606	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
Asthma Admissions	$8,900	$8,900	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
All Cardiovascular	$24,668	$24,668	No distributional information available. The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov).
All respiratory (ages 65+)	$24,622	$24,622	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
All respiratory (ages 0–	$10,385	$10,385	No distributions available. The COI point

Health Endpoint	Central Estimate of Value Per Statistical Incidence		
	2000 Income Level	2016 Income Level	Derivation of Distributions of Estimates
2)			estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Emergency Room Visits for Asthma	$384	$384	No distributional information available. Simple average of two unit COI values: (1) $311.55, from Smith et al. (1997) and (2) $260.67, from Stanford et al. (1999).
Respiratory Ailments Not Requiring Hospitalization			
Upper Respiratory Symptoms (URS)	$30	$30	Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, we assumed a uniform distribution between $9.2 and $43.1.
Lower Respiratory Symptoms (LRS)	$16	$19	Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. In the absence of information surrounding the frequency with which each of the 11 types of LRS occurs within the LRS symptom complex, we assumed a uniform distribution between $6.9 and $24.46.
Asthma Exacerbations	$43	$53	Asthma exacerbations are valued at $45 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Distributions of Estimates
	2000 Income Level	2016 Income Level	
			estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $15.6 and $70.8.
Acute Bronchitis	$360	$440	Assumes a 6-day episode, with the distribution of the daily value specified as uniform with the low and high values based on those recommended for related respiratory symptoms in Neumann et al. (1994). The low daily estimate of $10 is the sum of the mid-range values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high daily estimate was taken to be twice the value of a minor respiratory restricted-activity day, or $110.
Work Loss Days (WLDs)	Variable (U.S. median = $130)	Variable (U.S. median = $130)	No distribution available. Point estimate is based on county-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc.
Minor Restricted Activity Days (MRADs)	$51	$62	Median WTP estimate to avoid one MRAD from Tolley et al. (1986). Distribution is assumed to be triangular with a minimum of $22 and a maximum of $83, with a most likely value of $52. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom—for eye irritation—is $16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme.

Asthma-Related Emergency Room Visits Valuation

To value asthma emergency room visits, we used a simple average of two estimates from the health economics literature. The first estimate comes from Smith et al. (1997), who reported approximately 1.2 million asthma-related emergency room visits in 1987, at a total cost of $186.5 million (1987$). The average cost per visit that year was $155; in 2006$, that cost was

$400.88 (using the CPI-U for medical care to adjust to 2006$). The second estimate comes from Stanford et al. (1999), who reported the cost of an average asthma-related emergency room visit at $335.14, based on 1996–1997 data. A simple average of the two estimates yields a (rounded) unit value of $368.

Minor Restricted Activity Days Valuation

No studies are reported to have estimated WTP to avoid a minor restricted activity day. However, one of EPA's contractors, IEc (1994) has derived an estimate of willingness to pay to avoid a minor *respiratory* restricted activity day, using estimates from Tolley et al. (1986) of WTP for avoiding a combination of coughing, throat congestion and sinusitis. The IEc estimate of WTP to avoid a minor respiratory restricted activity day is $38.37 (1990$), or about $62.04 (2006$).

Although Ostro and Rothschild (1989) statistically linked ozone and minor restricted activity days, it is likely that most MRADs associated with ozone exposure are, in fact, minor *respiratory* restricted activity days. For the purpose of valuing this health endpoint, we used the estimate of mean WTP to avoid a minor respiratory restricted activity day.

Visibility Valuation

Reductions in NO_2 and SO_2 emissions along with the secondary formation of $PM_{2.5}$ would improve the level of visibility throughout the United States because these suspended particles and gases degrade visibility by scattering and absorbing light (U.S. EPA, 2009d). Visibility has direct significance to people's enjoyment of daily activities and their overall sense of wellbeing (U.S. EPA, 2009d). Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smokey Mountains National Park. This section discusses the measurement of the economic benefits of improved visibility.

Visual air quality (VAQ) is commonly measured as either light extinction, which is defined as the loss of light per unit of distance in terms of inverse megameters (Mm^{-1}) or the deciview (dv) metric (Pitchford and Malm, 1993), which is a logarithmic function of extinction. Extinction and deciviews are physical measures of the amount of visibility impairment (e.g., the amount of "haze"), with both extinction and deciview increasing as the amount of haze increases. Pitchford and Malm characterize a change of one deciview as "a small but perceptible scenic change under many circumstances." Light extinction is the optical characteristic of the atmosphere that occurs when light is either scattered or absorbed, which converts the light to heat. Particulate matter and gases can both scatter and absorb light. Fine particles with

Figure 6-4. Mandatory Class I Areas in the U.S.

Annual average visibility conditions (reflecting light extinction due to both anthropogenic and non-anthropogenic sources) vary regionally across the U.S. (U.S. EPA, 2009d). The rural East generally has higher levels of impairment than remote sites in the West, with the exception of urban-influenced sites such as San Gorgonio Wilderness (CA) and Point Reyes National Seashore (CA), which have annual average levels comparable to certain sites in the Northeast (U.S. EPA, 2004). Higher visibility impairment levels in the East are due to generally higher concentrations of fine particles, particularly sulfates, and higher average relative humidity levels. While visibility trends have improved in most Class I areas, the recent data show that these areas continue to suffer from visibility impairment. In eastern parks, average visual range has decreased from 90 miles to 15-25 miles, and in the West, visual range has decreased from 140 miles to 35-90 miles (U.S. EPA, 2004; U.S. EPA, 1999b).

EPA distinguishes benefits from two categories of visibility changes: residential visibility and recreational visibility. In both cases economic benefits are believed to consist of use values and nonuse values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and birdwatching. Nonuse values are based on people's beliefs that the environment ought to exist

free of human-induced haze. Nonuse values may be more important for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas. In previous assessments, EPA used a study on residential visibility valuation conducted in 1990 (McClelland et al., 1993). Subsequently, EPA designated the McClelland et al. study as significantly less reliable for regulatory benefit-cost analysis consistent with SAB advice (U.S. EPA-SAB, 1999). Although a wide range of published, peer-review literature supports a non-zero value for residential visibility (Brookshire et al., 1982; Rae, 1983; Tolley et al., 1986; Chestnut and Rowe, 1990c; McClleand et al., 1993; Loehman et al., 1994), the residential visibility benefits have not been calculated in this analysis.

For recreational visibility, only one existing study provides defensible monetary estimates of the value of visibility changes in a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Although there are a number of other studies in the literature, they were conducted in the early 1980s and did not use methods that are considered defensible by current standards. The Chestnut and Rowe study uses the CV method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[1] This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates.

For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas.[2] A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the United States are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[3] The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions

[1] An SAB advisory letter indicates that "many members of the Council believe that the Chestnut and Rowe study is the best available" (EPA-SAB-COUNCIL-ADV-00-002, 1999, p. 13). However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications (Chestnut and Dennis, 1997).

[2] The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

[3] For details of the visibility estimates discussed in this chapter, please refer to the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their WTP to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the United States. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the Benefits TSD for the Nonroad Diesel rulemaking (Abt Associates, 2003).

The Chestnut and Rowe study (Chestnut and Rowe, 1990a; 1990b), although representing the best available estimates, has a number of limitations. These include the following:

- The age of the study (late 1980s) will increase the uncertainty about the correspondence of the estimated values to those that might be provided by current or future populations.

- The survey focused only on populations in five states, so the application of the estimated values to populations outside those states requires that preferences of populations in the five surveyed states be similar to those of non-surveyed states.

- There is an inherent difficulty in separating values expressed for visibility improvements from an overall value for improved air quality. The Chestnut and Rowe study attempted to control for this by informing respondents that "other households are being asked about visibility, human health, and vegetation protections in urban areas and at national parks in other regions." However, most of the respondents did not feel that they were able to segregate visibility at national parks entirely from residential visibility and health effects.

- It is not clear exactly what visibility improvements the respondents to the Chestnut and Rowe survey were valuing. The WTP question asked about changes in average visibility, but the survey respondents were shown photographs of only summertime

conditions, when visibility is generally at its worst. It is possible that the respondents believed those visibility conditions held year-round, in which case they would have been valuing much larger overall improvements in visibility than what otherwise would be the case. For the purpose of the benefits analysis for this rule, EPA assumed that respondents provided values for changes in annual average visibility. Because most policies will result in a shift in the distribution of visibility (usually affecting the worst days more than the best days), the annual average may not be the most relevant metric for policy analysis.

- The survey did not include reminders of possible substitutes (e.g., visibility at other parks) or budget constraints. These reminders are considered to be best practice for stated preference surveys.

- The Chestnut and Rowe survey focused on visibility improvements in and around national parks and wilderness areas. The survey also focused on visibility improvements of national parks in the southwest United States. Given that national parks and wilderness areas exhibit unique characteristics, it is not clear whether the WTP estimate obtained from Chestnut and Rowe can be transferred to other national parks and wilderness areas, without introducing additional uncertainty.

In general, the survey design and implementation reflect the period in which the survey was conducted. Since that time, many improvements to the stated preference methodology have been developed. As future survey efforts are completed, EPA will incorporate values for visibility improvements reflecting the improved survey designs.

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the reductions in precursor emissions associated with this rule. A general WTP equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from this rule. The method for developing calibrated WTP functions is based on the approach developed by Smith et al. (2002). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1% increase in income is associated with a 0.9% increase in WTP for a given change in

visibility. A more detailed explanation of the visibility benefits methodology is provided in Appendix I of the PM NAAQS RIA (U.S. EPA, 2006).

One major source of uncertainty for the visibility benefits estimate is the benefits transfer process used. Judgments used to choose the functional form and key parameters of the estimating equation for WTP for the affected population could have significant effects on the size of the estimates. Assumptions about how individuals respond to changes in visibility that are either very small or outside the range covered in the Chestnut and Rowe study could also affect the results.

In addition, our estimate of visibility benefits is incomplete. For example, we anticipate improvement in visibility in residential areas for which we are currently unable to monetize benefits, such as the Northeastern and Central regions of the U.S. The value of visibility benefits in areas where we were unable to monetize benefits could also be substantial. EPA requests public comment on the approach taken here to quantify the monetary value of changes in visibility in Class I areas.

Growth in WTP Reflecting National Income Growth Over Time

Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity[1] of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases, the WTP for environmental improvements also increases. Although many analyses assume that the income elasticity of WTP is unit elastic (i.e., a 10% higher real income level implies a 10% higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two different ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the Science Advisory Board (SAB)

[1] Income elasticity is a common economic measure equal to the percentage change in WTP for a 1% change in income.

advised EPA to adjust WTP for increases in real income over time but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (U.S. EPA-SAB, 2000a). A recent advisory by another committee associated with the SAB, the Advisory Council on Clean Air Compliance Analysis, has provided conflicting advice. While agreeing with "the general principle that the willingness to pay to reduce mortality risks is likely to increase with growth in real income (U.S. EPA-SAB, 2004a, p. 52)" and that "The same increase should be assumed for the WTP for serious nonfatal health effects (U.S. EPA-SAB, 2004a, p. 52)," they note that "given the limitations and uncertainties in the available empirical evidence, the Council does not support the use of the proposed adjustments for aggregate income growth as part of the primary analysis (U.S. EPA-SAB, 2004a, p. 53)." Until these conflicting advisories have been reconciled, EPA will continue to adjust valuation estimates to reflect income growth using the methods described below, while providing sensitivity analyses for alternative income growth adjustment factors.

Based on a review of the available income elasticity literature, we adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2016 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Note that because of the variety of empirical sources used in deriving the income elasticities, there may appear to be inconsistencies in the magnitudes of the income elasticities relative to the severity of the effects (*a priori* one might expect that more severe outcomes would show less income elasticity of WTP). We have not imposed any additional restrictions on the empirical estimates of income elasticity. One explanation for the seeming inconsistency is the difference in timing of conditions. WTP for minor illnesses is often expressed as a short term payment to avoid a single episode. WTP for major illnesses and mortality risk reductions are based on longer term measures of payment (such as wages or annual income). Economic theory suggests that relationships become more elastic as the length of time grows, reflecting the ability to adjust spending over a longer time period. Based on this theory, it would be expected that WTP for reducing long term risks would

be more elastic than WTP for reducing short term risks. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The relative magnitude of the income elasticity of WTP for visibility compared with those for health effects suggests that visibility is not as much of a necessity as health, thus, WTP is more elastic with respect to income. The elasticity values used to adjust estimates of benefits in 2016 are presented in Table 6-12.

Table 6-12. Elasticity Values Used to Account for Projected Real Income Growth[a]

Benefit Category	*Central Elasticity Estimate*
Minor Health Effect	0.14
Severe and Chronic Health Effects	0.45
Premature Mortality	0.40
Visibility	0.90

[a] Derivation of estimates can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

In addition to elasticity estimates, projections of real gross domestic product (GDP) and populations from 1990 to 2020 are needed to adjust benefits to reflect real per capita income growth. For consistency with the emissions and benefits modeling, we used national population estimates for the years 1990 to 1999 based on U.S. Census Bureau estimates (Hollman, Mulder, and Kallan, 2000). These population estimates are based on application of a cohort-component model applied to 1990 U.S. Census data projections (U.S. Bureau of Census, 2000). For the years between 2000 and 2016, we applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. We used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.[1] We used projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2016.[2]

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, we calculated WTP adjustment factors for each of the elasticity estimates listed in Table 6-13. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. Note that, for premature mortality, we

[1] U.S. Bureau of Economic Analysis, Table 2A (1992$) (available at http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm.) and U.S. Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

[2] In previous analyses, we used the Standard and Poor's projections of GDP directly. This led to an apparent discontinuity in the adjustment factors between 2010 and 2011. We refined the method by applying the relative growth rates for GDP derived from the Standard and Poor's projections to the 2010 projected GDP based on the Bureau of Economic Analysis projections.

applied the income adjustment factor to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that because of a lack of data on the dependence of COI and income, and a lack of data on projected growth in average wages, no adjustments are made to benefits based on the COI approach or to work loss days and worker productivity. This assumption leads us to underpredict benefits in future years because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

Table 6-13. Adjustment Factors Used to Account for Projected Real Income Growth[a]

Benefit Category	*2016*
Minor Health Effect	1.06
Severe and Chronic Health Effects	1.19
Premature Mortality	1.16
Visibility	1.41

[a] Based on elasticity values reported in Table 6-3, U.S. Census population projections, and projections of real GDP per capita.

6.5 Unquantified Health and Welfare Benefits

This analysis is limited by the available data and resources. As such, we are not able to quantify several welfare benefit categories, as shown in Table 6-2. In this section, we provide a qualitative assessment of some of the primary welfare benefit categories from reducing NO_2 and SO_2 emissions: health and ecosystem benefits of reducing nitrogen and sulfur emissions and deposition and vegetation benefits from reducing ozone.[1] While we were unable to quantify how large these benefits might be as a result of the emission reductions achieved by this rule, previous EPA assessments show that these benefits could be substantial (U.S. EPA, 2008f; U.S. EPA, 2009c; U.S. EPA, 2007b; U.S. EPA, 1999b). The omission of these endpoints from the monetized results should not imply that the impacts are small or unimportant.

6.5.1 Ecosystem Services

Ecosystem services can be generally defined as the benefits that individuals and organizations obtain from ecosystems. EPA has defined ecological goods and services as the "outputs of ecological functions or processes that directly or indirectly contribute to social welfare or have the potential to do so in the future. Some outputs may be bought and sold, but

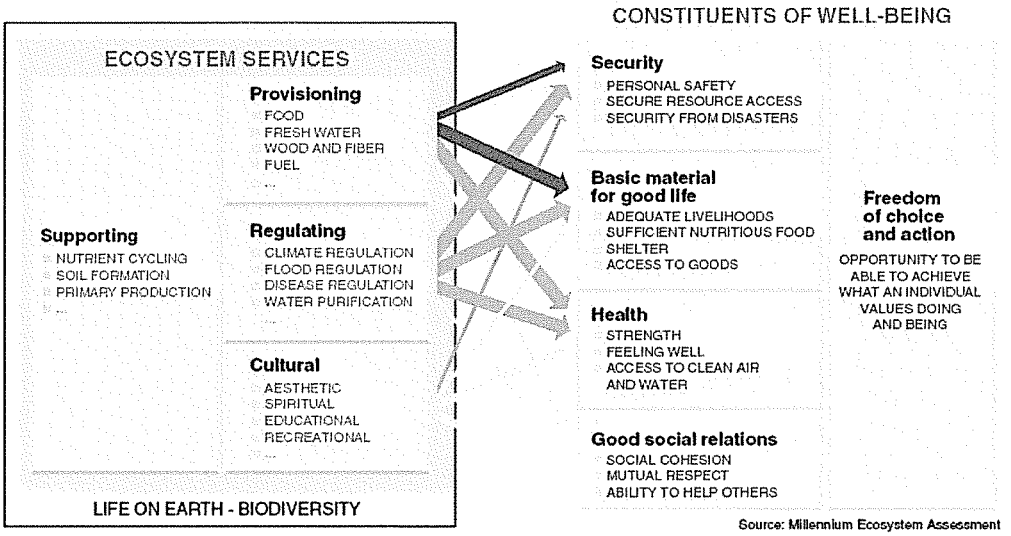
[1] Some quantitative estimates of the total value of certain recreational and environmental goods given current and historic emission levels are provided below. They do not reflect benefits that would accrue as a result of this result. However, these values would be expected to increase as emissions are decreased a result of this rule.

most are not marketed" (U.S. EPA, 2006b). Figure 6-5 provides the Millennium Ecosystem Assessment's schematic demonstrating the connections between the categories of ecosystem services and human well-being. The interrelatedness of these categories means that any one ecosystem may provide multiple services. Changes in these services can affect human well-being by affecting security, health, social relationships, and access to basic material goods (MEA, 2005).

In the Millennium Ecosystem Assessment (MEA, 2005), ecosystem services are classified into four main categories:

1. Provisioning: Products obtained from ecosystems, such as the production of food and water
2. Regulating: Benefits obtained from the regulation of ecosystem processes, such as the control of climate and disease
3. Cultural: Nonmaterial benefits that people obtain from ecosystems through spiritual enrichment, cognitive development, reflection, recreation, and aesthetic experiences
4. Supporting: Services necessary for the production of all other ecosystem services, such as nutrient cycles and crop pollination

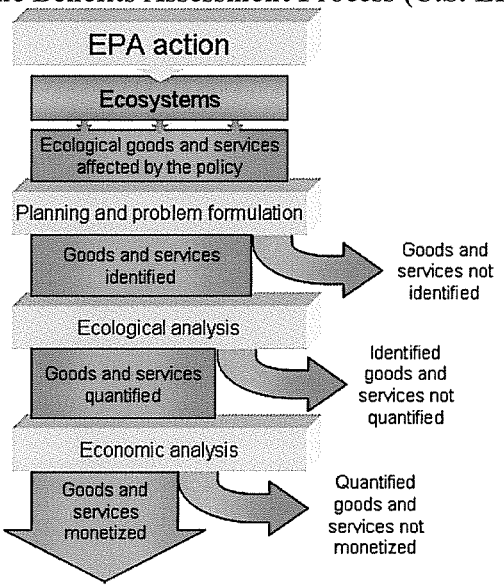
Figure 6-5. Linkages between Categories of Ecosystem Services and Components of Human Well-Being from Millennium Ecosystem Assessment (MEA, 2005)



The monetization of ecosystem services generally involves estimating the value of ecological goods and services based on what people are willing to pay (WTP) to increase ecological services or by what people are willing to accept (WTA) in compensation for reductions in them (U.S. EPA, 2006b). There are three primary approaches for estimating the monetary value of ecosystem services: market-based approaches, revealed preference methods,

and stated preference methods (U.S. EPA, 2006b). Because economic valuation of ecosystem services can be difficult, nonmonetary valuation using biophysical measurements and concepts also can be used. An example of a nonmonetary valuation method is the use of relative-value indicators (e.g., a flow chart indicating uses of a water body, such as boatable, fishable, swimmable, etc.). It is necessary to recognize that in the analysis of the environmental responses associated with any particular policy or environmental management action, only a subset of the ecosystem services likely to be affected are readily identified. Of those ecosystem services that are identified, only a subset of the changes can be quantified. Within those services whose changes can be quantified, only a few will likely be monetized, and many will remain nonmonetized. The stepwise concept leading up to the valuation of ecosystems services is graphically depicted in Figure 6-6.

Figure 6-6. Schematic of the Benefits Assessment Process (U.S. EPA, 2006b)



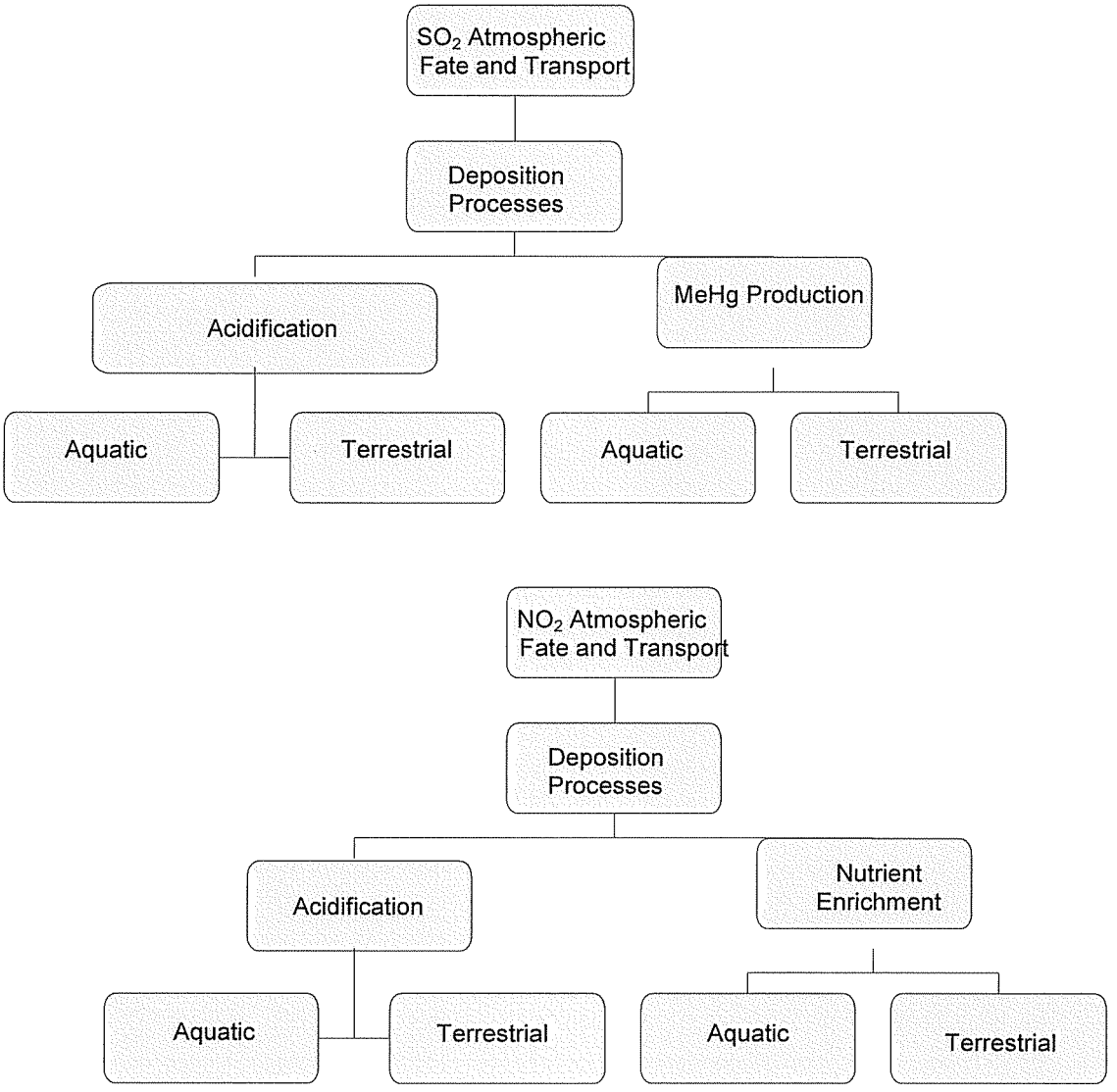
6.5.2 Ecosystem Benefits of Reduced Nitrogen and Sulfur Deposition

Science of Deposition

Nitrogen and sulfur emissions occur over large regions of North America. Once these pollutants are lofted to the middle and upper troposphere, they typically have a much longer lifetime and, with the generally stronger winds at these altitudes, can be transported long distances from their source regions. The length scale of this transport is highly variable owing to differing chemical and meteorological conditions encountered along the transport path (U.S. EPA, 2008f). Sulfur is primarily emitted as SO_2, and nitrogen can be emitted as NO, NO_2, or NH_3. Secondary particles are formed from NO_x and SO_X gaseous emissions and associated

chemical reactions in the atmosphere. Deposition can occur in either a wet (i.e., rain, snow, sleet, hail, clouds, or fog) or dry form (i.e., gases or particles). Together these emissions are deposited onto terrestrial and aquatic ecosystems across the U.S., contributing to the problems of acidification, nutrient enrichment, and methylmercury production as represented in Figure 6-7. Although there is some evidence that nitrogen deposition may have positive effects on agricultural and forest output through passive fertilization, it is likely that the overall value is very small relative to other health and welfare effects.

Figure 6-7. Schematics of Ecological Effects of Nitrogen and Sulfur Deposition



The lifetimes of particles vary with particle size. Accumulation-mode particles such as sulfates are kept in suspension by normal air motions and have a lower deposition velocity than coarse-mode particles; they can be transported thousands of kilometers and remain in the atmosphere for a number of days. They are removed from the atmosphere primarily by cloud processes. Particulates affect acid deposition by serving as cloud condensation nuclei and contribute directly to the acidification of rain. In addition, the gas-phase species that lead to the dry deposition of acidity are also precursors of particles. Therefore, reductions in NO_2 and SO_2 emissions will decrease both acid deposition and PM concentrations, but not necessarily in a linear fashion. (U.S. EPA, 2008f). Sulfuric acid is also deposited on surfaces by dry deposition and can contribute to environmental effects (U.S. EPA, 2008f).

Ecological Effects of Acidification

Deposition of nitrogen and sulfur can cause acidification, which alters biogeochemistry and affects animal and plant life in terrestrial and aquatic ecosystems across the U.S. Soil acidification is a natural process, but is often accelerated by acidifying deposition, which can decrease concentrations of exchangeable base cations in soils (U.S. EPA, 2008f). Major terrestrial effects include a decline in sensitive tree species, such as red spruce (*Picea rubens*) and sugar maple (*Acer saccharum*) (U.S. EPA, 2008f). Biological effects of acidification in terrestrial ecosystems are generally linked to aluminum toxicity and decreased ability of plant roots to take up base cations (U.S. EPA, 2008f). Decreases in the acid neutralizing capacity and increases in inorganic aluminum concentration contribute to declines in zooplankton, macro invertebrates, and fish species richness in aquatic ecosystems (U.S. EPA, 2008f).

Geology (particularly surficial geology) is the principal factor governing the sensitivity of terrestrial and aquatic ecosystems to acidification from nitrogen and sulfur deposition (U.S. EPA, 2008f). Geologic formations having low base cation supply generally underlie the watersheds of acid-sensitive lakes and streams. Other factors contribute to the sensitivity of soils and surface waters to acidifying deposition, including topography, soil chemistry, land use, and hydrologic flow path (U.S. EPA, 2008f).

Aquatic Ecosystems

Aquatic effects of acidification have been well studied in the U.S. and elsewhere at various trophic levels. These studies indicate that aquatic biota have been affected by acidification at virtually all levels of the food web in acid sensitive aquatic ecosystems. Effects have been most clearly documented for fish, aquatic insects, other invertebrates, and algae. Biological effects are primarily attributable to a combination of low pH and high inorganic

aluminum concentrations. Such conditions occur more frequently during rainfall and snowmelt that cause high flows of water and less commonly during low-flow conditions, except where chronic acidity conditions are severe. Biological effects of episodes include reduced fish condition factor[1], changes in species composition and declines in aquatic species richness across multiple taxa, ecosystems and regions. These conditions may also result in direct fish mortality (Van Sickle et al., 1996). Biological effects in aquatic ecosystems can be divided into two major categories: effects on health, vigor, and reproductive success; and effects on biodiversity. Surface water with ANC values greater than 50 μeq/L generally provides moderate protection for most fish (i.e., brook trout, others) and other aquatic organisms (U.S. EPA, 2009c). Table 6-14 provides a summary of the biological effects experienced at various ANC levels.

Table 6-14. Aquatic Status Categories

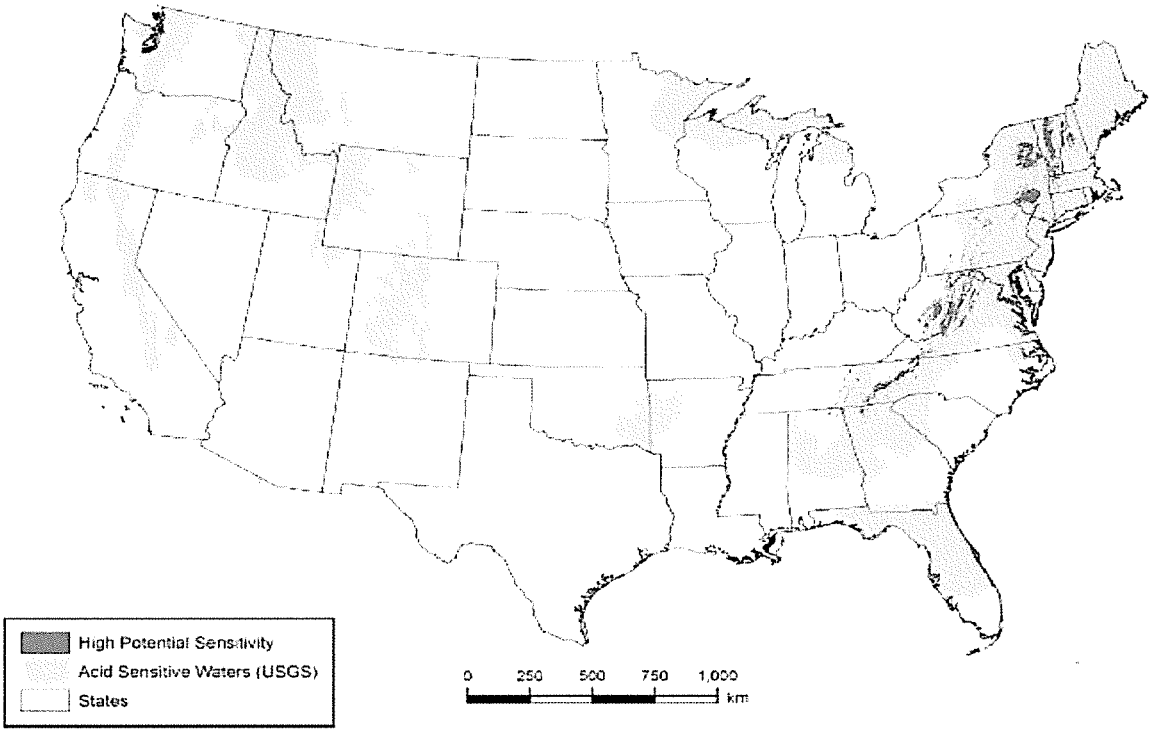
Category Label	ANC Levels	Expected Ecological Effects
Acute Concern	<0 micro equivalent per Liter (μeq/L)	Near complete loss of fish populations is expected. Planktonic communities have extremely low diversity and are dominated by acidophilic forms. The number of individuals in plankton species that are present is greatly reduced.
Severe Concern	0–20 μeq/L	Highly sensitive to episodic acidification. During episodes of high acidifying deposition, brook trout populations may experience lethal effects. Diversity and distribution of zooplankton communities decline sharply.
Elevated Concern	20–50 μeq/L	Fish species richness is greatly reduced (i.e., more than half of expected species can be missing). On average, brook trout populations experience sublethal effects, including loss of health, reproduction capacity, and fitness. Diversity and distribution of zooplankton communities decline.
Moderate Concern	50–100 μeq/L	Fish species richness begins to decline (i.e., sensitive species are lost from lakes). Brook trout populations are sensitive and variable, with possible sublethal effects. Diversity and distribution of zooplankton communities also begin to decline as species that are sensitive to acidifying deposition are affected.
Low Concern	>100 μeq/L	Fish species richness may be unaffected. Reproducing brook trout populations are expected where habitat is suitable. Zooplankton communities are unaffected and exhibit expected diversity and distribution.

A number of national and regional assessments have been conducted to estimate the distribution and extent of surface water acidity in the U.S. (U.S. EPA, 2008f). As a result, several regions of the U.S. have been identified as containing a large number of lakes and

[1] Condition factor is an index that describes the relationship between fish weight and length, and is one measure of sublethal acidification stress that has been used to quantify effects of acidification on an individual fish (U.S.EPA, 2008f).

streams that are seriously impacted by acidification. Figure 6-8 illustrates those areas of the U.S. where aquatic ecosystems are at risk from acidification.

Figure 6-8. Areas Potentially Sensitive to Aquatic Acidification (U.S. EPA, 2008f)



Because acidification primarily affects the diversity and abundance of aquatic biota, it also affects the ecosystem services that are derived from the fish and other aquatic life found in these surface waters.

While acidification is unlikely to have serious negative effects on, for example, water supplies, it can limit the productivity of surface waters as a source of food (i.e., fish). In the northeastern United States, the surface waters affected by acidification are not a major source of commercially raised or caught fish; however, they are a source of food for some recreational and subsistence fishermen and for other consumers. For example, there is evidence that certain population subgroups in the northeastern United States, such as the Hmong and Chippewa ethnic groups, have particularly high rates of self-caught fish consumption (Hutchison and Kraft, 1994; Peterson et al., 1994). However, it is not known if and how their consumption patterns are affected by the reductions in available fish populations caused by surface water acidification.

Inland surface waters support several cultural services, including aesthetic and educational services and recreational fishing. Recreational fishing in lakes and streams is among

the most popular outdoor recreational activities in the northeastern United States. Based on studies conducted in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $36 for recreational fishing (in 2007 dollars); therefore, the implied total annual value of freshwater fishing in the northeastern United States was $5.1 billion in 2006.[1] For recreation days, consumer surplus value is most commonly measured using recreation demand, travel cost models.

Another estimate of the overarching ecological benefits associated with reducing lake acidification levels in Adirondacks National Park can be derived from the contingent valuation (CV) survey (Banzhaf et al., 2006), which elicited values for specific improvements in acidification-related water quality and ecological conditions in Adirondack lakes. The survey described a base version with minor improvements said to result from the program, and a scope version with large improvements due to the program and a gradually worsening status quo. After adapting and transferring the results of this study and converting the 10-year annual payments to permanent annual payments using discount rates of 3% and 5%, the WTP estimates ranged from $48 to $107 per year per household (in 2004 dollars) for the base version and $54 to $154 for the scope version. Using these estimates, the aggregate annual benefits of eliminating all anthropogenic sources of NO_x and SO_x emissions were estimated to range from $291 million to $829 million (U.S. EPA, 2009c).[2]

In addition, inland surface waters provide a number of regulating services associated with hydrological and climate regulation by providing environments that sustain aquatic food webs. These services are disrupted by the toxic effects of acidification on fish and other aquatic life. Although it is difficult to quantify these services and how they are affected by acidification, some of these services may be captured through measures of provisioning and cultural services.

Terrestrial Ecosystems

Acidifying deposition has altered major biogeochemical processes in the U.S. by increasing the nitrogen and sulfur content of soils, accelerating nitrate and sulfate leaching from soil to drainage waters, depleting base cations (especially calcium and magnesium) from soils, and increasing the mobility of aluminum. Inorganic aluminum is toxic to some tree roots. Plants affected by high levels of aluminum from the soil often have reduced root growth, which restricts the ability of the plant to take up water and nutrients, especially calcium (U. S. EPA, 2008f).

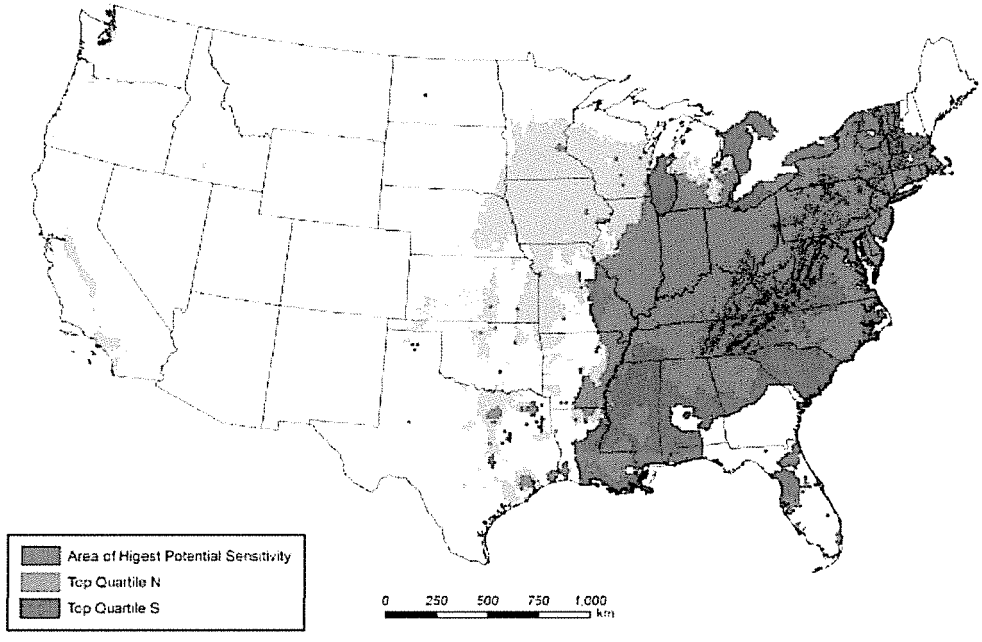
[1] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

[2] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

These direct effects can, in turn, influence the response of these plants to climatic stresses such as droughts and cold temperatures. They can also influence the sensitivity of plants to other stresses, including insect pests and disease (Joslin et al., 1992) leading to increased mortality of canopy trees. In the U.S., terrestrial effects of acidification are best described for forested ecosystems (especially red spruce and sugar maple ecosystems) with additional information on other plant communities, including shrubs and lichen (U.S. EPA, 2008f).

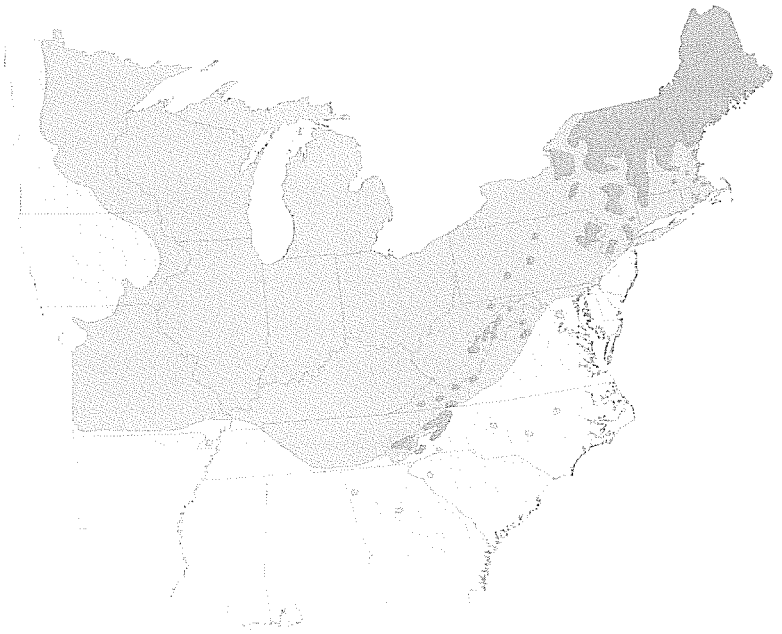
Certain ecosystems in the continental U.S. are potentially sensitive to terrestrial acidification, which is the greatest concern regarding nitrogen and sulfur deposition U.S. EPA (2008f). Figure 6-9 depicts the areas across the U.S. that are potentially sensitive to terrestrial acidification.

Figure 6-9. Areas Potentially Sensitive to Terrestrial Acidification (U.S. EPA, 2008f)



Both coniferous and deciduous forests throughout the eastern U.S. are experiencing gradual losses of base cation nutrients from the soil due to accelerated leaching from acidifying deposition. This change in nutrient availability may reduce the quality of forest nutrition over the long term. Evidence suggests that red spruce and sugar maple in some areas in the eastern U.S. have experienced declining health because of this deposition. For red spruce, (*Picea rubens*) dieback or decline has been observed across high elevation landscapes of the northeastern U.S., and to a lesser extent, the southeastern U.S., and acidifying deposition has been implicated as a causal factor (DeHayes et al., 1999). Figure 6-10 shows the distribution of red spruce (brown) and sugar maple (green) in the eastern U.S.

Figure 6-10. Distribution of Red Spruce (pink) and Sugar Maple (green) in the Eastern U.S. (U.S. EPA, 2008f)



Terrestrial acidification affects several important ecological endpoints, 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).

Forests in the northeastern United States provide several important and valuable provisioning services in the form of tree products. Sugar maples are a particularly important commercial hardwood tree species, providing timber and maple syrup. In the United States, sugar maple saw timber was nearly 900 million board feet in 2006 (USFS, 2006), and annual production of maple syrup was nearly 1.4 million gallons, accounting for approximately 19% of worldwide production. The total annual value of U.S. production in these years was approximately $160 million (NASS, 2008). Red spruce is also used in a variety of products including lumber, pulpwood, poles, plywood, and musical instruments. The total removal of red spruce saw timber from timberland in the United States was over 300 million board feet in 2006 (USFS, 2006).

Forests in the northeastern United States are also an important source of cultural ecosystem services—nonuse (i.e., existence value for threatened and endangered species), recreational, and aesthetic services. Red spruce forests are home to two federally listed species and one delisted species:

1. Spruce-fir moss spider (*Microhexura montivaga*)—endangered
2. Rock gnome lichen (*Gymnoderma lineare*)—endangered
3. Virginia northern flying squirrel (*Glaucomys sabrinus fuscus*)—delisted, but important

Forestlands support a wide variety of outdoor recreational activities, including fishing, hiking, camping, off-road driving, hunting, and wildlife viewing. Regional statistics on recreational activities that are specifically forest based are not available; however, more general data on outdoor recreation provide some insights into the overall level of recreational services provided by forests. More than 30% of the U.S. adult population visited a wilderness or primitive area during the previous year and engaged in day hiking (Cordell et al., 2008). From 1999 to 2004, 16% of adults in the northeastern United States participated in off-road vehicle recreation, for an average of 27 days per year (Cordell et al., 2005). The average consumer surplus value per day of off-road driving in the United States was $25 (in 2007 dollars), and the implied total annual value of off-road driving recreation in the northeastern United States was more than $9 billion (Kaval and Loomis, 2003). More than 5% of adults in the northeastern United States participated in nearly 84 million hunting days (U.S. FWS and U.S. Census Bureau, 2007). Ten percent of adults in northeastern states participated in wildlife viewing away from home on 122 million days in 2006. For these recreational activities in the northeastern United States, Kaval and Loomis (2003) estimated average consumer surplus values per day of $52 for hunting and $34 for wildlife viewing (in 2007 dollars). The implied total annual value of hunting and wildlife viewing in the northeastern United States was, therefore, $4.4 billion and $4.2 billion, respectively, in 2006.

As previously mentioned, it is difficult to estimate the portion of these recreational services that are specifically attributable to forests and to the health of specific tree species. However, one recreational activity that is directly dependent on forest conditions is fall color viewing. Sugar maple trees, in particular, are known for their bright colors and are, therefore, an essential aesthetic component of most fall color landscapes. A survey of residents in the Great Lakes area found that roughly 30% of residents reported at least one trip in the previous year involving fall color viewing (Spencer and Holecek, 2007). In a separate study conducted in Vermont, Brown (2002) reported that more than 22% of households visiting Vermont in 2001 made the trip primarily for viewing fall colors.

Two studies estimated values for protecting high-elevation spruce forests in the southern Appalachian Mountains. Kramer et al. (2003) conducted a contingent valuation study estimating households' WTP for programs to protect remaining high-elevation spruce forests from damages associated with air pollution and insect infestation. Median household WTP was estimated to be

roughly $29 (in 2007 dollars) for a smaller program, and $44 for the more extensive program. Jenkins et al. (2002) conducted a very similar study in seven Southern Appalachian states on a potential program to maintain forest conditions at status quo levels. The overall mean annual WTP for the forest protection programs was $208 (in 2007 dollars). Multiplying the average WTP estimate from these studies by the total number of households in the seven-state Appalachian region results in an aggregate annual range of $470 million to $3.4 billion for avoiding a significant decline in the health of high-elevation spruce forests in the Southern Appalachian region.[1]

Forests in the northeastern United States also support and provide a wide variety of valuable regulating services, including soil stabilization and erosion control, water regulation, and climate regulation. The total value of these ecosystem services is very difficult to quantify in a meaningful way, as is the reduction in the value of these services associated with total nitrogen and sulfur deposition. As terrestrial acidification contributes to root damages, reduced biomass growth, and tree mortality, all of these services are likely to be affected; however, the magnitude of these impacts is currently very uncertain.

6.5.3 Ecological Effects Associated with Gaseous Sulfur Dioxide

Uptake of gaseous sulfur dioxide in a plant canopy is a complex process involving adsorption to surfaces (leaves, stems, and soil) and absorption into leaves. SO_2 penetrates into leaves through to the stomata, although there is evidence for limited pathways via the cuticle. Pollutants must be transported from the bulk air to the leaf boundary layer in order to get to the stomata. When the stomata are closed, as occurs under dark or drought conditions, resistance to gas uptake is very high and the plant has a very low degree of susceptibility to injury. In contrast, mosses and lichens do not have a protective cuticle barrier to gaseous pollutants or stomates and are generally more sensitive to gaseous sulfur and nitrogen than vascular plants (U.S. EPA, 2008f). Acute foliar injury usually happens within hours of exposure, involves a rapid absorption of a toxic dose, and involves collapse or necrosis of plant tissues. Another type of visible injury is termed chronic injury and is usually a result of variable SO_2 exposures over the growing season. Besides foliar injury, chronic exposure to low SO_2 concentrations can result in reduced photosynthesis, growth, and yield of plants (U.S. EPA, 2008f). These effects are cumulative over the season and are often not associated with visible foliar injury. As with foliar injury, these effects vary among species and growing environment. SO_2 is also considered the

[1] These estimates reflect the marginal value of the service for the hypothetical program described in the survey, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

primary factor causing the death of lichens in many urban and industrial areas (Hutchinson et al., 1996).

In addition to the role of sulfate deposition on methylation, the technologies installed to reduce emissions of NO_x and SO_2 associated with this proposed rule would also reduce mercury emissions. EPA recently commissioned an information collection request that will soon provide greatly improved power industry mercury emissions estimates that will enable the Agency to better estimate mercury emissions changes from its air emissions control actions. For this reason, the Agency did not estimate Hg changes in this rule and will instead wait for these new data which will be available in the near future. Due to time and resource limitations, we were unable in any event to model mercury dispersion, deposition, 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 these mercury emissions.

6.5.4 Nitrogen Enrichment

Aquatic Enrichment

One of the main adverse ecological effects resulting from N deposition, particularly in the Mid-Atlantic region of the United States, is the effect associated with nutrient enrichment in estuarine waters. A recent assessment of 141 estuaries nationwide by the National Oceanic and Atmospheric Administration (NOAA) concluded that 19 estuaries (13%) suffered from moderately high or high levels of eutrophication due to excessive inputs of both N and phosphorus, and a majority of these estuaries are located in the coastal area from North Carolina to Massachusetts (NOAA, 2007). For estuaries in the Mid-Atlantic region, the contribution of atmospheric distribution to total N loads is estimated to range between 10% and 58% (Valigura et al., 2001).

Eutrophication in estuaries is associated with a range of adverse ecological effects. The conceptual framework developed by NOAA emphasizes four main types of eutrophication effects—low dissolved oxygen (DO), harmful algal blooms (HABs), loss of submerged aquatic vegetation (SAV), and low water clarity. Low DO disrupts aquatic habitats, causing stress to fish and shellfish, which, in the short-term, can lead to episodic fish kills and, in the long-term, can damage overall growth in fish and shellfish populations. Low DO also degrades the aesthetic qualities of surface water. In addition to often being toxic to fish and shellfish, and leading to fish kills and aesthetic impairments of estuaries, HABs can, in some instances, also be harmful to human health. SAV provides critical habitat for many aquatic species in estuaries and, in some instances, can also protect shorelines by reducing wave strength; therefore, declines

in SAV due to nutrient enrichment are an important source of concern. Low water clarity is the result of accumulations of both algae and sediments in estuarine waters. In addition to contributing to declines in SAV, high levels of turbidity also degrade the aesthetic qualities of the estuarine environment.

Estuaries in the eastern United States are an important source of food production, in particular fish and shellfish production. The estuaries are capable of supporting large stocks of resident commercial species, and they serve as the breeding grounds and interim habitat for several migratory species. To provide an indication of the magnitude of provisioning services associated with coastal fisheries, from 2005 to 2007, the average value of total catch was $1.5 billion per year. It is not known, however, what percentage of this value is directly attributable to or dependent upon the estuaries in these states.

In addition to affecting provisioning services through commercial fish harvests, eutrophication in estuaries may also affect the demand for seafood. For example, a well-publicized toxic pfiesteria bloom in the Maryland Eastern Shore in 1997, which involved thousands of dead and lesioned fish, led to an estimated $56 million (in 2007 dollars) in lost seafood sales for 360 seafood firms in Maryland in the months following the outbreak (Lipton, 1999).

Estuaries in the United States also provide an important and substantial variety of cultural ecosystem services, including water-based recreational and aesthetic services. The water quality in the estuary directly affects the quality of these experiences. For example, there were 26 million days of saltwater fishing coastal states from North Carolina to Massachusetts in 2006 (FWA and Census, 2007). Assuming an average consumer surplus value for a fishing day at $36 (in 2007 dollars) in the Northeast and $87 in the Southeast (Kaval and Loomis, 2003), the aggregate value was approximately $1.3 billion (in 2007 dollars).[1] In addition, almost 6 million adults participated in motorboating in coastal states from North Carolina to Massachusetts, for a total of nearly 63 million days annually during 1999–2000 (Leeworthy and Wiley, 2001). Using a national daily value estimate of $32 (in 2007 dollars) for motorboating (Kaval and Loomis (2003), the aggregate value of these coastal motorboating outings was $2 billion per year.[2] Almost 7 million participated in birdwatching for 175 million days per year, and more than 3 million participated in visits to non-beach coastal waterside areas.

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[1] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

[2] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

Estuaries and marshes have the potential to support a wide range of regulating services, including climate, biological, and water regulation; pollution detoxification; erosion prevention; and protection against natural hazards from declines in SAV (MEA, 2005). SAV can help reduce wave energy levels and thus protect shorelines against excessive erosion, which increases the risks of episodic flooding and associated damages to near-shore properties or public infrastructure or even contribute to shoreline retreat.

Terrestrial Enrichment

Terrestrial enrichment occurs when terrestrial ecosystems receive N loadings in excess of natural background levels, either through atmospheric deposition or direct application. Evidence presented in the Integrated Science Assessment (U.S. EPA, 2008f) supports a causal relationship between atmospheric N deposition and biogeochemical cycling and fluxes of N and carbon in terrestrial systems. Furthermore, evidence summarized in the report supports a causal link between atmospheric N deposition and changes in the types and number of species and biodiversity in terrestrial systems. Nitrogen enrichment occurs over a long time period; as a result, it may take as much as 50 years or more to see changes in ecosystem conditions and indicators. This long time scale also affects the timing of the ecosystem service changes.

One of the main provisioning services potentially affected by N deposition is grazing opportunities offered by grasslands for livestock production in the Central U.S. Although N deposition on these grasslands can offer supplementary nutritive value and promote overall grass production, there are concerns that fertilization may favor invasive grasses and shift the species composition away from native grasses. This process may ultimately reduce the productivity of grasslands for livestock production. Losses due to invasive grasses can be significant; for example, based on a bioeconomic model of cattle grazing in the upper Great Plains, Leitch, Leistritz, and Bangsund (1996) and Leistritz, Bangsund, and Hodur (2004) estimated $130 million in losses due to a leafy spurge infestation in the Dakotas, Montana, and Wyoming.[1] However, the contribution of N deposition to these losses is still uncertain.

6.5.5 Benefits of Reducing Ozone Effects on Vegetation and Ecosystems

Ozone causes discernible injury to a wide array of vegetation (U.S. EPA, 2006a; Fox and Mickler, 1996). In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts (U.S. EPA, 2006a). Studies

[1] These estimates reflect the total value of the service, not the marginal change in the value of the service as a result of the emission reductions achieved by this rule.

have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function (De Steiguer et al., 1990; Pye, 1988).

When ozone is present in the air, it can enter the leaves of plants, where it can cause significant cellular damage. Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through the stomata in leaves in a process called "uptake" (Winner and Atkinson, 1986). Once sufficient levels of ozone (a highly reactive substance), or its reaction products, reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns (U.S. EPA, 2006a; Tingey and Taylor, 1982). With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance, leading to reduced growth and/or reproduction. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, or more susceptible to disease, pest infestation, harsh weather (e.g., drought, frost) and other environmental stresses, which can all produce a loss in plant vigor in ozone-sensitive species that over time may lead to premature plant death. Furthermore, there is evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont (U.S. EPA, 2006a).

This ozone damage may or may not be accompanied by visible injury on leaves, and likewise, visible foliar injury may or may not be a symptom of the other types of plant damage described above. Foliar injury is usually the first visible sign of injury to plants from ozone exposure and indicates impaired physiological processes in the leaves (Grulke, 2003). When visible injury is present, it is commonly manifested as chlorotic or necrotic spots, and/or increased leaf senescence (accelerated leaf aging). Because ozone damage can consist of visible injury to leaves, it can also reduce the aesthetic value of ornamental vegetation and trees in urban landscapes, and negatively affects scenic vistas in protected natural areas.

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual

plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of ozone uptake through closure of stomata) (U.S. EPA, 2006a; Winner, 1994). After injuries have occurred, plants may be capable of repairing the damage to a limited extent (U.S. EPA, 2006a). Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants.

Because plants are at the base of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors (U.S. EPA, 2006a). In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems (U.S. EPA, 2006a, McBride et al., 1985; Miller et al., 1982). It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States (U.S. EPA, 2006a).

Ozone Effects on Forests

Air pollution can affect the environment and affect ecological systems, leading to changes in the ecological community and influencing the diversity, health, and vigor of individual species (U.S. EPA, 2006a). Ozone has been shown in numerous studies to have a strong effect on the health of many plants, including a variety of commercial and ecologically important forest tree species throughout the United States (U.S. EPA, 2007b).

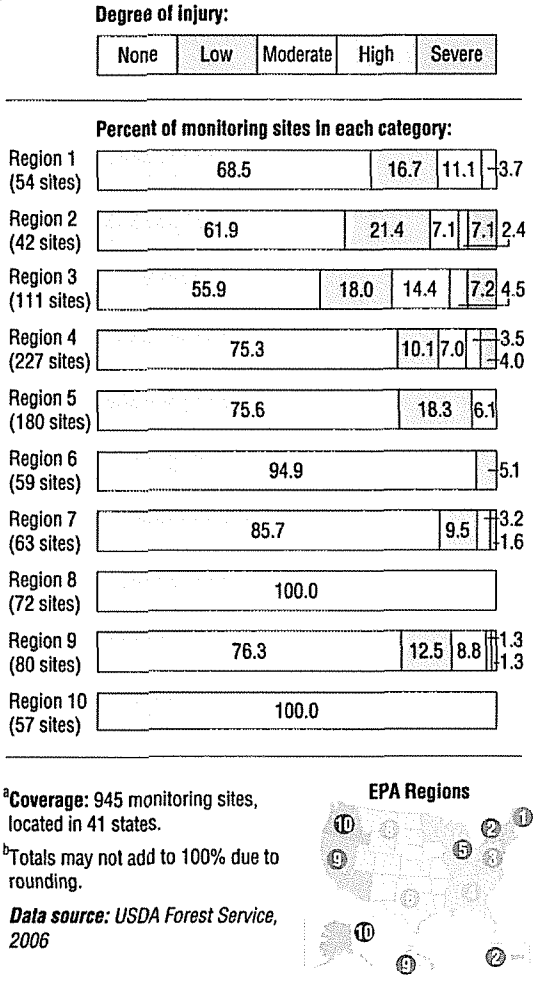
In the U.S., this data comes from the U.S. Department of Agriculture (USDA) Forest Service Forest Inventory and Analysis (FIA) program. As part of its Phase 3 program, formerly known as Forest Health Monitoring, FIA examines ozone injury to ozone-sensitive plant species at ground monitoring sites in forestland across the country (excluding woodlots and urban trees). FIA looks for damage on the foliage of ozone-sensitive forest plant species at each site that meets certain minimum criteria. Because ozone injury is cumulative over the course of the

growing season, examinations are conducted in July and August, when ozone injury is typically highest.

Monitoring of ozone injury to plants by the USDA Forest Service has expanded over the last 10 years from monitoring sites in 10 states in 1994 to nearly 1,000 monitoring sites in 41 states in 2002. The data underlying the indictor in Figure 6-11 are based on averages of all observations collected in 2002, the latest year for which data are publicly available at the time the study was conducted, and are broken down by U.S. EPA Regions. Ozone damage to forest plants is classified using a subjective five-category biosite index based on expert opinion, but designed to be equivalent from site to site. Ranges of biosite values translate to no injury, low or moderate foliar injury (visible foliar injury to highly sensitive or moderately sensitive plants, respectively), and high or severe foliar injury, which would be expected to result in tree-level or ecosystem-level responses, respectively (U.S. EPA, 2006a; Coulston, 2004). The highest percentages of observed high and severe foliar injury, which are most likely to be associated with tree or ecosystem-level responses, are primarily found in the Mid-Atlantic and Southeast regions.

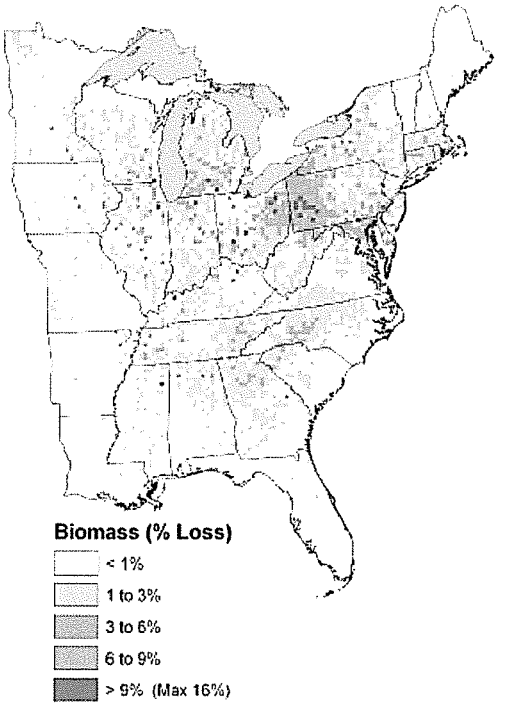
Assessing the impact of ground-level ozone on forests in the eastern United States involves understanding the risks to sensitive tree species from ambient ozone concentrations and accounting for the prevalence of those species within the forest. As a way to quantify the risks to particular plants from ground-level ozone, scientists have developed ozone-exposure/tree-response functions by exposing tree seedlings to different ozone levels and measuring reductions in growth as "biomass loss." Typically, seedlings are used because they are easy to manipulate and measure their growth loss from ozone pollution. The mechanisms of susceptibility to ozone within the leaves of seedlings and mature trees are identical, and the decreases predicted using the seedlings should be related to the decrease in overall plant fitness for mature trees, but the magnitude of the effect may be higher or lower depending on the tree species (Chappelka and Samuelson, 1998). In areas where certain ozone-sensitive species dominate the forest community, the biomass loss from ozone can be significant. Significant biomass loss can be defined as a more than 2% annual biomass loss, which would cause long term ecological harm as the short-term negative effects on seedlings compound to affect long-term forest health (Heck, 1997).

Figure 6-11. Ozone Injury to Forest Plants in U.S. by EPA Regions, 2002[a, b]



Some of the common tree species in the United States that are sensitive to ozone are black cherry (*Prunus serotina*), tulip-poplar (*Liriodendron tulipifera)*, and eastern white pine (*Pinus strobus)*. Ozone-exposure/tree-response functions have been developed for each of these tree species, as well as for aspen (*Populus tremuliodes*), and ponderosa pine (*Pinus ponderosa*) (U.S. EPA, 2007b). Other common tree species, such as oak (*Quercus* spp.*)* and hickory (*Carya* spp.), are not as sensitive to ozone. Consequently, with knowledge of the distribution of sensitive species and the level of ozone at particular locations, it is possible to estimate a "biomass loss" for each species across their range. As shown in Figure 6-12, current ambient levels of ozone are associated with significant biomass loss across large geographic areas (U.S. EPA, 2009b). However, this information is unavailable this rule.

Figure 6-12. Estimated Black Cherry, Yellow Poplar, Sugar Maple, Eastern White Pine, Virginia Pine, Red Maple, and Quaking Aspen Biomass Loss due to Current Ozone Exposure, 2006-2008 (U.S. EPA, 2009b)



To estimate the biomass loss for forest ecosystems across the eastern United States, the biomass loss for each of the seven tree species was calculated using the three-month, 12-hour W126 exposure metric at each location, along with each tree's individual C-R functions. The W126 exposure metric was calculated using monitored ozone data from CASTNET and AQS sites, and a three-year average was used to mitigate the effect of variations in meteorological and soil moisture conditions. The biomass loss estimate for each species was then multiplied by its prevalence in the forest community using the U.S. Department of Agriculture (USDA) Forest Service IV index of tree abundance calculated from Forest Inventory and Analysis (FIA) measurements (Prasad, 2003). Sources of uncertainty include the ozone-exposure/plant-response functions, the tree abundance index, and other factors (e.g., soil moisture). Although these factors were not considered, they can affect ozone damage (Chappelka, 1998).

Ozone damage to the plants including the trees and understory in a forest can affect the ability of the forest to sustain suitable habitat for associated species particularly threatened and endangered species that have existence value – a nonuse ecosystem service - for the public. Similarly, damage to trees and the loss of biomass can affect the forest's provisioning services in the form of timber for various commercial uses. In addition, ozone can cause discoloration of

leaves and more rapid senescence (early shedding of leaves), which could negatively affect fall-color tourism because the fall foliage would be less available or less attractive. Beyond the aesthetic damage to fall color vistas, forests provide the public with many other recreational and educational services that may be impacted by reduced forest health including hiking, wildlife viewing (including bird watching), camping, picnicking, and hunting. Another potential effect of biomass loss in forests is the subsequent loss of climate regulation service in the form of reduced ability to sequester carbon (Felzer et al., 2005).

Ozone Effects on Crops and Urban Ornamentals

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). Damage to crops from ozone exposures includes yield losses (i.e., in terms of weight, number, or size of the plant part that is harvested), as well as changes in crop quality (i.e., physical appearance, chemical composition, or the ability to withstand storage) (U.S. EPA, 2007b). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the United States" (U.S. EPA, 2006a). In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields, directly affecting the amount and quality of the provisioning service provided by the crops in question, associated with observed ozone levels (Kopp et al., 1985; Adams et al., 1986; Adams et al., 1989). According to the Ozone Staff Paper, there has been no evidence that crops are becoming more tolerant of ozone (U.S. EPA, 2007b). Using the Agriculture Simulation Model (AGSIM) (Taylor, 1994) to calculate the agricultural benefits of reductions in ozone exposure, U.S. EPA estimated that meeting a W126 standard of 21 ppm-hr would produce monetized benefits of approximately $160 million to $300 million (inflated to 2006 dollars) (U.S. EPA, 2007b).[1]

Urban ornamentals are an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels. Because ozone causes visible foliar injury, the aesthetic value of ornamentals (such as petunia, geranium, and poinsettia) in urban landscapes would be reduced (U.S. EPA, 2007b). Sensitive ornamental species would require more frequent replacement and/or increased maintenance (fertilizer or pesticide application) to maintain the desired appearance because of exposure to ambient ozone (U.S. EPA, 2007b). In addition, many businesses rely on healthy-looking vegetation for their

[1] These estimates illustrate the value of vegetation effects from a substantial reduction of ozone concentrations, not the marginal change in ozone concentrations anticipated a result of the emission reductions achieved by this rule.

livelihoods (e.g., horticulturalists, landscapers, Christmas tree growers, farmers of leafy crops, etc.) and a variety of ornamental species have been listed as sensitive to ozone (Abt Associates, 1995). The ornamental landscaping industry is valued at more than $30 billion (inflated to 2006 dollars) annually, by both private property owners/tenants and by governmental units responsible for public areas (Abt Associates, 1995). Therefore, urban ornamentals represent a potentially large unquantified benefit category. This aesthetic damage may affect the enjoyment of urban parks by the public and homeowners' enjoyment of their landscaping and gardening activities. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, we cannot conduct a quantitative analysis to estimate these effects.

6.5.6 Unquantified SO_2 and NO_2-Related Human Health Benefits

Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the Integrated Science Assessment for Sulfur Dioxide concluded that there is a causal relationship between respiratory health effects and short-term exposure to SO_2 (U.S. EPA, 2008). The immediate effect of SO_2 on the respiratory system in humans is bronchoconstriction. 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. 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. We did not quantify these benefits due to time constraints.

Epidemiological researchers have associated NO_2 exposure with adverse health effects in numerous toxicological, clinical and epidemiological studies, as described in the Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Final Report) (U.S. EPA, 2008c). The NO_2 ISA provides a comprehensive review of the current evidence of health and environmental effects of NO_2. The NO_2 ISA concluded that the evidence "is sufficient to infer a

likely causal relationship between short-term NO_2 exposure and adverse effects on the respiratory system" (ISA, section 5.3.2.1). These epidemiologic and experimental studies encompass a number of endpoints including [Emergency Department (ED)] visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, airway inflammation, and lung function. Effect estimates from epidemiologic studies conducted in the United States and Canada generally indicate a 2-20% increase in risks for ED visits and hospital admissions and higher risks for respiratory symptoms (ISA, section 5.4). The NO_2 ISA concluded that the relationship between short-term NO_2 exposure and premature mortality was "suggestive but not sufficient to infer a causal relationship" because it is difficult to attribute the mortality risk effects to NO_2 alone. Although the NO_2 ISA stated that studies consistently reported a relationship between NO_2 exposure and mortality, the effect was generally smaller than that for other pollutants such as PM. We did not quantify these benefits due to time constraints.

6.6 Social Cost of Carbon and Greenhouse Gas Benefits

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.[1]

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[2] in 2010, in

[1] 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

[2] 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

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.

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 2016, in 2007 dollars are provided in Table 6-15.

Table 6-15. Social Cost of Carbon (SCC) Estimates (per tonne of CO_2) for 2016 (in 2007$)[a]

Discount Rate and Statistic		*SCC estimate*
5%	Average	$5.77
3%	Average	$23.7
2.5%	Average	$37.9
3%	95%ile	$72.3

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

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 2009) 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

"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).

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 from the February 2010 date of promulgation of the Light Duty Vehicle rule referenced above 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 reductions in CO_2 emissions for the range of policy scenarios, we estimate the dollar value of the climate related benefits captured by the models for each analysis year. For internal consistency, the annual benefits 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%.[1] These estimates are provided in Table 6-16.

Table 6-16. Monetized Benefits of CO_2 Emissions Reductions in 2016 (in millions of 2007$)[a]

Discount Rate and Statistic		*SCC estimate*
5%	Average	$140
3%	Average	$570
2.5%	Average	$910
3%	95%ile	$1,700

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

[1] 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.

6.7 Benefits Results

Applying the impact and valuation functions described previously in this chapter to the estimated changes in ozone and PM yields estimates of the changes in physical damages (e.g., premature mortalities, cases, admissions, and change in light extinction) and the associated monetary values for those changes. Estimates of health impacts among Eastern and Western states, are presented in Table 6-15. Monetized values for both health and welfare endpoints within the trading region are presented in Table 6-16, along with total aggregate monetized benefits. All of the monetary benefits are in constant-year 2007 dollars.

Not all known PM- and ozone-related health and welfare effects could be quantified or monetized. The monetized value of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health and welfare benefits plus B, the sum of the nonmonetized health and welfare benefits; this B represents both uncertainty and a bias in this analysis, as it reflects those benefits categories that we are unable quantify in this analysis.

Table 6-17. Estimated Reduction in Incidence of Adverse Health Effects of the Proposed Toxics Rule (95% confidence intervals)[a]

Health Effect	*Eastern U.S.*	*Western U.S.*	*Total*
PM-Related endpoints			
Premature death			
Pope et al. (2002) (age >30)	6,700 (1,900—12,000)	120 (33—200)	6,800 (1,900—12,000)
Laden et al. (2006) (age >25)	17,000 (7,900—26,000)	300 (140—470)	17,000 (8,100—27,000)
Infant (< 1 year)	29 (-32—90)	1 (-1—2)	30 (-33—92)
Chronic bronchitis	4,400 (150—8,600)	97 (3—190)	4,500 (150—8,800)
Non-fatal heart attacks (age > 18)	11,000 (2,700—18,000)	190 (48—330)	11,000 (2,700—19,000)
Hospital admissions—respiratory (all ages)	1,600 (650—2,600)	24 (10—39)	1,700 (660—2,600)
Hospital admissions—cardiovascular (age > 18)	3,500 (2,500—4,200)	50 (35—61)	3,600 (2,500—4,200)
Emergency room visits for asthma (age < 18)	6,900 (3,500—10,000)	52 (27—78)	6,900 (3,600—10,000)
Acute bronchitis (age 8-12)	10,000 (-2,300—23,000)	250 (-57—560)	11,000 (-2,400—23,000)
Lower respiratory symptoms (age 7-14)	120,000 (47,000—200,000)	3,000 (1,100—4,800)	130,000 (48,000—200,000)
Upper respiratory symptoms (asthmatics age 9-18)	93,000 (17,000—170,000)	2,300 (420—4,100)	95,000 (18,000—170,000)
Asthma exacerbation (asthmatics 6-18)	110,000 (4,000—380,000)	2,700 (96—9,300)	120,000 (4,100—390,000)
Lost work days (ages 18-65)	830,000 (710,000—960,000)	20,000 (17,000—22,000)	850,000 (720,000—980,000)
Minor restricted-activity days (ages 18-65)	5,000,000 (4,000,000—5,900,000)	110,000 (94,000—140,000)	5,100,000 (4,100,000—6,000,000)

[a] Estimates rounded to two significant figures; column values will not sum to total value.
[b] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

Table 6-18. Estimated Economic Value of Health and Welfare Benefits (95% confidence intervals, billions of 2007$)[a]

Health Effect		*Eastern U.S.*[b]	*Western U.S.*	*Total*
Adult premature death (Pope et al. 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$53 ($4.2—$160)	$0.9 ($0.1—$2.8)	$54 ($4.3—$160)
7% discount rate	$PM_{2.5}$	$48 ($3.8—$140)	$0.8 ($0. 1—$2.5)	$48 ($3.8—$150)
Adult premature death (Laden et al. 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$140 ($12—$390)	$2.4 ($0.2—$6.9)	$140 ($12—$400)
7% discount rate	$PM_{2.5}$	$120 ($11—$350)	$2.2 ($0.2—$6.3)	$120 ($11—$360)
Infant premature death	$PM_{2.5}$	$0.3 ($-0.3—$1.2)	<$0.01	$0.3 ($-0.3—$1.2)
Chronic Bronchitis	$PM_{2.5}$	$2.1 ($0.1—$9.6)	$0.05 (<$0.01—$0.2)	$2.1 ($0.1—$9.8)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$1.2 ($0.2—$2.9)	$1.2 ($0.2—$2.9)	$1.2 ($0.2—$2.8)
7% discount rate	$PM_{2.5}$	$1.1 ($0.2—$2.8)	$1.2 ($0.2—$2.9)	$1.1 ($0.2—$2.8)
Hospital admissions—respiratory	$PM_{2.5}$	<$0.01	<$0.01	$0.02 ($0. 01—$0.03)
Hospital admissions—cardiovascular	$PM_{2.5}$	<$0.01	<$0.01	$0.1 ($0.05—$0.14)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1—$0.1)	<$0.01	$0.1 ($0.1—$0.1)
Minor restricted-activity days	$PM_{2.5}$	$0.3 ($0.2—$0.5)	<$0.01	$0.3 ($0.2—$0.5)
CO_2-related benefits (3% discount rate)	CO_2			$0.57
Monetized total Benefits (Pope et al. 2002 $PM_{2.5}$ mortality estimate)				
3% discount rate		$57 ($4.6—$170)	$1 ($0.1—$3.1)	$59 ($4.6—$180)
7% discount rate		$52 ($4.1—$160)	$0.9 ($0.1—$2.8)	$53 ($4.2—$160)

(continued)

Table 6-18. Estimated Economic Value of Health and Welfare Benefits (95% confidence intervals, billions of 2007$)[a] (continued)

Health Effect	*Eastern U.S.*[b]	*Western U.S.*	*Total*
Monetized total Benefits (Laden et al. 2006 $PM_{2.5}$ mortality and Levy et al. 2005 ozone mortality estimates)			
3% discount rate	$140 ($12—$410)	$2.5 ($0.2—$7.2)	$140 ($12—$410)
7% discount rate	$130 ($11—$370)	$2.2 ($0.2—$6.6)	$130 ($11—$370)

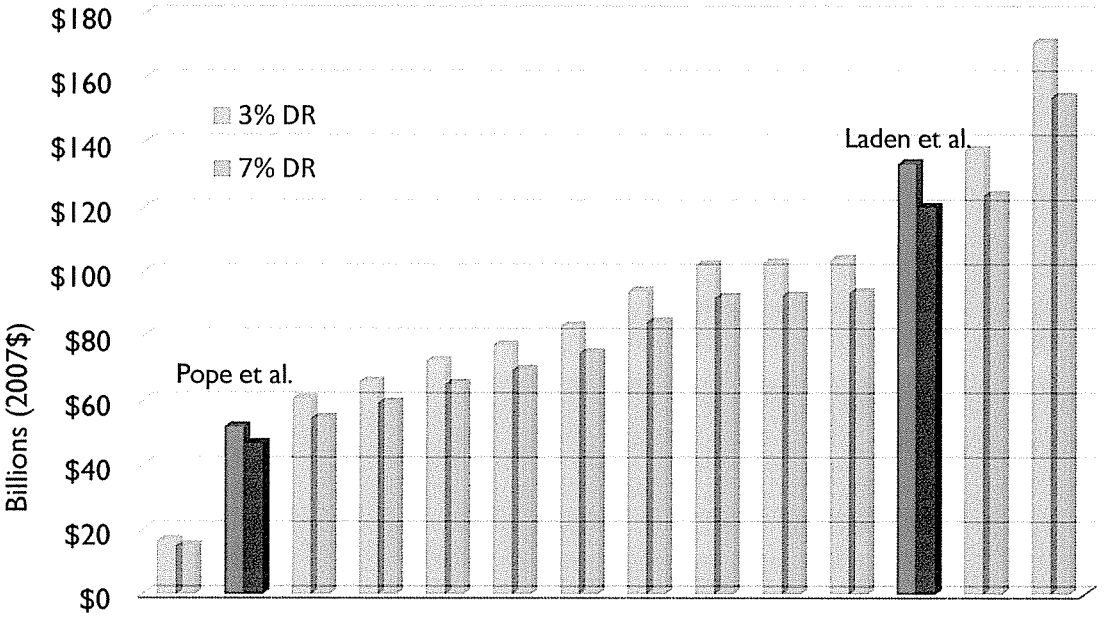
[a] Economic value adjusted to 2007$ using GDP deflator. Estimates rounded to two significant figures. The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts. Confidence intervals reflect random sampling error and not the additional uncertainty associated with benefits scaling described above. The net present value of reduced CO2 emissions are calculated differently than other benefits. The same discount rate used to discount the value of damages from future emissions (SCC at 5, 3, 2.5 percent) is used to calculate net present value of SCC for internal consistency. This table shows monetized CO2 co-benefits at discount rates at 3 and 7 percent that were calculated using the global average SCC estimate at a 3% discount rate because the interagency workgroup on this topic deemed this marginal value to be the central value. In section 6.6 we also report he monetized CO2 co-benefits using discount rates of 5 percent (average), 2.5 percent (average), and 3 percent (95^{th} percentile).

[b] Monetary value of endpoints marked with dashes are < $100,000.

Total monetized co-benefits are dominated by benefits of mortality risk reductions. The primary analysis projects that the proposed Toxics Rule will result in between 6,800 and 17,000 $PM_{2.5}$ -related avoided premature deaths annually in 2016. Our estimate of total monetized co-benefits in 2016 proposed Toxics Rule is between $59 billion and $140 billion using a 3 percent discount rate and between $53 billion and $130 using a 7 percent discount rate. Health benefits account for between 93 and 97 percent of total benefits depending on the $PM_{2.5}$ estimates used, in part because we are unable to quantify most of the non-health benefits. The next largest benefit is for reductions in chronic illness (CB and nonfatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, visibility, MRADs and work loss days account for the majority of the remaining benefits. The remaining categories each account for a small percentage of total benefit; however, they represent a large number of avoided incidences affecting many individuals. A comparison of the incidence table to the monetary benefits table reveals that there is not always a close correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are over 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the

more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of WTP. As such, the true value of these effects may be higher than that reported in Table 6-18. Figure 6-13 summarizes an array of $PM_{2.5}$-related monetized benefits estimates based on alternative epidemiology and expert-derived PM-mortality estimate.

Figure 6-13. Economic Value of Estimated $PM_{2.5}$- Related Premature Mortalities Avoided According to Epidemiology or Expert-Derived PM Mortality Risk Estimate[a]



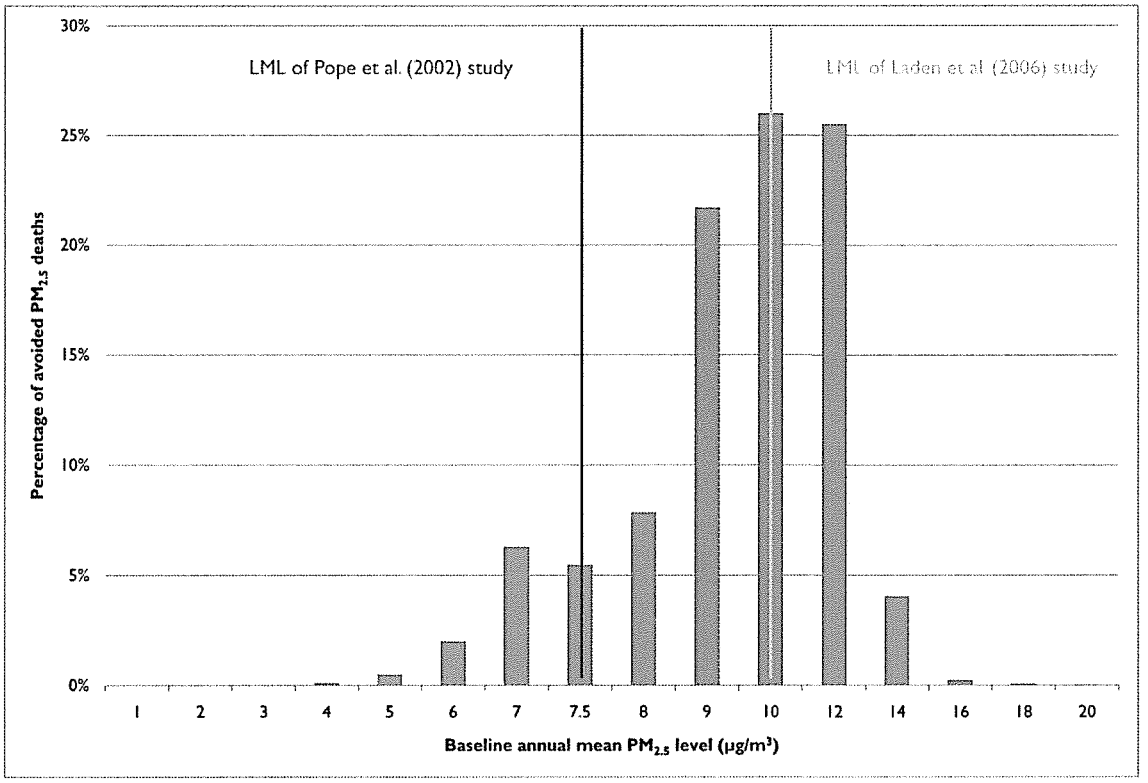
Based on our review of the current body of scientific literature, EPA estimated PM-related mortality without applying an assumed concentration threshold. EPA's Integrated Science Assessment for Particulate Matter (U.S. EPA, 2009b), which was reviewed by EPA's Clean Air Scientific Advisory Committee (U.S. 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 also recognizing potential uncertainty about the exact shape of the concentration-response function. Consistent with this finding, we incorporated a "Lowest Measured Level" (LML) assessment, which is a method EPA has employed in several recent RIA's including the 2010 proposed Transport Rule (U.S. EPA, 2010c).

This approach summarizes the distribution of avoided PM mortality impacts according to the baseline (i.e. pre-Toxics Rule) $PM_{2.5}$ levels experienced by the population receiving the $PM_{2.5}$ mortality benefit (Figures 6-14 and 6-15). We identify on this figure the lowest air quality levels measured in each of the two primary epidemiological studies EPA uses to quantify 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 the LML analysis 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. While this figure describes the relationship between baseline $PM_{2.5}$ exposure and mortality for the air quality modeled policy case, we expect the distribution of mortality impacts to be fairly similar between the two cases.

Some proportion of the avoided PM-related impacts we estimate in this analysis occur among populations exposed at or above the LML of the Laden et al. (2006) study, while a majority of the impacts occur at or above the LML of the Pope et al. (2002) study (Figure 5-17), increasing our confidence in the PM mortality analysis. Approximately 30% of the avoided impacts occur at or above an annual mean $PM_{2.5}$ level of 10 $\mu g/m^3$ (the LML of the Laden et al. 2006 study); about 86% occur at or above an annual mean $PM_{2.5}$ level of 7.5 $\mu g/m^3$ (the LML of the Pope et al. 2002 study). 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.

While the LML of each study is important to consider when characterizing and interpreting the overall level PM-related benefits, as discussed earlier in this chapter, EPA believes that both cohort-based mortality estimates are suitable for use in air pollution health impact analyses. When estimating PM mortality impacts using risk coefficients drawn from the Laden et al. analysis of the Harvard Six Cities and the Pope et al. analysis of the American Cancer Society cohorts there are innumerable other attributes that may affect the size of the reported risk estimates—including differences in population demographics, the size of the cohort, activity patterns and particle composition among others. The LML assessment presented here provides a limited representation of one key difference between the two studies.

Figure 6-14. Percentage of Total PM-Related Mortalities Avoided by Baseline Air Quality Level

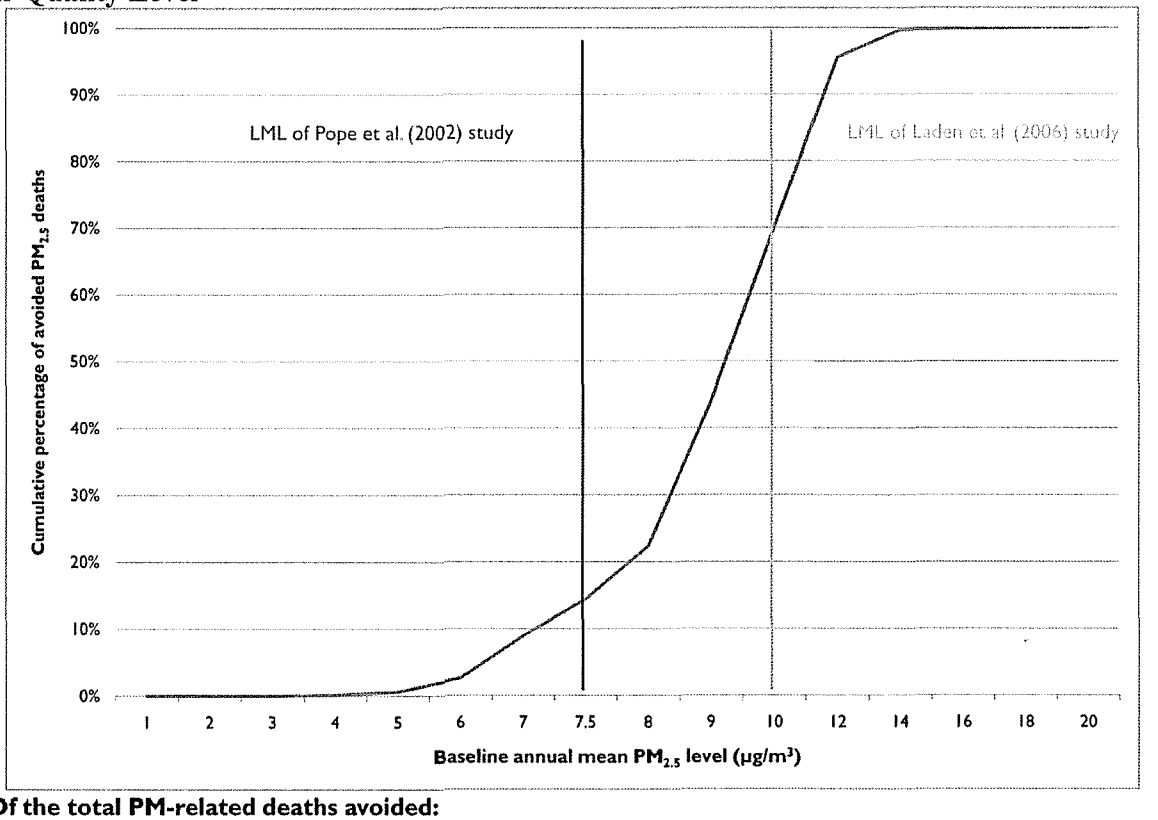


Of the total PM-related deaths avoided:

86% occur among population exposed to PM levels at or above the LML of the **Pope et al.** study.

30% occur among population exposed to PM levels at or above the LML of the Laden et al. study.

Figure 6-15. Cumulative Percentage of Total PM-Related Mortalities Avoided by Baseline Air Quality Level



Of the total PM-related deaths avoided:

86% occur among population exposed to PM levels at or above the LML of the **Pope et al.** study.
30% occur among population exposed to PM levels at or above the LML of the Laden et al. study.

6.8 Discussion

This analysis demonstrates the significant health and welfare co-benefits of the Toxics Rule. We estimate that by 2016 the rule will have reduced the number of $PM_{2.5}$ and ozone-related premature mortalities by between 6,800 and 17,000, produce substantial non-mortality benefits and significantly improve visibility in Class 1 areas. This rule promises to yield significant welfare impacts as well, though the quantification of those endpoints in this RIA is incomplete. These significant health and welfare benefits suggest the important role that pollution from the EGU sector plays in the public health impacts of air pollution.

Inherent in any complex RIA such as this one are multiple sources of uncertainty. Some of these we characterized through our quantification of statistical error in the concentration response relationships and our use of the expert elicitation-derived PM mortality functions. Others, including the projection of atmospheric conditions and source-level emissions, the projection of baseline morbidity rates, incomes and technological development are unquantified.

When evaluated within the context of these uncertainties, the health impact and monetized benefits estimates in this RIA can provide useful information regarding the public health impacts attributable to EGUs.

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APPENDIX C.
CO-BENEFITS APPENDIX

In section 1 of this appendix to the co-benefits chapter we report the results of the air quality modeled scenario before it was adjusted to account for the updated emissions scenario. In section 2 we characterize the distribution of avoided $PM_{2.5}$-related premature deaths according to the baseline level of air pollution-related risk of the population.

C.1 $PM_{2.5}$-Related Health Impacts and Monetized Benefits of the Air Quality Modeled Scenario

As noted in Chapter 6 of the RIA, the air quality modeling performed for the RIA does not reflect the changes in emissions of $PM_{2.5}$ precursors associated with the revised policy case. For this reason, we updated the benefits analysis to account for the updated policy case using methods described in the benefits chapter. In this appendix, we detail the results of the benefits analysis associated with the modeled scenario. Chapter 4 of the RIA describes in detail the air quality modeling results. As described in the benefits chapter, the chief difference between the modeled and revised cases relate to the level and distribution of SO_2 and NOx emission reductions:

> While the modeled and revised policy case achieve roughly similar levels of SO_2 reductions (2.35M versus 2.06M, respectively), the modeled case concentrates SO_2 reductions primarily among a few Midwestern and southeastern states, while the revised case distributes SO_2 reductions more evenly across both the Midwest, southeast and west. Likewise, the modeled case generates the largest NOx reductions in the Midwest, while the revised case shifts these reductions to western states including Montana, Colorado and Utah.

Tables C-1 and C-2 summarize the $PM_{2.5}$-related health impacts and monetized benefits by each health endpoint. Figure C-1 illustrates the distribution of avoided PM-related deaths by county across the U.S.

Table C-1. Estimated Reduction in Incidence of Adverse Health Effects of the Proposed Toxics Rule (95% confidence intervals)[A]

Health Effect	*Eastern U.S.*	*Western U.S.*	*Total*
PM-Related endpoints			
Premature Mortality			
Pope et al. (2002) (age >30)	7,700 (2,100—13,000)	85 (24—150)	7,800 (2,200—13,000)
Laden et al. (2006) (age >25)	20,000 (9,000—30,000)	220 (100—340)	20,000 (9,100—31,000)
Infant (< 1 year)	33 (-37—100)	0.4 (-1—1)	34 (-38—100)
Chronic Bronchitis	5,000 (170—9,800)	67 (2—130)	5,100 (170—9,900)
Non-fatal heart attacks (age > 18)	12,000 (3,100—21,000)	130 (34—240)	12,000 (3,100—21,000)
Hospital admissions—respiratory (all ages)	1,900 (740—2,900)	17 (7—27)	1,900 (750—3,000)
Hospital admissions—cardiovascular (age > 18)	4,100 (2,700—4,800)	36 (25—43)	4,100 (2,700—4,900)
Emergency room visits for asthma (age < 18)	7,900 (4,000—12,000)	35 (18—51)	7,900 (4,000—12,000)
Acute bronchitis (age 8-12)	12,000 (-2,700—26,000)	170 (-38—370)	12,000 (-2,800—26,000)
Lower respiratory symptoms (age 7-14)	140,000 (54,000—220,000)	2,000 (760—3,200)	140,000 (55,000—230,000)
Upper respiratory symptoms (asthmatics age 9-18)	110,000 (20,000—190,000)	1,500 (280—2,700)	110,000 (20,000—190,000)
Asthma exacerbation (asthmatics 6-18)	130,000 (4,500—440,000)	1,800 (64—6,200)	130,000 (4,600—440,000)
Lost work days (ages 18-65)	950,000 (810,000—1,100,000)	13,000 (11,000—15,000)	970,000 (820,000—1,100,000)
Minor restricted-activity days (ages 18-65)	5,700,000 (4,600,000—6,700,000)	79,000 (64,000—94,000)	5,700,000 (4,700,000—6,800,000)

[A] Estimates rounded to two significant figures; column values will not sum to total value.
[B] The negative estimates for certain endpoints are the result of the weak statistical power of the study used to calculate these health impacts and do not suggest that increases in air pollution exposure result in decreased health impacts.

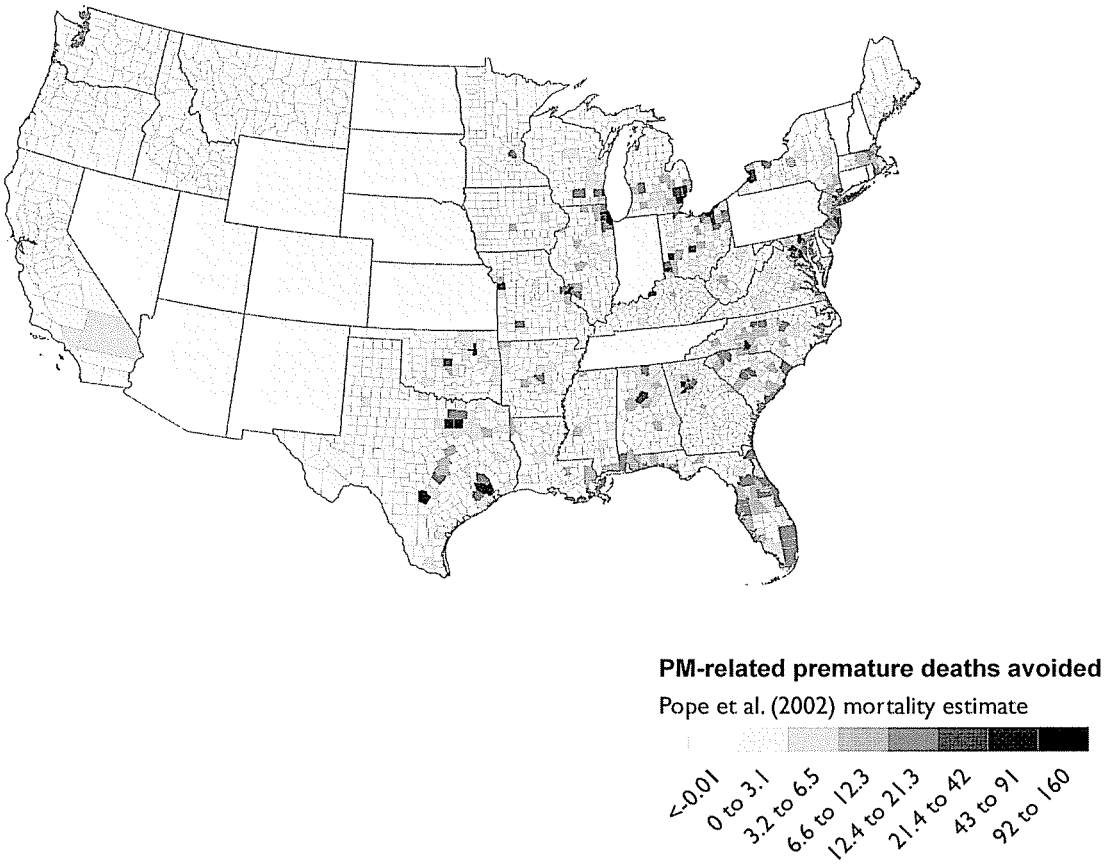
Table C-2. Estimated Economic Value of Health and Welfare Benefits (95% confidence intervals, billions of 2006$)[a]

Health Effect		*Eastern U.S.*	*Western U.S.*	*Total*
Premature Mortality (Pope et al. 2002 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$60 ($4.8—$180)	$0.7 ($0.1—$2)	$61 ($4.8—$180)
7% discount rate	$PM_{2.5}$	$54 ($4.3—$160)	$0.6 ($0.01—$1.8)	$55 ($4.3—$170)
Premature mortality (Laden et al. 2006 PM mortality estimate)				
3% discount rate	$PM_{2.5}$	$150 ($13—$440)	$1.7 ($0.2—$4.9)	$160 ($14—$450)
7% discount rate	$PM_{2.5}$	$140 ($12—$400)	$1.5 ($0.1—$4.5)	$140 ($12—$400)
Infant mortality	$PM_{2.5}$	$0.3 ($-0.3—$1.3)	<$0.01	$0.3 ($-0.3—$1.3)
Chronic Bronchitis	$PM_{2.5}$	$2.4 ($0.1—$11)	$0.03 ($0.01—$0.15)	$2.4 ($0.1—$11)
Non-fatal heart attacks				
3% discount rate	$PM_{2.5}$	$1.3 ($0.2—$3.3)	$0.02 (<$0.01—$0.04)	$1.3 ($0.2—$3.3)
7% discount rate	$PM_{2.5}$	$1.3 ($0.2—$3.2)	$0.014 (<$0.01—$0.04)	$1.3 ($0.2—$0.5)
Hospital admissions—respiratory	$PM_{2.5}$	$0.03 ($0.01—$0.04)	<$0.01	$0.03 ($0.01—$0.04)
Hospital admissions—cardiovascular	$PM_{2.5}$	$0.1 ($0.1—$0.2)	<$0.01	$0.1 ($0.1—$0.2)
Emergency room visits for asthma	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Acute bronchitis	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lower respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Upper respiratory symptoms	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Asthma exacerbation	$PM_{2.5}$	<$0.01	<$0.01	<$0.01
Lost work days	$PM_{2.5}$	$0.1 ($0.1—$0.1)	<$0.01	$0.1 ($0.1—$0.1)
Minor restricted-activity days	$PM_{2.5}$	$0.4 ($0.2—$0.5)	<$0.01	$0.4 ($0.2—$0.5)
Recreational visibility, Class I areas	$PM_{2.5}$	$2.1	$0.1	$2.2
Social cost of carbon (3% discount rate, 2014 value)	CO_2			

Monetized total Benefits (Pope et al. 2002 $PM_{2.5}$ mortality and Bell et al. 2004 ozone mortality estimates)			
3% discount rate	**$64 ($5.2—$200)**	**$0.7 ($0.1—$2.2)**	**$65 ($5.2—$200)**
7% discount rate	**$59 ($4.7—$180)**	**$0.7 ($0.1—$2)**	**$59 ($4.7—$180)**
Monetized total Benefits (Laden et al. 2006 $PM_{2.5}$ mortality and Levy et al. 2005 ozone mortality estimates)			
3% discount rate	**$160 ($14—$460)**	**$1.8 ($0.2—$5.2)**	**$160 ($14—$470)**
7% discount rate	**$140 ($13—$420)**	**$1.6 ($0.1—$4.7)**	**$140 ($13—$420)**

[A] Estimates rounded to two significant figures.

Figure C-1. Estimated Reduction in Excess $PM_{2.5}$-Related Premature Deaths Estimated to Occur in Each County in 2016 as a Result of the Proposed Rule



C.2 Characterizing the distribution of health impacts across the population

This analysis aims to answer two principal questions regarding the distribution of $PM_{2.5}$ co-benefits resulting from the implementation of the proposed Toxics Rule:

1. What is the baseline distribution of $PM_{2.5}$-related mortality risk according to race, income and education?
2. To what extent does the Toxics Rule deliver $PM_{2.5}$ co-benefits among those populations at greatest risk in the baseline?

C.2.1 Methodology

As a first step, we estimate the level of $PM_{2.5}$-related mortality risk in each county in the continental U.S. in 2005, which provides a baseline against which projected changes in $PM_{2.5}$ risk attributable to the Toxics rule may be compared. This portion of the analysis follows an approach described elsewhere (Fann et al. 2011a, Fann et al. 2011b), wherein modeled 2005 $PM_{2.5}$ levels are used to calculate the proportion of total mortality risk attributable to $PM_{2.5}$ in each county. Within each county, we next estimate the distribution of these $PM_{2.5}$ mortality risks for all adult populations as well as risk according to the race, income and educational attainment of the population.

Our approach to calculating $PM_{2.5}$ mortality risk is generally consistent with the primary analysis with the two exceptions: the PM mortality risk coefficients used to quantify impacts and the baseline mortality rates used to calculate mortality impacts. We substitute risk estimates drawn from the Krewski et al. (2009) extended analysis of the ACS cohort. In particular, we applied the all-cause mortality risk estimate random effects Cox model that controls for 44 individual and 7 ecological covariates, using average exposure levels for 1999-2000 over 116 U.S. cities (Krewski et al. 2009) (RR=1.06, 95% confidence intervals 1.04—1.08 per 10$\mu g/m^3$ increase in $PM_{2.5}$). When estimating PM mortality impacts among populations according to level of education, we applied $PM_{2.5}$ mortality risk coefficients modified by educational attainment: less than grade 12 (RR = 1.082, 95% confidence intervals 1.024—1.144 per 10 $\mu g/m^3$ change), grade 12 (RR = 1.072, 95% confidence intervals 1.020—1.127 per 10 $\mu g/m^3$ change), and greater than grade 12 (RR = 1.055, 95% confidence intervals 1.018—1.094 per 10 $\mu g/m^3$ change). The principal reason we applied risk estimates from the Krewski study was to ensure that the risk coefficients used to estimate of all-cause mortality risk and education-modified mortality risk were drawn from a consistent modeling framework.

The other key difference between this sensitivity analysis and the primary analysis relates to the baseline mortality rates. As described in the benefits chapter, we calculate PM-related

mortality risk relative to baseline mortality rates in each county. Traditionally we have applied county-level age and sex stratified rates when calculating these impacts (Abt, 2008). For the calculation of PM impacts by race, we incorporated race-specific (stratified by white/black/asian/Native American) baseline mortality rates.

This approach yields a distribution of county-level risks stratified by each of these population variables (race, income and educational attainment). We next identified the counties at the median and upper 95th percentile of the distribution. The second step of the analysis was to repeat the sequence above by estimating $PM_{2.5}$ mortality risk in 2016 prior to, and after, the implementation of the proposed Toxics Rule.

C.2.2 Results

The level of $PM_{2.5}$ mortality risk among all populations declines significantly between 2005, 2016 prior to, and then after, the implementation of the Toxics rule (Figures C-2—C4). In each figure we outline in yellow those counties at or above the 2005 median risk level. The number of counties at or above this level falls significantly between 2005 and the implementation of the 2016 Toxics rule, suggesting that the combination of this rule as well as others being implemented between 2005 and 2016 are reducing greatly the level of PM mortality risk among adult populations.

We next stratify the PM mortality risk according to race, income and educational attainment. For these analyses we estimated the change in PM mortality risk between 2005 and 2016 among populations living in those counties at the upper 95th percentile of the mortality risk in the baseline; we then compared the change in risk among these populations living in high-risk counties with populations living in all other counties. Figures C-5—C-7 summarize these results.

Figure C-2. Distribution of $PM_{2.5}$ Mortality Risk in 2005

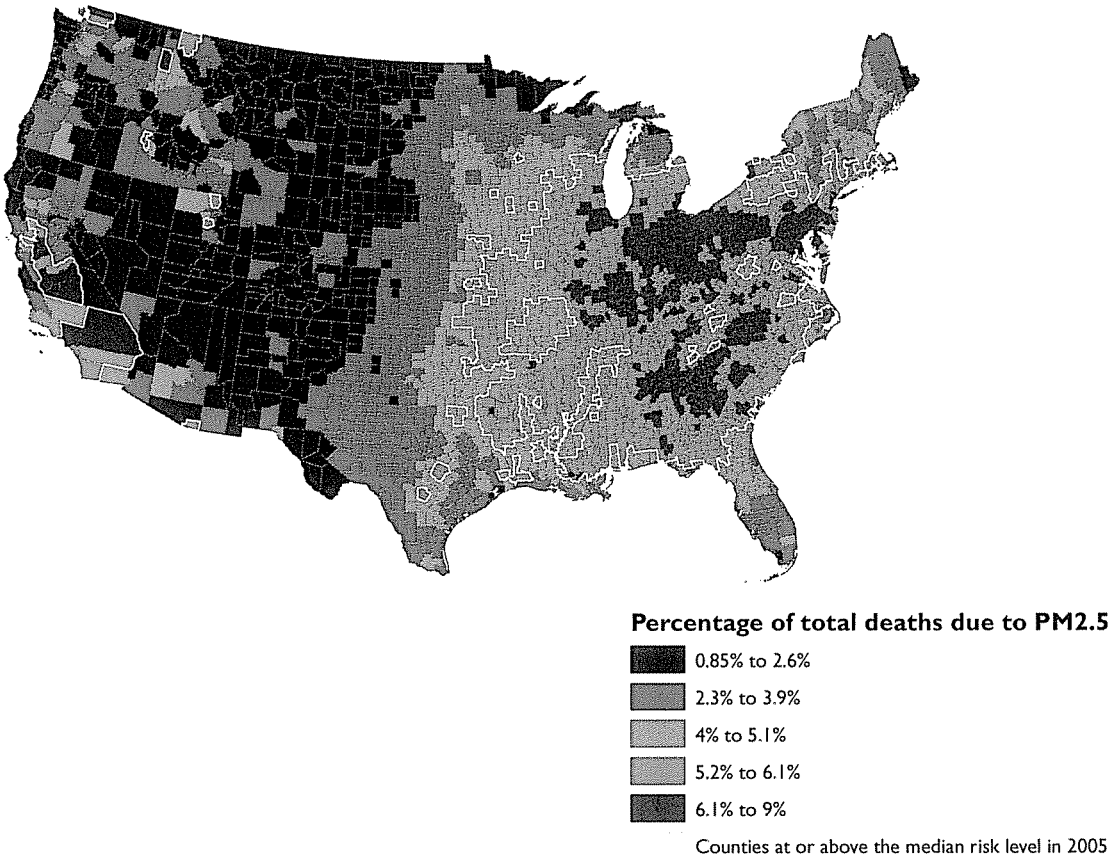


Figure C-3. Distribution of $PM_{2.5}$ Mortality Risk in 2016 (prior to the implementation of the Toxics Rule)

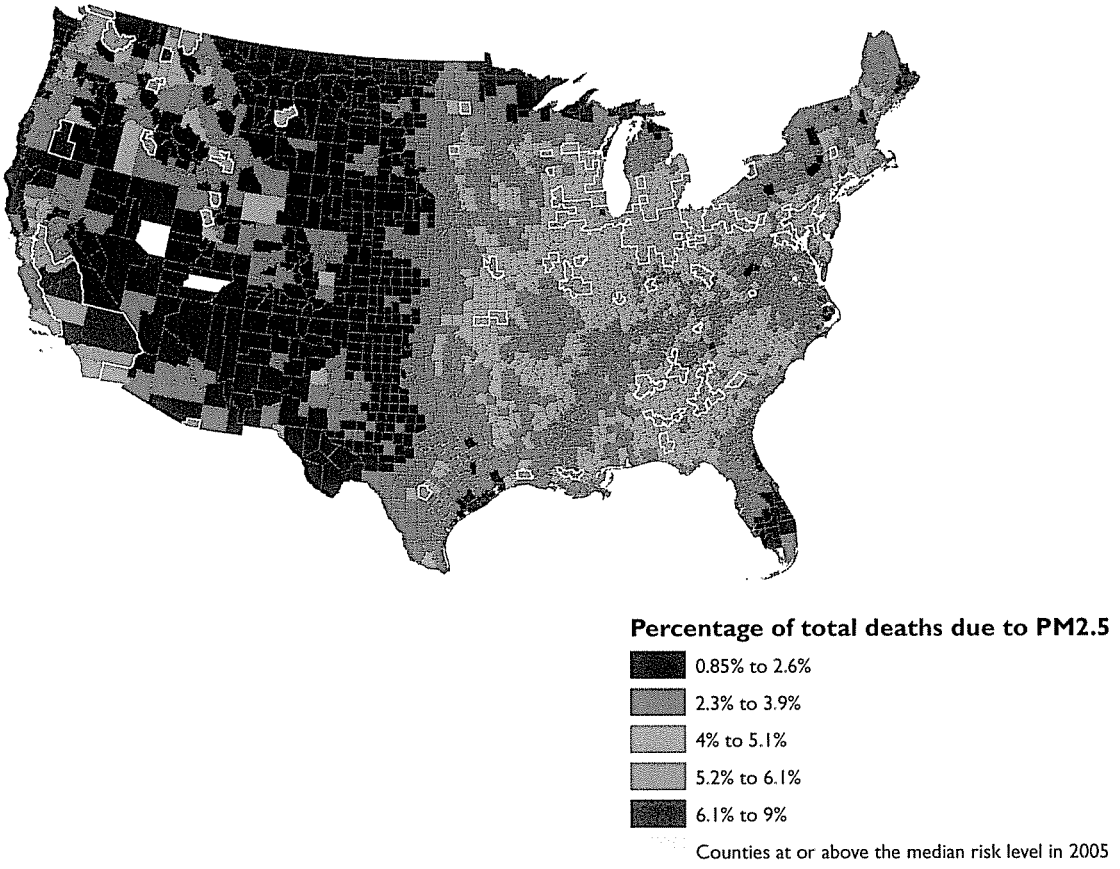


Figure C-4. Distribution of $PM_{2.5}$ Mortality Risk in 2016 (after the implementation of the Toxics Rule)

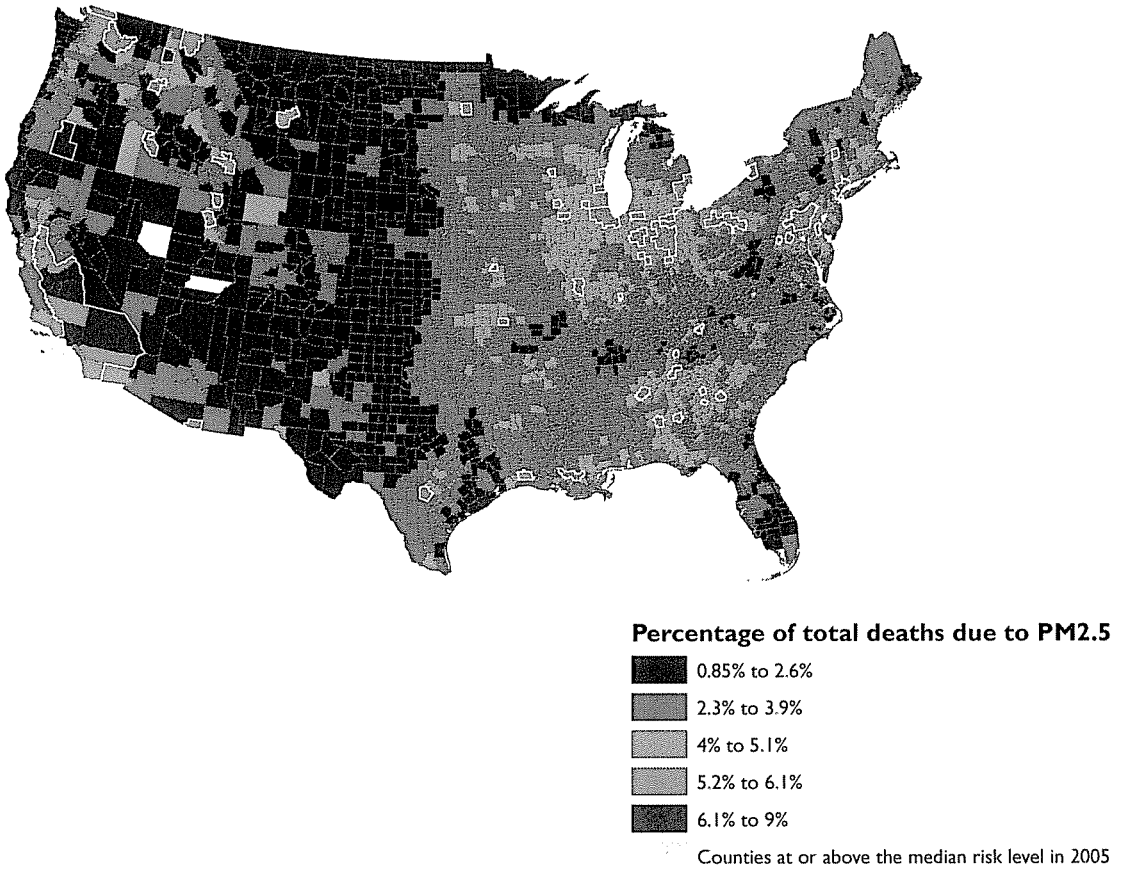


Figure C-5. Change in the Percentage of $PM_{2.5}$-Attributable Deaths by Race between 2005 and the Implementation of the Toxics Rule

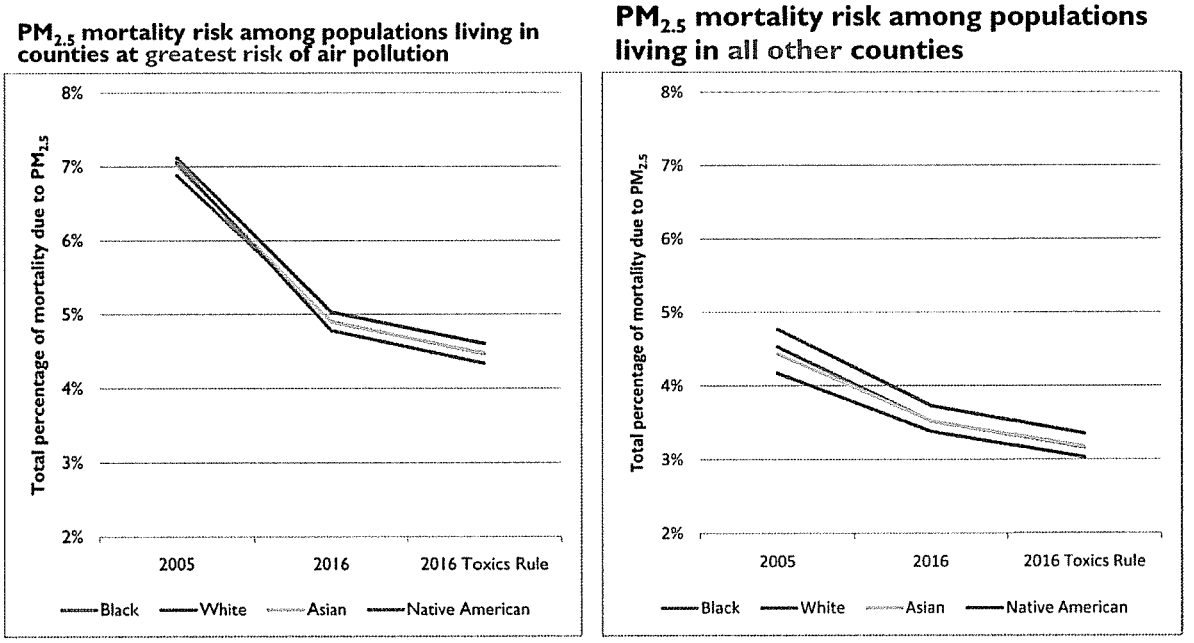


Figure C-6. Change in the Percentage of $PM_{2.5}$-Attributable Deaths among Populations by Poverty Level between 2005 and the Implementation of the Toxics Rule

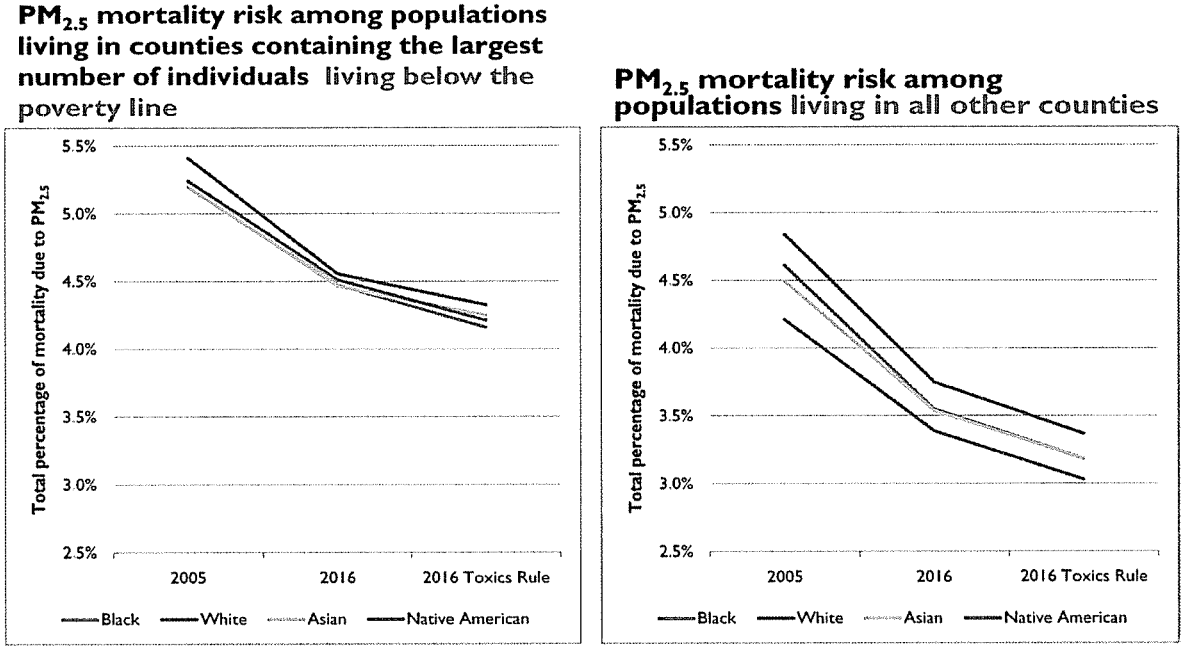
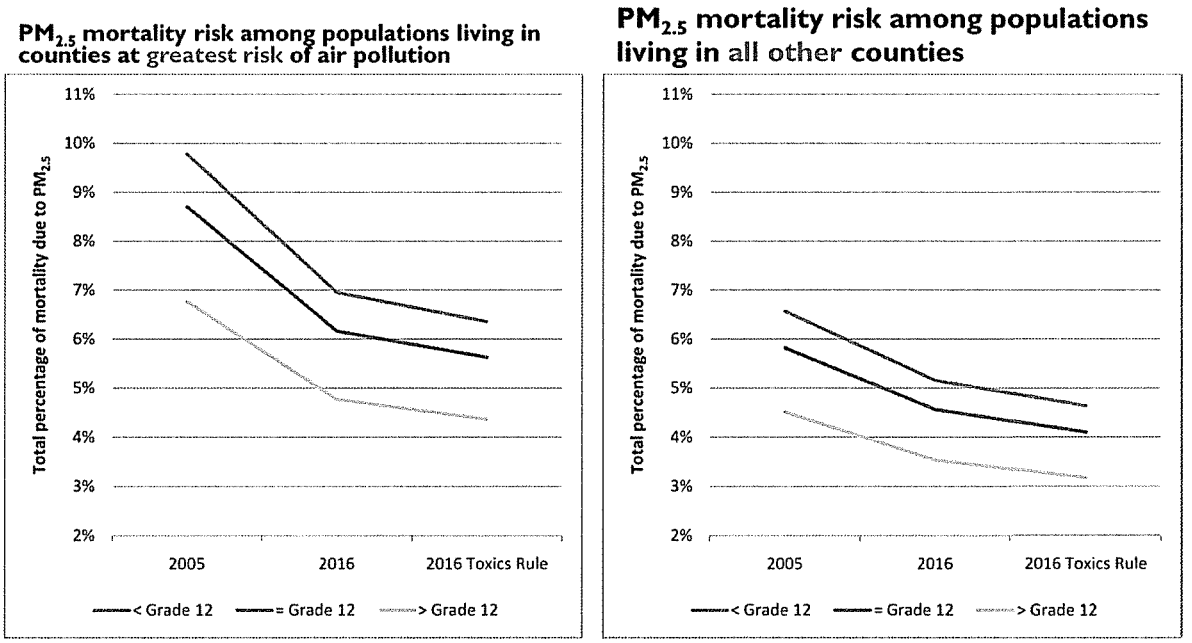


Figure C-7. Change in the Percentage of PM2.5-Attributable Deaths among Populations by Educational Attainment between 2005 and the Implementation of the Toxics Rule



Chapter 7
ELECTRIC POWER SECTOR PROFILE

This chapter discusses important aspects of the power sector that relate to the Air Toxics Rule, including the types of power-sector sources affected by the Rule, and provides background on the power sector and electric generating units (EGUs).

7.1 Power Sector Overview

The production and delivery of electricity to customers consists of three distinct segments: generation, transmission, and distribution.

7.1.1 Generation

Electricity generation is the first process in the delivery of electricity to consumers. Most of the existing capacity for generating electricity involves creating heat to rotate turbines which, in turn, create electricity. The power sector consists of over 17,000 generating units, comprising fossil-fuel-fired units, nuclear units, and hydroelectric and other renewable sources dispersed throughout the country (see Table 7-1).

Table 7-1. Existing Electricity Generating Capacity by Energy Source, 2009

Energy Source	Number of Generators	Generator Nameplate Capacity (MW)	Generator Net Summer Capacity (MW)
Coal	1,436	338,723	314,294
Petroleum	3,757	63,254	56,781
Natural Gas	5,470	459,803	401,272
Other Gases	98	2,218	1,932
Nuclear	104	106,618	101,004
Hydroelectric Conventional	4,005	77,910	78,518
Wind	620	34,683	34,296
Solar Thermal and Photovoltaic	110	640	619
Wood and Wood Derived Fuels	353	7,829	6,939
Geothermal	222	3,421	2,382
Other Biomass	1,502	5,007	4,317
Pumped Storage	151	20,538	22,160
Other	48	1,042	888
Total	17,876	1,121,686	1,025,400

Source: EIA Electric Power Annual 2009, Table 1.2

These electric generating sources provide electricity for commercial, industrial, and residential uses, each of which consumes roughly a quarter to a third of the total electricity produced (see Table 7-2). Some of these uses are highly variable, such as heating and air

conditioning in residential and commercial buildings, while others are relatively constant, such as industrial processes that operate 24 hours a day.

Table 7-2. Total U.S. Electric Power Industry Retail Sales in 2009 (Billion kWh)

		Sales/Direct Use (Billion kWh)	Share of Total End Use
Retail Sales	Residential	1,364	37%
	Commercial	1,307	35%
	Industrial	917	25%
	Transportation	8	0.2%
Direct Use		127	3%
Total End Use		**3,724**	**100%**

Source: EIA Electric Power Annual 2009, Table 7.2

In 2009, electric generating sources produced 3,950 billion kWh to meet electricity demand. Roughly 70 percent of this electricity was produced through the combustion of fossil fuels, primarily coal and natural gas, with coal accounting for almost half of the total (see Table 7-3).

Table 7-3. Electricity Net Generation in 2009 (Billion kWh)

	Net Generation (Billion kWh)	Fuel Source Share
Coal	1,756	44.5%
Petroleum	39	1.0%
Natural Gas	921	23.3%
Other Gases	11	0.3%
Nuclear	799	20.2%
Hydroelectric	273	6.9%
Other	151	3.8%
Total	3,950	100.0%

Source: EIA Electric Power Annual 2009, Table 1.1

Note: Retail sales and net generation are not equal because net generation includes net exported electricity and loss of electricity that occurs through transmission and distribution.

Coal-fired generating units typically supply "base-load" electricity, the portion of electricity loads which are continually present, and typically operate throughout the day. Along with nuclear generation, these coal units meet the part of demand that is relatively constant. Although much of the coal fleet operates as base load, there can be notable differences across various facilities (see Table 7-4). For example, coal-fired units less than 100 MW in size comprise 37 percent of the total number of coal-fired units, but only 6 percent of total coal-fired capacity. Gas-fired generation is better able to vary output and is the primary option used to meet the variable portion of the electricity load and typically supplies "peak" power, when there is increased demand for electricity (for example, when businesses operate throughout the day or

when people return home from work and run appliances and heating/air-conditioning), versus late at night or very early in the morning, when demand for electricity is reduced.

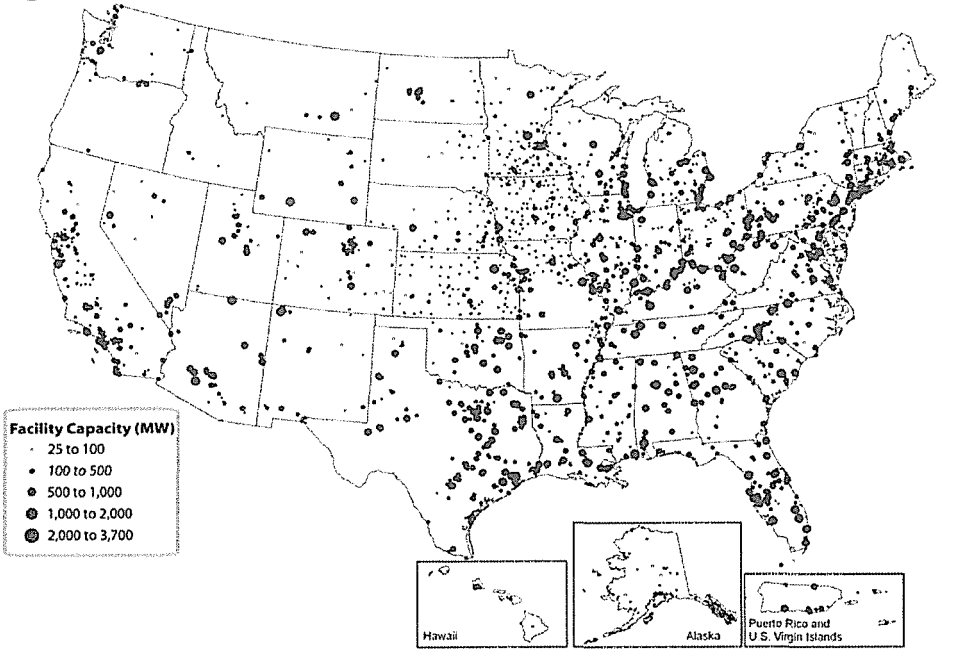
Table 7-4. Coal Steam Electricity Generating Units, by Size, Age, Capacity, and Efficiency (Heat Rate)

Unit Size Grouping (MW)			No. Units	% of All Units	Avg. Age	Avg. Capacity (MW)	Total Capacity (MW)	% Total Capacity	Avg. Heat Rate (Btu/kWh)
0	*to*	25	193	15%	45	15	2,849	1%	11,154
>25	*to*	49	108	9%	42	38	4,081	1%	11,722
50	*to*	99	162	13%	47	75	12,132	4%	11,328
100	*to*	149	269	21%	49	141	38,051	12%	10,641
150	*to*	249	81	6%	43	224	18,184	6%	10,303
250	*and up*		453	36%	34	532	241,184	76%	10,193
Totals			1,266				316,480		

Source: National Electric Energy Data System (NEEDS) v.4.10

Notes: A lower heat rate indicates a higher level of efficiency. Table is limited to coal-steam units online in 2010 or earlier.

Figure 7-1. Fossil Fuel-Fired Electricity Generating Units, by Size



Notes/Source: National Electric Energy Data System (NEEDS 4.10) (EPA, December 2010). This map displays facilities in the NEEDS 4.10 IPM frame. NEEDS reflects available capacity on-line by the end of 2011; this includes committed new builds and committed retirements. In areas with a dense concentration of facilities, some facilities may be obscured.

7.1.2 Transmission

Transmission is the term used to describe the movement of electricity over a network of high voltage lines, from electric generators to substations where power is stepped down for local distribution. In the US and Canada, there are three separate interconnected networks of high voltage transmission lines[1], each operating at a common frequency. Within each of these transmission networks, there are multiple areas where the operation of power plants is monitored and controlled to ensure that electricity generation and load are kept in balance. In some areas, the operation of the transmission system is under the control of a single regional operator; in others, individual utilities coordinate the operations of their generation, transmission, and distribution systems to balance their common generation and load needs.

7.1.3 Distribution

Distribution of electricity involves networks of lower voltage lines and substations that take the higher voltage power from the transmission system and step it down to lower voltage levels to match the needs of customers. The transmission and distribution system is the classic example of a natural monopoly, in part because it is not practical to have more than one set of lines running from the electricity generating sources to substations or from substations to residences and business.

Transmission has generally been developed by the larger vertically integrated utilities that typically operate generation and distribution networks. Distribution is handled by a large number of utilities that often purchase and sell electricity, but do not generate it. Transmission and distribution have been considered differently from generation in efforts to restructure the industry. As discussed below, electricity restructuring has focused primarily on efforts to reorganize the industry to encourage competition in the generation segment of the industry, including ensuring open access of generation to the transmission and distribution services needed to deliver power to consumers. In many state efforts, this has also included separating generation assets from transmission and distribution assets into distinct economic entities. Transmission and distribution remain price-regulated throughout the country based on the cost of service.

[1] These three network interconnections are the western US and Canada, corresponding approximately to the area west of the Rocky Mountains; eastern US and Canada, not including most of Texas; and a third network operating in most of Texas. These are commonly referred to as the Western Interconnect Region, Eastern Interconnect Region, and ERCOT, respectively.

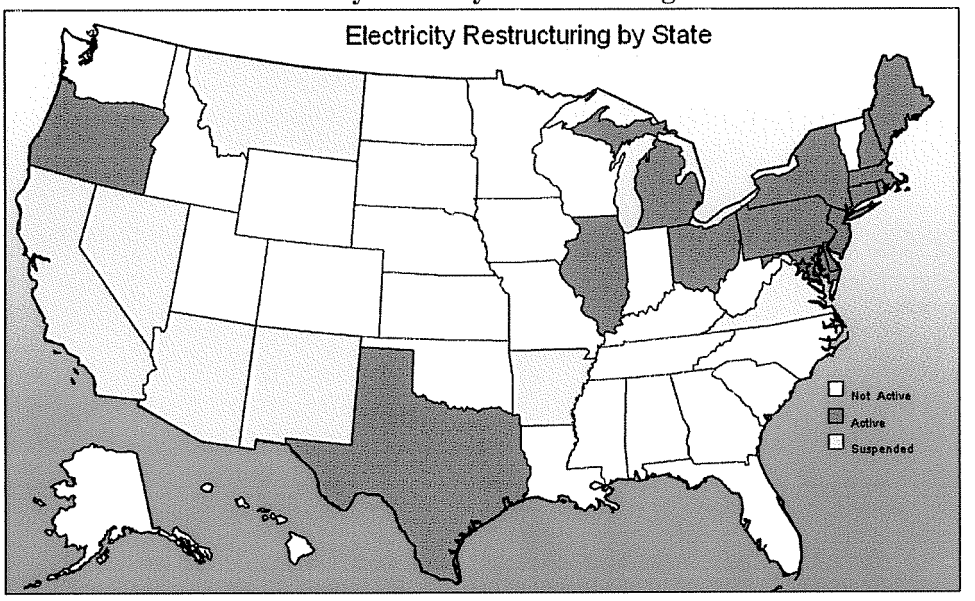
7.2 Deregulation and Restructuring

The process of restructuring and deregulation of wholesale and retail electric markets has changed the structure of the electric power industry. In addition to reorganizing asset management between companies, restructuring sought a functional unbundling of the generation, transmission, distribution, and ancillary services the power sector has historically provided, with the aim of enhancing competition in the generation segment of the industry.

Beginning in the 1970s, government policy shifted against traditional regulatory approaches and in favor of deregulation for many important industries, including transportation (notably commercial airlines), communications, and energy, which were all thought to be natural monopolies (prior to 1970) that warranted governmental control of pricing. However, deregulation efforts in the power sector were most active during the 1990s. Some of the primary drivers for deregulation of electric power included the desire for more efficient investment choices, the possibility of lower electric rates, reduced costs of combustion turbine technology that opened the door for more companies to sell power, and complexity of monitoring utilities' cost of service and establishing cost-based rates for various customer classes (see Figure 7-2).

The pace of restructuring in the electric power industry slowed significantly in response to market volatility and financial turmoil associated with bankruptcy filings of key energy companies in California. By the end of 2001, restructuring had either been delayed or suspended in eight states that previously enacted legislation or issued regulatory orders for its implementation (shown as "Suspended" in Figure 7-2 below). Another 18 other states that had seriously explored the possibility of deregulation in 2000 reported no legislative or regulatory activity in 2001 (DOE, EIA, 2003a) ("Not Active" in Figure 7-2 below). Currently, there are 15 states where price deregulation of generation (restructuring) has occurred ("Active" in Figure 7-2 below). The effort is more or less at a standstill; there have been no recent proposals to the Federal Energy Regulatory Commission (FERC) for actions aimed at wider restructuring, and no additional states have begun retail deregulation activity

Figure 7-2. Status of State Electricity Industry Restructuring Activities



Source: EIA http://www.eia.doe.gov/cneaf/electricity/page/restructuring/restructure_elect.html (September 2010).

7.3 Pollution and EPA Regulation of Emissions

The burning of fossil fuels, which generates about 70 percent of our electricity nationwide, results in air emissions of Hazardous Air Pollutants (HAPs): mercury, acid gasses, and non-mercury metallic particulates. Additionally, SO_2 and NO_x emissions from the power sector are important precursors in the formation of fine particles and ozone (NO_x only). The power sector is a major contributor of all of these pollutants.

Fossil fuel-fired units vary widely in their air emissions levels for HAPs, particularly when uncontrolled. In 2009, based on the Utility MACT Information Collection Request, HCl emissions from coal-fired units range from less than 0.00002 lb/mmBtu (for a unit with a scrubber) to over 0.1 lb/mmBtu. Mercury emissions range from less than 0.3 lb/TBtu to more than 20 lbs/TBtu. Emissions of fine particulates less than or equal to 2.5 microns ($PM_{2\,5}$) range from 0.002 lb/mmBtu to over 0.06 lb/mmBtu. For an uncontrolled plant, acid gas, mercury, and particulate emissions are directly related to the elemental profile and ash content of the coal burned.

Oil-fired units also have a wide range of HAP emissions. In 2009, based on the Utility MACT Information Collection Request, HCl emissions from oil-fired units range from less than 0.00001 lb/mmBtu (for a unit with a scrubber) to over 0.003 lb/mmBtu. Mercury emissions

range from less than 0.01 lb/TBtu to more than 60 lbs/TBtu. Emissions $PM_{2.5}$ range from less than 0.004 lb/mmBtu to over 0.07 lb/mmBtu.

7.4 Pollution Control Technologies

Acid gas HAPs (e.g., hydrogen chloride (HCl), hydrogen fluoride (HF), sulfur dioxide (SO_2)) from coal-fired power plants are controlled by fuel selection, fuel blending, or post combustion controls. Fossil fuels, and particularly coal, vary widely in the content of pollutants like chlorine (Cl), fluorine (F), sulfur (S) and other HAPs, making fuel blending and/or switching an effective method for reducing emissions of HAPs. In general, it is easier to switch fuels within a coal rank (rather than across a coal rank) due to similar heat contents and other characteristics. Completely switching fuels across ranks tends to trigger more costly modifications. As a compromise, blending is employed when a complete fuel switch adversely affects the unit. Electric generating units (EGUs) may also choose to retrofit post combustion controls to achieve superior pollutant removal. Post-combustion controls typically remove larger proportions of HCl and HF than SO_2 due to differences in molecular weight.

Acid gas emissions (including SO_2) can be reduced with flue gas desulfurization (FGD, also known as "scrubbers") or with dry sorbent injection (DSI). EGUs may choose either "wet" and "dry" configurations of scrubbers. Wet scrubbers can use a variety of reagents including crushed limestone, quick lime, and magnesium-enhanced lime. The choice of reagent affects performance, size, capital and operating costs. Current wet scrubber technology is capable of removing at least 99 percent of HF and HCl emissions while simultaneously achieving 96 percent SO_2 removal. Modern dry FGD technology incorporates a lime-based slurry with a downstream fabric filter to remove at least 93 percent SO_2 while also capturing over 99 percent HCL and HF. An alternative to wet and dry scrubber technology is dry sorbent injection (DSI), which injects an alkaline powdered material (post combustion) to react with the acid gases. The reacted product is removed by particulate matter (PM) control device. DSI technology is most efficient with a baghouse present downstream but can function with an electrostatic precipitator (ESP) downstream as well. Under these circumstances, the ESP requires more reagent per molecule of acid gas removed as compared to a similar operation with a baghouse. Finally, DSI may employ a multitude of sorbents (trona[1], sodium carbonate, calcium carbonate – and their bicarbonate counterparts) for a more tailored approach to reduce emissions based on the source, cost, and unit and fuel characteristics.

[1] Trona refers to the chemical compound sodium sesquicarbonate.

Mercury capture requires multiple controls to achieve removal. Upon combustion, mercury exits the furnace in three forms: elemental, oxidized, and as a particulate. Elemental mercury is emitted out of the stack. The particulate form is bound to the ash and removed by PM control equipment - either ESP or fabric filter. A portion of mercury that has converted to oxidized compounds may be removed by either a wet scrubber or by activated carbon injection (ACI). The removal mechanism is different between these two control devices; the wet FGD system captures oxidized mercury because it is water soluble, while activated carbon injection provides a unique physical surface to which oxidized mercury can absorb. Mercury oxidation can occur at multiple locations within a unit as long as an oxidizing agent (namely, a halogen) is present for reaction; this allows the unit operator some latitude in selecting a control method and injection point based on existing equipment at the particular source. Halogen can be introduced to the fuel prior to combustion, injected directly into the furnace, introduced upstream of a selective catalytic reduction (SCR) system (primarily used for NO_x control but which also promotes mercury oxidation), infused with the activated carbon injections, or the unit operator may increase halogens by blending in higher chlorine fuels (e.g., Powder River Basin fuel blended with bituminous coal). Operating a wet FGD for SO_2 control alongside selective catalytic reduction (SCR) for NO_x control with sufficient halogen present will remove more than 90 percent of the mercury within the flue gas stream. On the other hand, if the existing unit is absent a wet FGD, activated carbon injection (ACI) can be employed for mercury capture with at least 90% removal using a downstream fabric filter; an ESP results in less efficient mercury removal with ACI.

Non-mercury heavy metals and organics are removed by PM control equipment such as fabric filters (FF) and electrostatic precipitators (ESP). Unlike mercury, the heavy metals (e.g., selenium and arsenic) are non-volatile and affix to the ash. Likewise, any organics surviving the high temperature combustion process are non-volatile and bind to the ash. Both control technologies are capable of removing more than 99 percent of particulates greater than 2.5 microns in size ($PM_{2\,5}$) from the emissions stream. ESPs sap relatively little pressure (energy) from the flue gas but are less flexible to fuel switching, since their design basis focuses on a specific intended fuel. Fuel switching or blending that increases gas flow rate, ash resistivity, or particle loading may render an existing ESP insufficient for removing particulate matter. ESPs also suffer from ash re-entrainment, which is the release of particulate matter from the last compartment due to the self cleaning action). On the other hand, an ESP with sufficient design margin may succeed with these fuel alterations. Conversely, a fabric filter does not suffer from these particulate removal limitations. Moreover, the fabric filter readily lends itself to mercury and acid gas removal since DSI and ACI operate more efficiently with a baghouse. When

considering retrofit PM control options, a unit with an existing ESP will examine upgrading the precipitator as an alternative to installing a new fabric filter to achieve emission reductions.

For more detail on the cost and performance assumptions of pollution controls, see the documentation for the Integrated Planning Model (IPM), a dynamic linear programming model that EPA uses to examine air pollution control policies for various air emissions throughout the United States for the entire power system. Documentation for IPM can be found at www.epa.gov/airmarkets/epa-ipm.

7.5 Air Regulation of the Power Sector

At the federal level, efforts to reduce emissions have been occurring since 1970. Policy makers have recognized the need to address these harmful emissions, and incremental steps have been taken to ensure that the country meets air quality standards. The Toxics Rule is the next step towards further protecting public health by reducing harmful HAP emissions.

7.5.1 SO_2 and NO_X Reduction

Even before widespread regulation of SO_2 and NO_x for the power sector, total suspended particulate matter (TSP) was a related target of state and federal action. Because larger particulates are visible as dark smoke from smokestacks, most states had regulations by 1970 limiting the opacity of emissions. Requirements for taller smokestacks also mitigated local impacts of TSP. Notably, such regulations effectively addressed large-diameter, filterable particulate matter rather than condensable particulate matter (such as $PM_{2.5}$) associated with SO_2 and NO_x emissions, which are not visible at the smokestack and have impacts far from their sources.

Federal regulation of SO_2 and NO_x emissions at power plants began with the 1970 Clean Air Act. The Act required the Agency to develop New Source Performance Standards (NSPS) for a number of source categories including coal-fired power plants. The first NSPS for power plants (subpart D) required new units to limit SO_2 emissions either by using scrubbers or by using low sulfur coal. NO_x was required to be limited through the use of low NO_x burners. A new NSPS (subpart Da), promulgated in 1978, tightened the standards for SO_2, requiring scrubbers on all new units.

The 1990 Clean Air Act Amendments (CAAA) placed a number of new requirements on power plants. The Acid Rain Program, established under Title IV of the 1990 CAAA, requires major reductions of SO_2 and NO_x emissions. The SO_2 program sets a permanent cap on the total amount of SO_2 that can be emitted by electric power plants in the contiguous United States at

about one-half of the amount of SO_2 these sources emitted in 1980. Using a market-based cap and trade mechanism allows flexibility for individual combustion units to select their own methods of compliance with the SO_2 reduction requirements. The program uses a more traditional approach to NO_x emissions limitations for certain coal-fired electric utility boilers, with the objective of achieving a 2 million ton reduction from projected NO_x emission levels that would have been emitted in 2000 without implementation of Title IV.

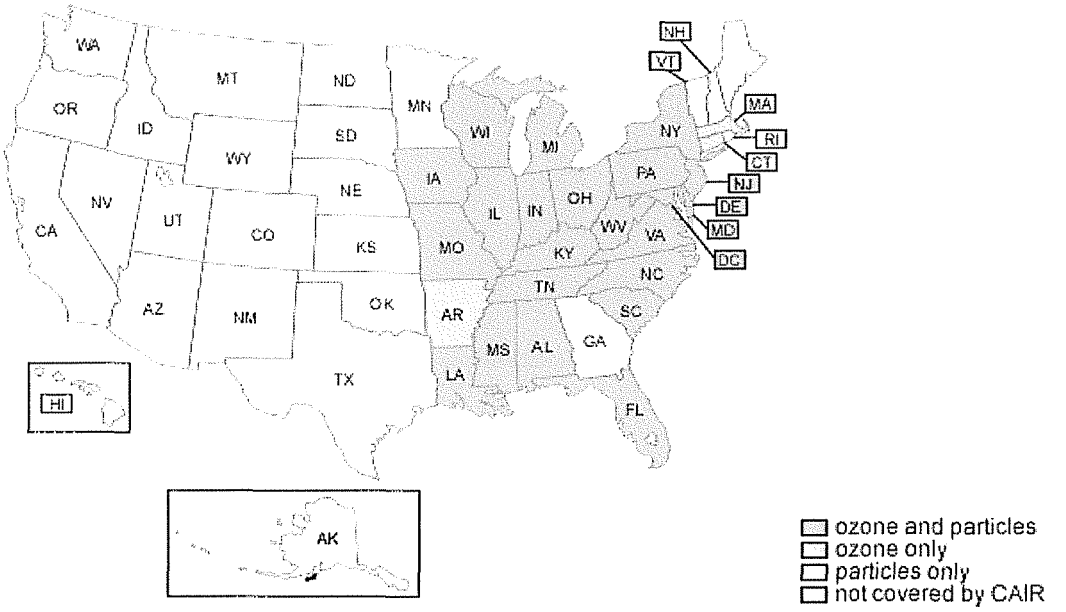
The Acid Rain Program comprises two phases for SO_2 and NO_x. Phase I applied primarily to the largest coal-fired electric generating sources from 1995 through 1999 for SO_2 and from 1996 through 1999 for NO_x. Phase II for both pollutants began in 2000. For SO_2, it applies to thousands of combustion units generating electricity nationwide; for NO_x it generally applies to affected units that burned coal during 1990 through 1995. The Acid Rain Program has led to the installation of a number of scrubbers on existing coal-fired units as well as significant fuel switching to lower sulfur coals. Under the NO_x provisions of Title IV, most existing coal-fired units installed low NO_x burners.

The CAAA also placed much greater emphasis on control of NO_x to reduce ozone nonattainment. This led to the formation of several regional NO_x trading programs as well as intrastate NO_x trading programs in states such as Texas. The northeastern states of the Ozone Transport Commission (OTC) required existing sources to meet Reasonably Available Control Technology (RACT) limits on NO_x in 1995 and in 1999 began an ozone-season cap and trade program to achieve deeper reductions. In 1998, EPA promulgated regulations (the NO_x SIP Call) that required 21 states in the eastern United States and the District of Columbia to reduce NO_x emissions that contributed to nonattainment in downwind states using the cap and trade approach. This program began in May of 2003 and has resulted in the installation of significant amounts of selective catalytic reduction.

The Clean Air Interstate Rule (CAIR) built on EPA's efforts in the NO_x SIP call to address specifically interstate pollution transport for ozone, and was EPA's first attempt to address interstate pollution transport for $PM_{2.5}$. It required significant reductions in emissions of SO_2 and NO_x in 28 states and the District of Columbia (see Figure 7-3 below). EGUs were found to be a major source of the SO_2 and NO_x emissions which contributed to fine particle concentrations and ozone problems downwind. Although the D.C. Circuit remanded the rule to EPA in 2008, it did so without vacatur, allowing the rule to remain in effect while EPA addresses the remand. Thus, CAIR is continuing to help states address ozone and $PM_{2.5}$ nonattainment and improve visibility by reducing transported precursors of SO_2 and NO_x through the

implementation of three separate cap and trade compliance programs for annual NO_x, ozone season NO_x, and annual SO_2 emissions from power plants.

Figure 7-3. States Covered under the Clean Air Interstate Rule



Perhaps in anticipation of complying with CAIR, especially the more stringent second phase that was set to begin in 2015, several sources have recently been installing or planning to install advanced controls for SO_2 and NO_x to begin operating in the 2010 to 2015 timeframe. Many EPA New Source Review (NSR) settlements also require controls in those years, as do state rules in Georgia, Illinois, and Maryland. States like North Carolina, New York, Connecticut, Massachusetts, and Delaware have also moved to control these emissions to address nonattainment. Thus both federal and state efforts are continuing to bring about sizeable reductions in SO_2 and NO_x from the power sector.

On July 6, 2010, the U.S. EPA proposed the Transport Rule, designed to replace CAIR. A December 2008 court decision kept the requirements of CAIR in place temporarily but directed EPA to issue a new rule to implement the Clean Air Act requirements concerning the transport of air pollution across state boundaries. The proposed rule would require 31 states and the District of Columbia to reduce SO_2 and NOx emissions, which contribute to ozone and fine particle pollution in other states, beginning in 2012 (see Figure 7-4).

Figure 7-4. States Covered under the Transport Rule

7.5.2 HAP Regulation

In 2000, EPA made a finding that it was appropriate and necessary to regulate coal- and oil-fired electric utility steam generating units (EGUs) under CAA section 112 and listed EGUs pursuant to CAA section 112(c). On March 29, 2005 (70 FR 15,994), EPA published a final rule (Section 112(n) Revision Rule) that removed EGUs from the list of sources for which regulation under CAA section 112 was required. That rule was published in conjunction with a rule requiring reductions in emissions of mercury from electric utility steam generating units pursuant to section 111 of the CAA (Clean Air Mercury Rule (CAMR), May 18, 2005, 70 FR 28606). The Section 112(n) Revision Rule was vacated on February 8, 2008, by the U.S. Court of Appeals for the District of Columbia Circuit. As a result of that vacatur, CAMR was also vacated and EGUs remain on the list of sources that must be regulated under CAA section 112. This action provides EPA's proposed rule in response to the court's decisions. Under authority of section 112 of the Clean Air Act (CAA), EPA is proposing a national emissions standard for HAP emissions, covering EGUs (also known as the Toxics Rule). The pollution control required to make HAP reductions will also result in SO_2 and NO_X reductions.

7.6 Revenues, Expenses, and Prices

Due to lower retail electricity sales, total utility operating revenues declined in 2009 to $276 billion from a peak of almost $300 billion in 2009. However, operating expenses were appreciably lower and as a result, net income actually rose modestly compared to 2008 (see

Table 7-5). Recent economic events have put downward pressure on electricity demand, thus dampening electricity prices (utility revenues), but have also reduced the price and cost of fossil fuels and other expenses. Electricity sales and revenues associated with the generation, transmission, and distribution of electricity are expected to rebound and increase modestly by 2015, where they are projected to be roughly $360 billion (see Table 7-6).

Table 7-5. Revenue and Expense Statistics for Major U.S. Investor-Owned Electric Utilities for 2009 ($millions)

Utility Operating Revenues	**276,124**
Electric Utility	249,303
Other Utility	26,822
Utility Operating Expenses	**244,243**
Electric Utility	219,544
Operation	154,925
Production	118,816
Cost of Fuel	40,242
Purchased Power	67,630
Other	10,970
Transmission	6,742
Distribution	3,947
Customer Accounts	5,203
Customer Service	3,857
Sales	178
Administrative and General	15,991
Maintenance	14,092
Depreciation	20,095
Taxes and Other	29,081
Other Utility	24,698
Net Utility Operating Income	**31,881**

Source: EIA Electric Power Annual 2009, Table 8.1

Note: This data does not include information for public utilities.

Table 7-6. Projected Revenues by Service Category in 2015 for Public Power *and* Investor-Owned Utilities (billions)

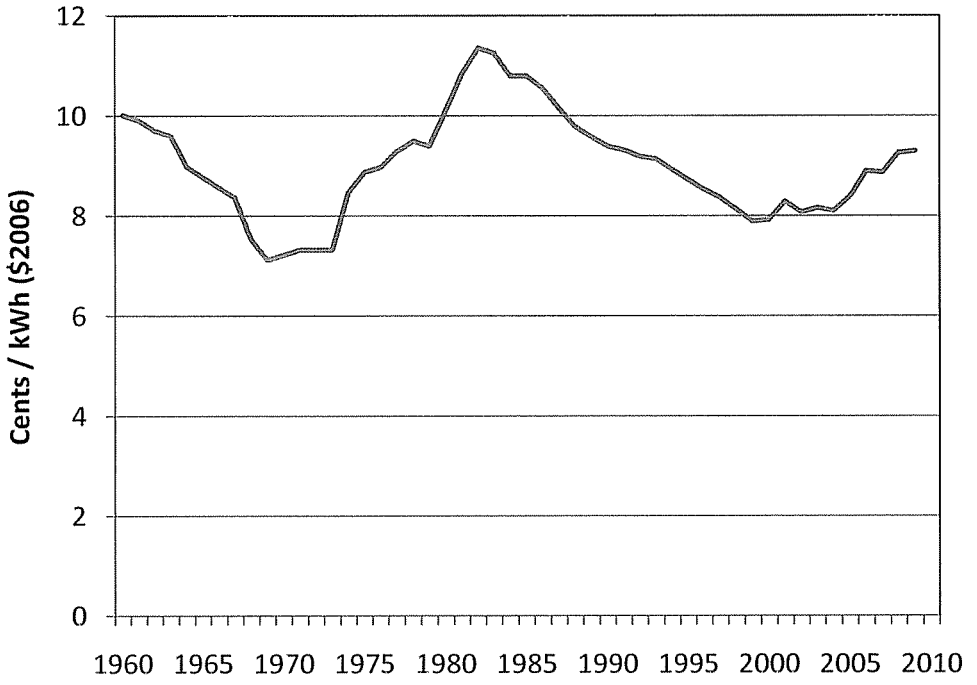
Generation	$195
Transmission	$36
Distribution	$129
	$360

Notes: Data is from EIA's AEO 2011, and is derived by taking either total electricity use (for generation) or sales (transmission and distribution) and multiplying by forecasted prices by service category from Table 8 (Electricity Supply, Disposition, Prices, and Emissions).

Based on EIA's Annual Energy Outlook 2011, Table 7-6 shows that in the base case, the power sector is expected to derive revenues of $360 billion in 2015. Table 7-5 shows that investor-owned utilities (IOUs) earned income of about 11.5% compared to total revenues in 2009. Assuming the same income ratio from IOUs (with no income kept by public power), and using the same proportion of power sales from public power as observed in 2009, EPA projects that the power sector will expend over $320 billion in 2015 alone to generate, transmit, and distribute electricity to end-use consumers.

Over the past 50 years, real retail electricity prices have ranged from around 7 cents per kWh in the early 1970's, to around 11 cents, reached in the early 1980's. Generally, retail electricity prices do not change rapidly and do not display the variability of other energy or commodity prices. Retail rate regulation has largely insulated consumers from the rising and falling wholesale electricity price signals whose variation on an hourly, daily, and seasonal basis is critical for driving lowest-cost matching of supply and demand. In fact, the real price of electricity today is lower than it was in the early 1960s and 1980s (see Figure 7-5).

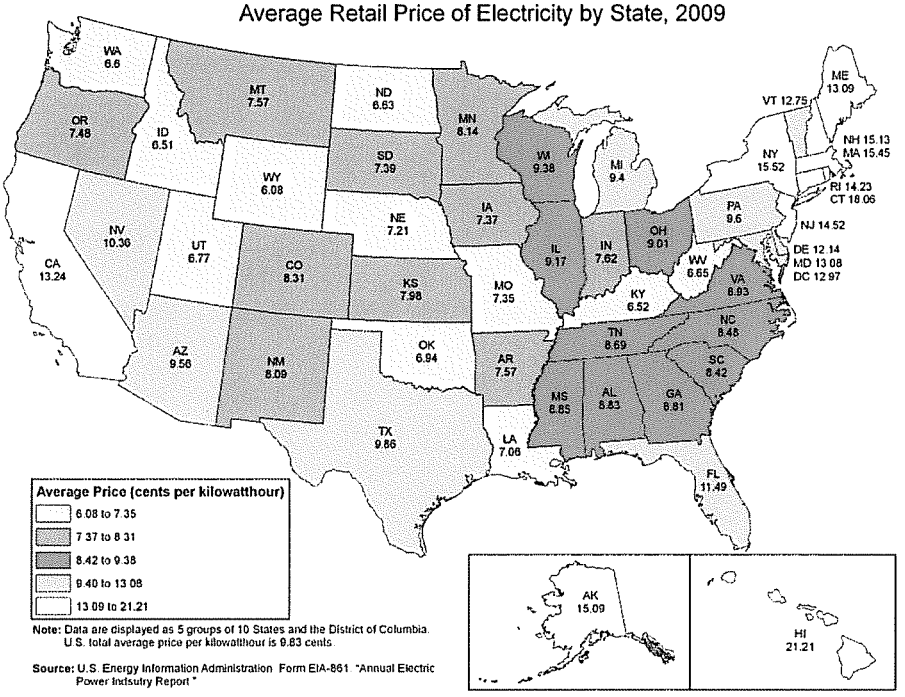
Figure 7-5. National Average Retail Electricity Price (1960 – 2009)



Source: EIA's Annual Energy Review 2009

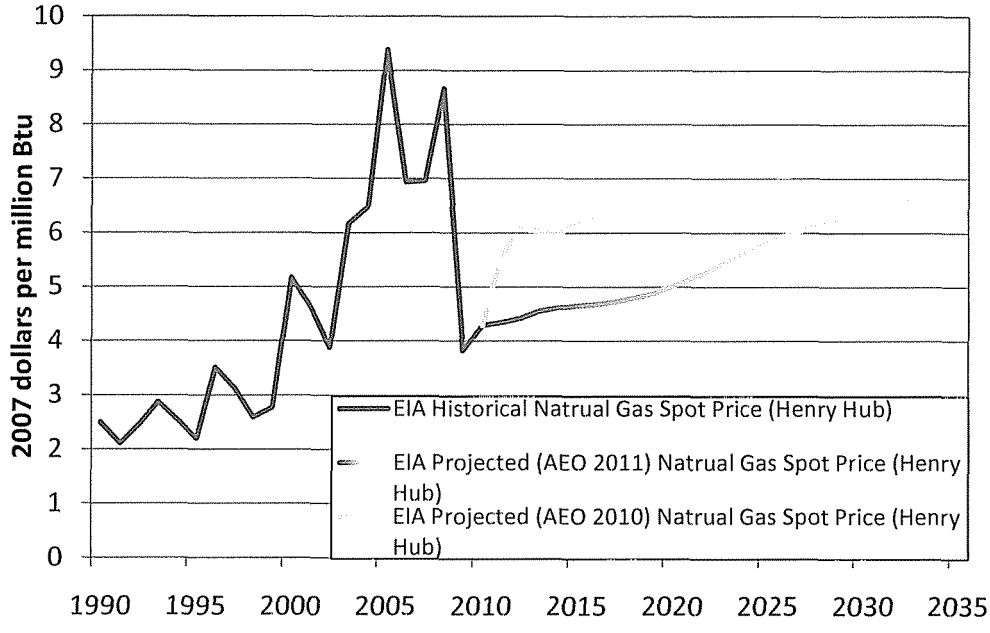
On a state-by-state basis, retail electricity prices vary considerably. The Northeast and California have average retail prices that can be as much as double those of other states. (see Figure 7-6)

Figure 7-6. Average Retail Electricity Price by State (cents/kWh), 2009



The natural gas market in the United States has historically experienced significant price volatility from year to year, between seasons within a year, and can even undergo major price swings during short-lived weather events (such as cold snaps leading to short-run spikes in heating demand). Over the last decade, gas prices have ranged from $3 per mmBtu to as high as $9 on an annual average basis (see Figure 7-7). During that time, the daily price of natural gas reached as high as $15/mmBtu. Recent forecasts of natural gas have also experienced considerable revision as new sources of gas have been discovered and come to market.

Figure 7-7. Natural Gas Spot Price, Annual Average (Henry Hub)

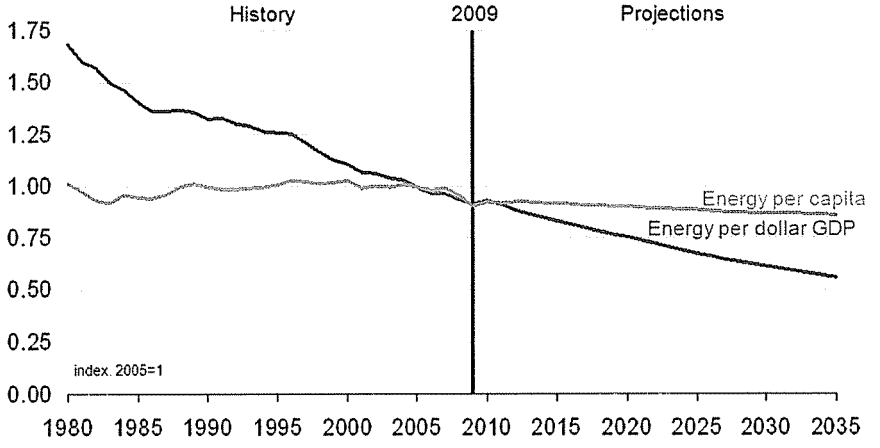


Source: EIA

7.7 Electricity Demand and Demand Response

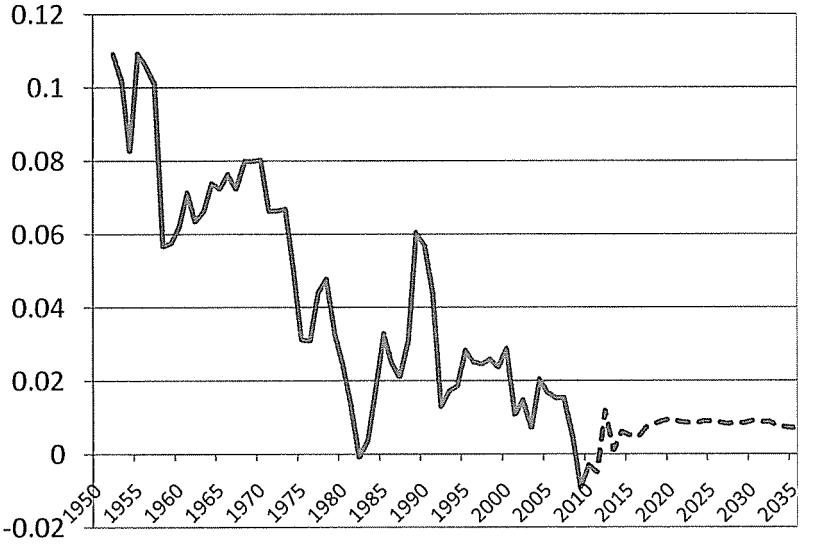
Electricity performs a vital and high-value function in the economy. Historically, growth in electricity consumption has been closely aligned with economic growth. Overall, the U.S. economy has become more efficient over time, producing more output (GDP) per unit of energy input, with per capita energy use fairly constant over the past 30 years (see Figure 7-8). The growth rate of electricity demand has also been in overall decline for the past sixty years (see Figure 7-9), with several key drivers that are worth noting. First, there has been a significant structural shift in the U.S. economy towards less energy-intensive sectors, like services. Second, companies have strong financial incentives to reduce energy expenditures. Third, companies are responding to the marketplace and continually develop and bring to market new technologies that reduce energy consumption. Fourth, complementary policies and energy efficiency standards at the state and Federal level have helped address market failures. These broader changes have altered the outlook for future electricity growth (see Figure 7-9).

Figure 7-8. Energy Use per Capita and per 2005 Dollar of GDP



Source: EIA AEO 2011

Figure 7-9. Electricity Growth Rate (3 Year Rolling Average) and Projections from the Annual Energy Outlook 2011



Source: EIA Annual Energy Review 2009 and Annual Energy Outlook 2011

Energy efficiency initiatives have become more common, and investments in energy efficiency are projected to continue to increase for the next 5 to 10 years, driven in part by the growing number of states that have adopted energy efficiency resource standards. These investments, and other energy efficiency policies at both the state and federal level, create incentives to reduce energy consumption and peak load. According to EIA, demand-side

management provided actual peak load reductions of 31.7 GW in 2009. For context, the current coal fleet is roughly 320 GW of capacity.

Demand for electricity, especially in the short run, is not very sensitive to changes in prices and is considered relatively price inelastic, although some demand reduction does occur in response to price. With that in mind, EPA modeling does not typically incorporate a "demand response" in its electric generation modeling (Chapter 8) to the increases in electricity prices typically projected for EPA rulemakings. Electricity demand is considered to be constant in EPA modeling applications and the reduction in production costs that would result from lower demand is not considered in the primary analytical scenario that is modeled. This leads to some overstatement in the private compliance costs that EPA estimates. Notably, the "compliance costs" are the changes in the electric power generation costs in the base case and pollution control options that are evaluated in Chapter 8. In simple terms, it is the resource costs of what the power industry will directly expend to comply with EPA's requirements.

7.8 Reference

EIA Electric Power Annual 2009. DOE/EIA-0348 (2008). Available at: http://www.eia.doe.gov/cneaf/electricity/epa/epa_sum.htm

EIA Annual Energy Outlook 2011

Chapter 8
COST, ECONOMIC, AND ENERGY IMPACTS

This chapter reports the cost, economic, and energy impact analysis performed for the Toxics Rule. EPA used the Integrated Planning Model (IPM), developed by ICF Consulting, to conduct its analysis. IPM is a dynamic linear programming model that can be used to examine air pollution control policies for SO_2, NO_x, Hg, HCl, and other air pollutants throughout the United States for the entire power system. Documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm, and updates specific to the Toxics Rule modeling are in the "Documentation Supplement for EPA Base Case v.4.10_PTox – Updates for Proposed Toxics Rule."

8.1 Background

Over the last decade, EPA has on several occasions used IPM to consider control options for reducing power-sector emissions. Many EPA analyses with IPM have focused on legislative proposals with national scope, such as EPA's IPM analyses of the Clean Air Planning Act (S.843 in 108th Congress), the Clean Power Act (S.150 in 109th Congress), the Clear Skies Act of 2005 (S.131 in 109th Congress), the Clear Skies Act of 2003 (S.485 in 108th Congress), and the Clear Skies Manager's Mark (of S.131). These analyses are available at EPA's website: (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html). EPA also analyzed several multi-pollutant reduction scenarios in July 2009 at the request of Senator Tom Carper to illustrate the costs and benefits of multiple levels of SO_2 and NO_x control in the power sector.

In addition, EPA conducted extensive state-by-state analysis of control levels and associated emissions projections related to upwind pollution contribution across state borders to downwind air quality monitors for the proposed Transport Rule. More details on this analysis can be found in the Federal Register[1] and Significant Contribution Approach TSD for the proposed Transport Rule.[2]

As discussed in Chapter 7, the proposed Toxics Rule coincides with a period when many new pollution controls are being installed. Many are needed for compliance with NSR settlements and state rules, while others may have been planned in expectation of CAIR and its replacement, the Transport Rule. Because CAIR remains in effect until it is replaced by the Transport Rule, the power sector is continuing to make emission reductions in the eastern US.

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[1] F R. 45210
[2] http://www.epa gov/airquality/transport/tech html

The base case in this RIA assumes that the Transport Rule is in effect and takes into account emissions reductions associated with the implementation of all federal rules, state rules and statutes, and other binding, enforceable commitments in place by December 2010 that are applicable to the power industry and which govern the installation and operation of pollution controls in the timeframe covered in the analysis.

EPA has made these base case assumptions recognizing that the power sector will install a significant amount of pollution controls in response to several requirements. The inclusion of the proposed Transport Rule and other regulatory actions (including federal, state, and local actions) in the base case is necessary in order to reflect the level of controls that are likely to be in place in response to other requirements apart from the Toxics Rule. This base case will provide meaningful projections of how the power sector will respond to all the regulatory requirements for air emissions in totality, while isolating the incremental impacts of the proposed Toxics Rule relative to a base case with other air emission reduction requirements separate from today's action. While the Transport Rule could change when it is finalized, EPA believes that this updated modeling of the proposed Transport Rule is a satisfactory representation of requirements under the CAA that address air transport under 110(a)(2)(D)(i)(I), and subsequent analyses for the Toxics Rule after its proposal should reflect the Transport Rule as finalized.

The model's base case features an updated Title IV SO_2 allowance bank assumption and incorporates updates related to the Energy Independence and Security Act of 2007. Some modeling assumptions, most notably the projected demand for electricity, are based on the 2010 Annual Energy Outlook from the Energy Information Administration (EIA). In addition, the model includes existing policies affecting emissions from the power sector: the Title IV of the Clean Air Act (the Acid Rain Program); the NO_x SIP Call; various New Source Review (NSR) settlements;[1] and several state rules[2] affecting emissions of SO_2, NO_x, Hg, and CO_2 that were finalized through Fall of 2010. IPM includes state rules that have been finalized and/or approved by a state's legislature or environmental agency. The IPM documentation TSD contains details

[1] The NSR settlements include agreements between EPA and Southern Indiana Gas and Electric Company (Vectren), Public Service Enterprise Group, Tampa Electric Company, We Energies (WEPCO), Virginia Electric & Power Company (Dominion), Santee Cooper, Minnkota Power Coop, American Electric Power (AEP), East Kentucky Power Cooperative (EKPC), Nevada Power Company, Illinois Power, Mirant, Ohio Edison, Kentucky Utilities, Hoosier Energy, Salt River Project, Westar, Puerto Rico Power Authority, Duke Energy, American Municipal Power, and Dayton Power and Light. These agreements lay out specific NO_x, SO_2, and other emissions controls for the fleets of these major Eastern companies by specified dates. Many of the pollution controls are required between 2010 and 2015.

[2] These include current and future state programs in Alabama, Arizona, California, Colorado, Connecticut, Delaware, Georgia, Illinois, Kansas, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Montana, New Hampshire, New Jersey, New York, North Carolina, Oregon, Pennsylvania, Tennessee, Texas, Utah, Washington, West Virginia, and Wisconsin the cover certain emissions from the power sector.

on all of these other legally binding and enforceable commitments for installation and operation of pollution controls. This chapter focuses on results of EPA's analysis with IPM for the model's 2015 run-year in connection with the compliance date for the proposed Toxics Rule.

The proposed Toxics Rule establishes National Emissions Standards for Hazardous Air Pollutants (NESHAPS) for the "electric utility steam generating unit" source category, which includes those units that combust coal or oil for the purpose of generating electricity for sale and distribution through the national electric grid to the public.

Coal-fired electric utility steam generating units include electric utility steam generating units that burn coal, coal refuse, or a synthetic gas derived from coal either exclusively, in any combination together, or in any combination with other supplemental fuels. Examples of supplemental fuels include petroleum coke and tire-derived fuels. The NESHAP establishes standards for HAP emissions from both coal- and oil-fired EGUs and will apply to any existing, new, or reconstructed units located at major or area sources of HAP. Although all HAP are pollutants of interest, those of particular concern are hydrogen fluoride (HF), hydrogen chloride (HCl), dioxins/furans, and HAP metals, including antimony, arsenic, beryllium, cadmium, chromium, cobalt, mercury, manganese, nickel, lead, and selenium.

This rule affects any fossil fuel fired combustion unit of more than 25 megawatts electric (MWe) that serves a generator that produces electricity for sale. A unit that cogenerates steam and electricity and supplies more than one-third of its potential electric output capacity and more than 25 MWe output to any utility power distribution system for sale is also considered an electric utility steam generating unit. The rule would affect roughly 1,400 coal and oil or gas fired steam units with a nameplate capacity greater than 25 MW.

Tables 8-1 and 8-2 show the control requirements of the Toxics Rule that EPA has analyzed in the RIA. For further discussion about the scope and requirements of the Toxics Rule, see the Toxics Rule preamble or Chapter 2 of this RIA.

Table 8-1. Emissions Limitations for Coal-Fired and Solid Oil-Derived Fuel-Fired Electric Utility Steam Generating Units

Subcategory	Total particulate matter	Hydrogen chloride	Mercury
Existing coal-fired unit designed for coal ≥ 8,300 Btu/lb	0.03 lb/MMBtu (0.2 lb/MWh)	0.002 lb/MMBtu (0.02 lb/MWh)	1 lb/TBtu (0.02 lb/GWh)
Existing coal-fired unit designed for coal < 8,300 Btu/lb	0.03 lb/MMBtu (0.2 lb/MWh)	0.002 lb/MMBtu (0.02 lb/MWh)	11 lb/TBtu (0.2 lb/GWh) 4 lb/TBtu* (0.04 lb/GWh*)
Existing - IGCC	0.05 lb/MMBtu (0.3 lb/MWh)	0.0005 lb/MMBtu (0.003 lb/MWh)	3 lb/TBtu (0.02 lb/GWh)
Existing – Solid oil-derived	0.2 lb/MMBtu (2 lb/MWh)	0.005 lb/MMBtu (0.05 lb/MWh)	0.2 lb/TBtu (0.002 lb/GWh)
New coal-fired unit designed for coal ≥ 8,300 Btu/lb	0.05 lb/MWh	0.3 lb/GWh	0.00001 lb/GWh
New coal-fired unit designed for coal < 8,300 Btu/lb	0.05 lb/MWh	0.3 lb/GWh	0.04 lb/GWh
New – IGCC	0.05 lb/MWh*	0.3 lb/GWh*	0.00001 lb/GWh*
New – Solid oil-derived	0.05 lb/MWh	0.0003 lb/MWh	0.002 lb/GWh

Note: lb/MMBtu = pounds pollutant per million British thermal units fuel input
lb/TBtu = pounds pollutant per trillion British thermal units fuel input
lb/MWh = pounds pollutant per megawatt-electric output
lb/GWh = pounds pollutant per gigawatt-electric output

* Beyond-the-floor limit as discussed elsewhere.

Table 8-2. Emissions Limitations for Liquid Oil-Fired Electric Utility Steam Generating Units

Subcategory	Total HAP metals	Hydrogen chloride	Hydrogen fluoride	Mercury
Existing – Liquid oil	0.00003 lb/MMBtu (0.0003 lb/MWh)	0.0003 lb/MMBtu (0.003 lb/MWh)	0.0002 lb/MMBtu (0.002 lb/MWh)	0.05 lb/TBtu (0.0006 lb/GWh)
New – Liquid oil	0.0004 lb/MWh	0.0005 lb/MWh	0.0005 lb/MWh	0.0001 lb/GWh

EPA used the Integrated Planning Model (IPM) v.4.10 to assess the impacts of the proposed emission limitations for coal-fired electricity generating units (EGU) in the contiguous United States. IPM modeling did not subject oil-fired units to policy criteria.[1] Furthermore, IPM modeling did not include generation outside the contiguous U.S., where EPA is aware of only 2 facilities that would be subject to the coal-fired requirements of the proposed rule. Given the limited number of potentially impacted facilities, limited availability of input data to inform the modeling, and limited connection to the continental grid, EPA did not model the impacts of the proposed rule beyond the contiguous U.S.

Mercury emissions are modeled as a function of mercury content of the fuel type(s) consumed at each plant in concert with that plant's pollutant control configuration. HCl emissions are projected in a similar fashion using the chlorine content of the fuel(s). For both mercury and HCl, EGUs in the model must emit at or below the proposed mercury and HCl emission rate standards in order to operate from 2015 onwards. EGUs may change fuels and/or install additional control technology to meet the standard, or they may choose to retire if it is more economic for the power sector to meet electricity demand with other sources of generation. See IPM documentation for more details.

Total PM emissions are calculated exogenously, using EPA's Source Classification Code (SCC) and control-based emissions factors. SCC is a classification system that describes a generating unit's characteristics. In the policy case, EPA assumes that most coal- and solid-oil derived fuel-fired EGUs require a fabric filter (also known as a baghouse) to meet the total PM standard.

[1] EPA was not able to model the impacts of the proposed rule on oil-fired units. EPA plans to include an analysis of impacts on oil-fired units for the final rule.

Instead of emission limitations for the organic HAP, EPA is proposing that if requested, owners or operators of EGUs submit to the delegated authority or EPA, as appropriate, documentation showing that an annual performance test meeting the requirements of the proposed rule was conducted. IPM modeling assumes compliance with these work practice standards.

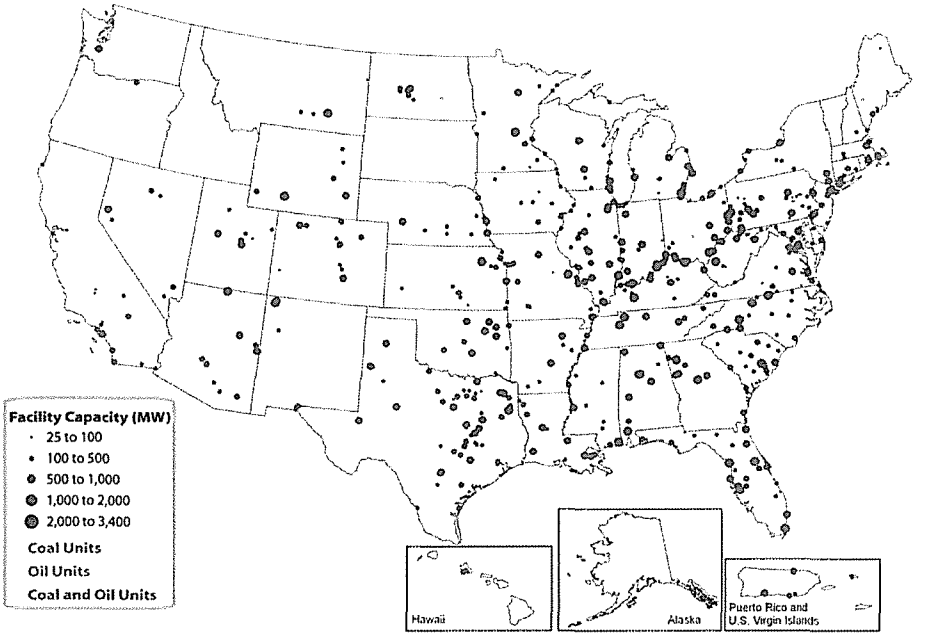
Electricity demand is anticipated to grow by roughly 1 percent per year, and total electricity demand is projected to be 4,104 billion kWh by 2015. Table 8-3 shows current electricity generation alongside EPA's base case projection for 2015 generation using IPM. EPA's IPM modeling for this rule relies on EIA's *Annual Energy Outlook for 2010*'s electric demand forecast for the US and employs a set of EPA assumptions regarding fuel supplies and the performance and cost of electric generation technologies as well as pollution controls. The base case includes the proposed Transport Rule (which upon finalization will replace the Clean Air Interstate Rule currently in place) as well as other existing state and federal programs for emissions control from electric generating units.

Table 8-3. 2009 U.S. Electricity Net Generation and EPA Base Case Projections for 2015-2030 (Billion kWh)

	Historical	Base Case		
	2009	2015	2020	2030
Coal	1,756	2,002	2,022	2,060
Oil	39	0.11	0.13	0.19
Natural Gas	921	694	834	1,162
Nuclear	799	825	835	814
Hydroelectric	273	286	286	286
Non-hydro Renewables	144	251	287	328
Other	18	45	46	0
Total	**3,950**	**4,104**	**4,309**	**4,704**

Source: 2009 data from EIA Electric Power Annual 2009, Table 2.1; Projections from Integrated Planning Model run by EPA, 2011.

Figure 8-1. Geographic Distribution of Affected Units, by Facility, Size and Fuel Source in 2012



Source/Notes: National Electric Energy Data System (NEEDS 4.10) (EPA, December 2010). This map displays all fossil facilities in the NEEDS 4.10 IPM framework. NEEDS reflects available capacity on-line by the end of 2011; this includes committed new builds and committed retirements. In areas with a dense concentration of facilities, some facilities may be obscured.

As noted above, IPM has been used for evaluating the economic and emission impacts of environmental policies for over a decade. The economic modeling presented in this chapter has been developed for specific analyses of the power sector. Thus, the model has been designed to reflect the industry as accurately as possible. To that end, EPA uses a series of capital charge factors in IPM that embody financial terms for the various types of investments that the power sector considers for meeting future generation and environmental constraints. The model applies a discount rate of 6.15% for optimizing the sector's decision-making over time. IPM's discount rate, designed to represent a broad range of private-sector decisions for power generation, rates differs from discount rates used in other analyses in this RIA, such as the benefits and macroeconomic analyses which each assume alternative social discount rates of 3% and 7%. EPA uses the best available information from utilities, financial institutions, debt rating agencies, and government statistics as the basis for the capital charge rates and the discount rate used for power sector modeling in IPM.

More detail on IPM can be found in the model documentation, which provides additional information on the assumptions discussed here as well as all other assumptions and inputs to the

model (http://www.epa.gov/airmarkets/progsregs/epa-ipm). Updates specific to Toxics Rule modeling are also in the "Documentation Supplement for EPA Base Case v.4.10_PTox – Updates for Proposed Toxics Rule."

8.2 Projected Emissions

The proposed Toxics Rule is anticipated to achieve substantial emissions reductions. Since the technologies available to meet the emission reduction requirements of the rule reduce multiple air pollutants, EPA expects the proposed Toxics Rule to yield a broad array of pollutant reductions from the power sector. The primary pollutants of concern under the proposed Toxics Rule from the power sector are mercury, acid gases such as hydrogen chloride (HCl), and HAP metals, including antimony, arsenic, beryllium, cadmium, chromium, cobalt, mercury, manganese, nickel, lead, and selenium. EPA has extensively analyzed mercury emissions from the power sector, and IPM modeling assesses the mercury contents in all coals and the removal efficiencies of relevant emission control technologies (e.g., ACI). For the proposed Toxics Rule, EPA has included the ability to model emissions and the pollution control technologies associated with HCl (a proposed surrogate for acid gas emissions). Like SO_2, HCl is removed by both scrubbers and DSI, a control technology included in this updated version of the model. In addition to a better representation of the pollution controls available to reduce HCl in IPM, the detailed coal supply curves used in the model have been updated to reflect the chlorine content of coals, which corresponds with the supply region, coal grade, and sulfur, mercury, and ash content of each coal type. This information is critical for accurately projecting future HCl emissions, and for understanding how the power sector will respond to a policy requiring reductions of multiple HAPs.

Generally, existing pollution control technologies reduce emissions across a range of pollutants. For example, both FGD and SCR can achieve notable reductions in mercury in addition to their primary targets of SO_2 and NO_X reductions. DSI will reduce HCl emissions while also yielding substantial SO_2 emission reductions. Since there are many avenues to reduce emissions, and because the power sector is a highly complex and dynamic industry, EPA employs IPM in order to reflect the relevant components of the power sector accurately, while also providing a sophisticated view of how the industry could respond to particular policies to reduce emissions. For more detail on how EPA models emissions from the power sector, including recent updates to include acid gases, see "Documentation Supplement for EPA Base Case v.4.10_PTox – Updates for Proposed Toxics Rule."

Under the proposed Toxics Rule, EPA projects annual HCl emissions reductions of 87 percent in 2015, SO_2 emission reductions of 53 percent, and annual NO_x emissions reductions of 7 percent from the power sector by 2015, relative to the base case. In addition, EPA projects Hg emissions to be reduced by 67 percent relative to the base case (see Table 8-4). Mercury emission projections in EPA's base case are affected by the incidental capture of mercury in other pollution control technologies (such as FGD and SCR) as described above. The emission rate limitations for mercury in the proposed Toxics Rule would be the Clean Air Act's legal constraint on all possible mercury air emissions that could occur from the fuel combusted for power generation at the affected sources. In this analysis, the mercury content of all of the coal burned at affected units in the base case would yield 75 tons of mercury emissions if no emissions were subsequently captured. From this perspective, the emission rate limitations in the proposed Toxics Rule would assure a 91% reduction in air emissions of mercury from the coal-fired units subject to the policy.

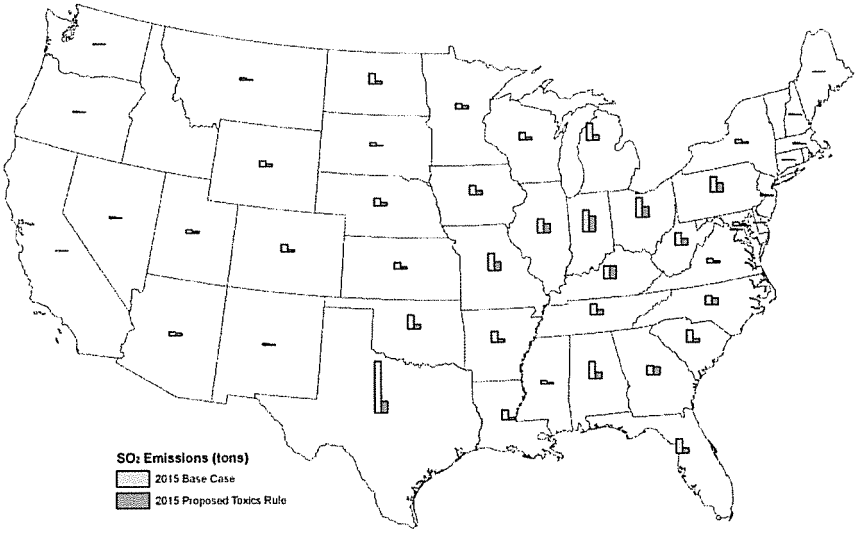
Table 8-4. Projected Emissions of SO_2, NO_X, Mercury, Hydrogen Chloride, CO_2, and PM with the Base Case and with the Proposed Toxics Rule, 2015

		SO_2 (million tons)	NO_X (million tons)	Mercury (tons)	HCl (thousand tons)	$PM_{2.5}$ (thousand tons)	CO_2 (million metric tonness)
Base	All EGUs	3.9	2.0	26.7*	77.8	285.5	2,243
	Coal > 25 MW	3.8	1.8	24.4*	74.4	277.0	1,928
Toxics Rule	All EGUs	1.8	1.9	8.7	10.2	202.3	2,219
	Coal > 25 MW	1.7	1.6	6.4	6.6	193.0	1,873

*Note: For the purposes of the RIA, EPA modeled a case that included state mercury-specific regulations and voluntary ACI, which underestimates potential base case mercury emissions by an estimated 4.7 tons because EPA cannot rely on those mercury reductions to be permanent. As a result of modeling this optimistic scenario, EPA has underestimated both costs and benefits; however, EPA does not expect that net benefits are likely to change significantly on the basis of these reductions.

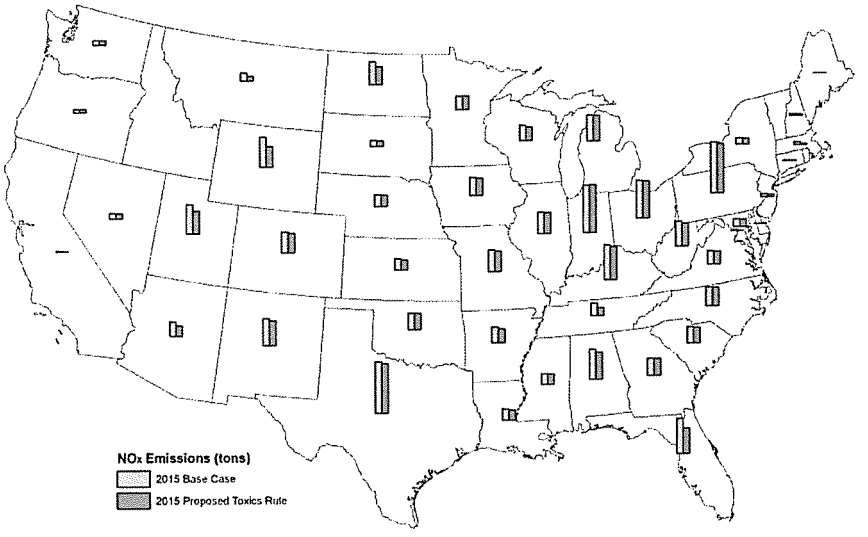
Source: Integrated Planning Model run by EPA, 2011

Figure 8-2. SO_2 Emissions from the Power Sector in 2015 with and without the Toxics Rule



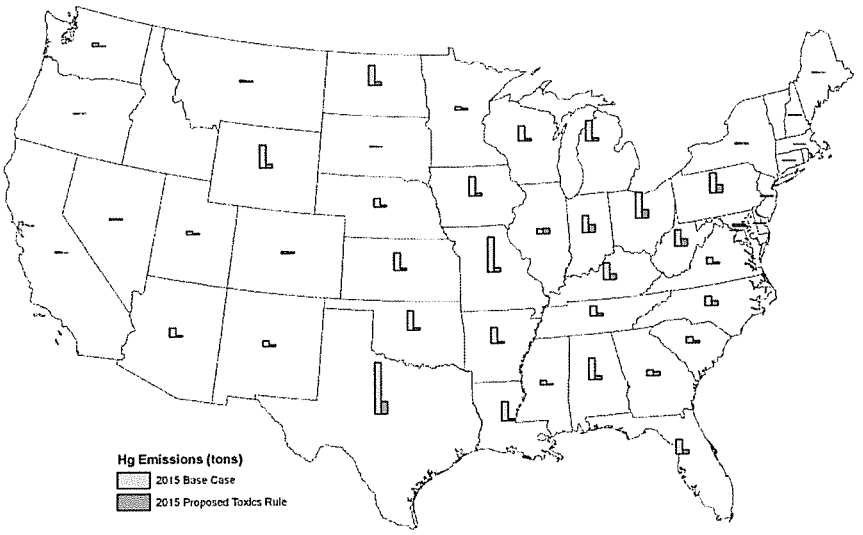
Source: 2015 emissions include coal steam (including IGCC and petroleum coke) or oil steam units >25 MW from IPM v4.10 base case and control case projections (EPA, February 2011)

Figure 8-3. NO_X Emissions from the Power Sector in 2015 with and without the Toxics Rule



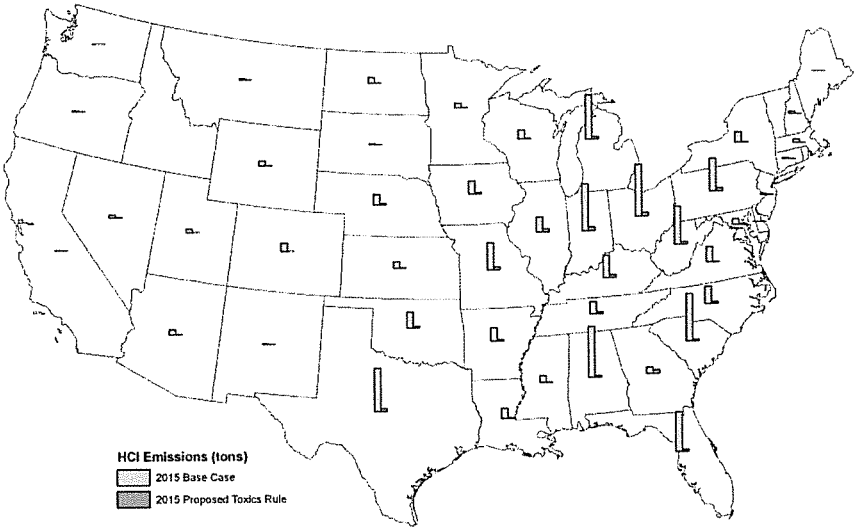
Source: 2015 emissions include coal steam (including IGCC and petroleum coke) or oil steam units >25 MW from IPM v4.10 base case and control case projections (EPA, February 2011)

Figure 8-4. Mercury Emissions from the Power Sector in 2015 with and without the Toxics Rule



Source: 2015 emissions include coal steam (including IGCC and petroleum coke) or oil steam units >25 MW from IPM v4.10 base case and control case projections (EPA, February 2011)

Figure 8-5. Hydrogen Chloride Emissions from the Power Sector in 2015 with and without the Toxics Rule



Source: 2015 emissions include coal steam (including IGCC and petroleum coke) or oil steam units >25 MW from IPM v4.10 base case and control case projections (EPA, February 2011)

8.3 Projected Compliance Costs

The power industry's "compliance costs" are represented in this analysis as the change in electric power generation costs between the base case and policy case in which the sector pursues pollution control approaches to meet the proposed Toxics Rule HAP emission standards. In simple terms, these costs are the resource costs of what the power industry will directly expend to comply with EPA's requirements.

EPA projects that the annual incremental compliance cost of the proposed Toxics Rule is $10.9 billion in 2015 ($2007). The annual incremental cost is the projected additional cost of complying with the proposed rule in the year analyzed, and includes the amortized cost of capital investment and the ongoing costs of operating additional pollution controls, needed new capacity, shifts between or amongst various fuels, and other actions associated with compliance.

Table 8-5. Annualized Compliance Cost for the Proposed Toxics Rule for Coal-fired Generation

	2015	2020	2030
Annualized Compliance Cost (billions of 2007$)	$10.9	$10.1	$10.0

Source: Integrated Planning Model run by EPA, 2011.

EPA's projection of $10.9 billion in additional costs in 2015 should be put into context for power sector operations. As shown in section 7.6, the power sector is expected in the base case to expend over $320 billion in 2015 to generate, transmit, and distribute electricity to end-use consumers. Therefore, the projected costs of compliance with the Toxics Rule amount to less than a 3.5% increase in the cost to meet electricity demand, while securing public health benefits that are several times more valuable (as described in Chapter 5). EPA plans to estimate the social cost for the final rule using a general equilibrium approach that incorporates the effect on electricity price change through the economy.

8.4 Projected Compliance Actions for Emissions Reductions

Fossil fuel-fired electric generating units are projected to achieve HAP emission reductions through a combination of compliance options. These actions include improved operation of existing controls, additional pollution control installations, coal switching (including blending of coals), and generation shifts towards more efficient units and lower-emitting generation technologies (e.g., some reduction of coal-fired generation with an increase of

generation from natural gas). In addition, there will be some affected sources that find it uneconomic to invest in new pollution control equipment and will be removed from service. These facilities are generally amongst the oldest and least efficient power plants, and typically run infrequently. In order to ensure that any retirements resulting from the proposed Toxics Rule do not adversely impact the ability of affected sources and electric utilities from meeting the demand for electricity, EPA has conducted an analysis of the impacts of projected retirements on electric reliability. This analysis is discussed in TSD titled: "Resource Adequacy and Reliability in the IPM Projections for the Toxics Rule" which is available in the docket.

The requirements under the proposed Toxics Rule are largely met through the installation of pollution controls (see Figure 8-6). To a lesser extent, there is a small degree of shifting within and across various ranks and types of coals, and a relatively small shift from coal-fired generation to greater use of natural gas and non-emitting sources of electricity (e.g., renewables and nuclear) (see Table 8-6). The largest share of emissions reductions occur from coal-fired units installing new pollution control devices, such as FGD, ACI, and fabric filters; a smaller share of emission reductions come from fuel shifts and unit retirements. Mercury emission reductions are largely driven by SCR/FGD combinations and ACI installations. HCl emission reductions are largely driven by FGD and DSI installations, which also incidentally provide substantial SO_2 reductions in the policy case. Mercury, $PM_{2.5}$, and HCl emission reductions are also facilitated by the installation of fabric filters, which boost mercury and HCl removal efficiencies of ACI and DSI, respectively.

As shown in Figure 8-6, this analysis projects that by 2015, the proposed Toxics Rule will drive the installation of an additional 24 GW of FGD (scrubbers), 56 GW of DSI, 93 GW of additional ACI, and 3 GW of SCR. Additionally, EPA is assuming for the purposes of this analysis that a subset of all covered coal-fired EGUs will require a fabric filter in order to meet the total PM standard. This assumption results in an additional 49 GW of fabric filter retrofits, for a total of 165 GW by 2015. For more information, see section 8.14.

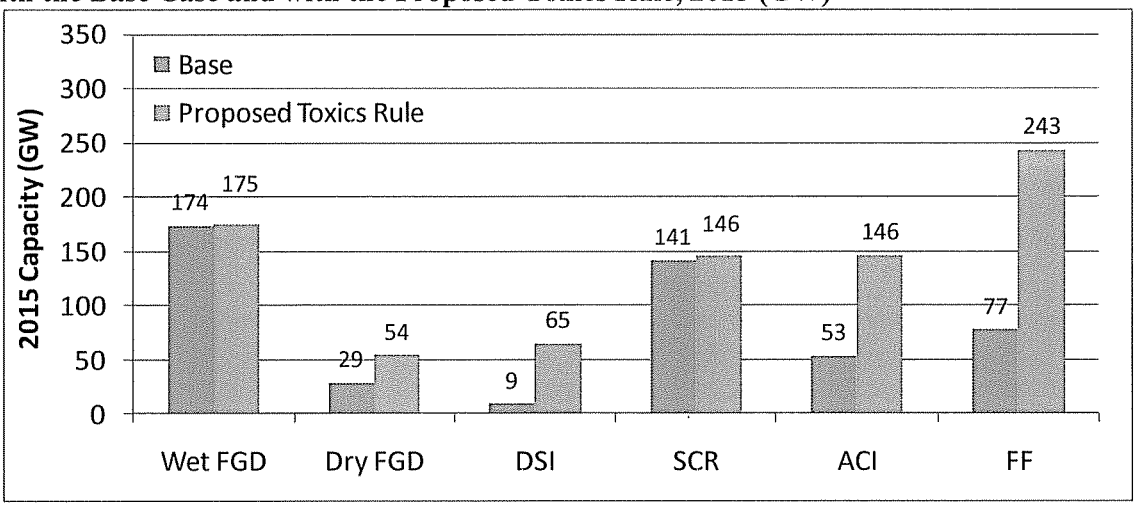
Table 8-6 below provides the estimated compliance costs broken down by control technology. The total costs, plus the estimated additional fuel costs totaling $2.9 billion, account for the estimated $10.9 billion annual compliance cost in 2015.

Table 8-6. Capital, FOM, and VOM Costs by Control Technology for the Proposed Toxics Rule (millions of 2007$)

	Dry FGD + FF	DSI	FF	ACI	FGD Upgrade	Waste Coal FGD	Total
Capital	1,421	428	1,092	1,498	669	94	5,201
FOM	252	71	41	48	0	20	431
VOM	377	1,241	105	627	0	66	2,416
2015 Annual Capital+FOM+VOM	2,050	1,740	1,238	2,173	669	179	8,048

Source: Integrated Planning Model run by EPA, 2011.

Figure 8-6. Retrofit Pollution Control Installations on Coal-fired Capacity (by Technology) with the Base Case and with the Proposed Toxics Rule, 2015 (GW)



Note: The difference between controlled capacity in the base case and under the proposed Toxics Rule may not necessarily equal new retrofit construction, since controlled capacity above reflects incremental operation of dispatchable controls in 2015. For this reason, and due to rounding, numbers in the text above may not reflect the increments displayed in this figure. See IPM Documentation for more information on dispatchable controls.

Source: Integrated Planning Model run by EPA, 2011.

8.5 Projected Generation Mix

Table 8-7and Figure 8-7 show the generation mix in the base case and in the proposed Toxics Rule policy case. In 2015, coal-fired generation is projected to decline slightly and natural-gas-fired generation is projected to increase slightly relative to the base case. Coal-fired generation is projected to increase above 2009 actual levels. The vast majority (over 95%) of base case coal capacity is projected to remain in service under the proposed Toxics Rule. In

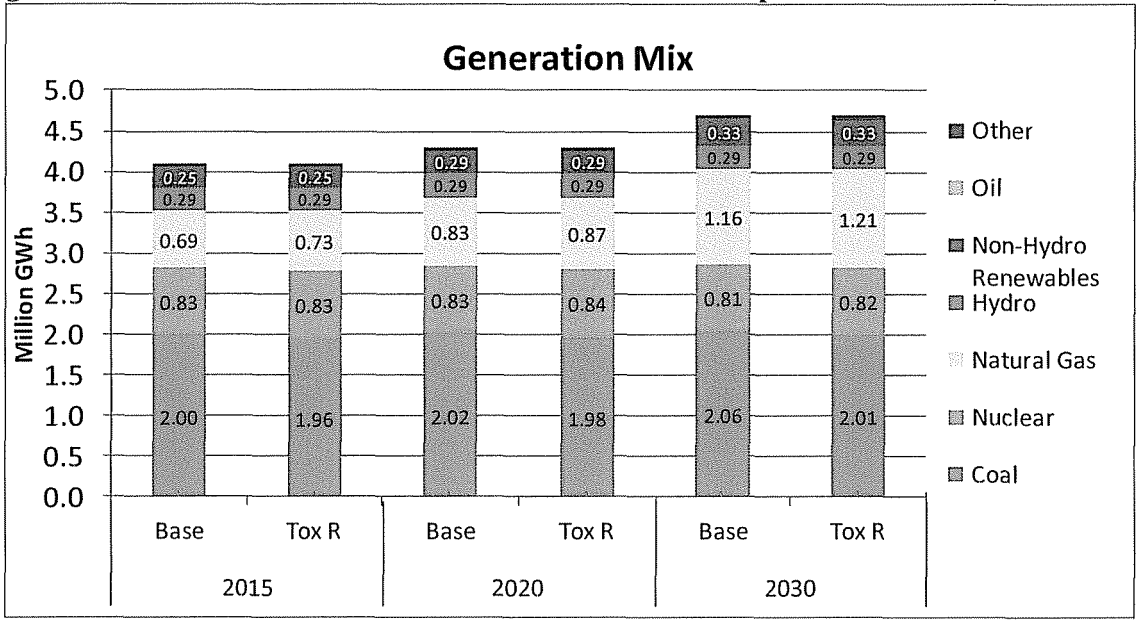
addition, the operating costs of complying coal-fired units are not so affected as to result in major changes in the electricity generation mix.

Table 8-7. Generation Mix with the Base Case and the Proposed Toxics Rule, 2015 (Thousand GWh)

	2009	2015			
	Historical	Base Case	Policy Case	Change from Base	Percent Change
Coal	1,756	2,002	1,961	-41	-2.0%
Oil	39	0.11	0.11	0.01	8.6%
Natural Gas	921	694	730	36	5.2%
Nuclear	799	825	831	6	0.7%
Hydroelectric	273	286	288	2	0.7%
Non-hydro Renewables	144	251	250	-1	-0.5%
Other	18	45	46	0.5	1.1%
Total	**3,950**	**4,104**	**4,106**	**2**	**0.1%**

Note: Numbers may not add due to rounding.
Source: 2009 data from EIA Electric Power Annual 2009, Table 2.1; 2015 projections are from the Integrated Planning Model run by EPA, 2011.

Figure 8-7. Generation Mix with the Base Case and with Proposed Toxics Rule, 2015-2030



Source: Integrated Planning Model run by EPA, 2011.

8.6 Projected Retirements

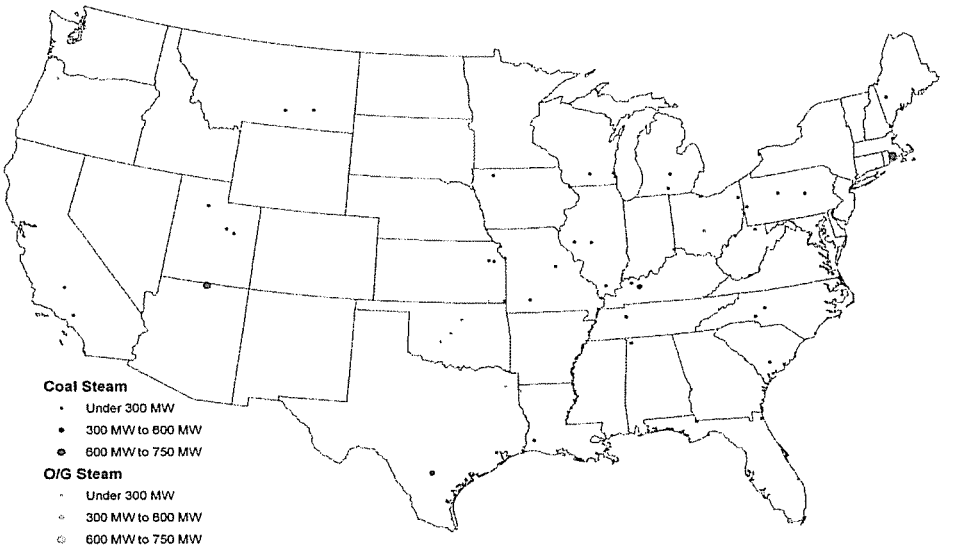
Relative to the base case, about 9.9 GW of coal-fired capacity is projected to be uneconomic to maintain (roughly 3 percent of all coal-fired capacity) by 2015. Uneconomic units, for the most part, are older, smaller, and less frequently used generating units that are dispersed throughout the country (see Table 8-7 and Figure 8-8). For the proposed Toxics Rule, EPA has examined whether these closures may adversely impact reserve margins and reliability planning. The IPM model is specifically designed to ensure that generation resource availability is maintained in the projected results subject to reserve margins in 32 modeling regions for the contiguous US, which must be preserved either by using existing resources or through the construction of new resources. IPM also addresses reliable delivery of generation resources by limiting the ability to transfer power between regions using the bulk power transmission system. Within each model region, IPM assumes that adequate transmission capacity is available to deliver any resources located in, or transferred to, the region. The IPM model projects available capacity given certain constraints such as reserve margins and transmission capability but does not constitute a detailed reliability analysis. For example, the IPM model does not examine frequency response. For more detail on IPM's electric load modeling and power system operation, please see IPM documentation (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html) and the TSD on Resource Adequacy and Reliability in the IPM Projections for the Toxics Rule.

Table 8-8. Characteristics of Incremental Coal Retirements and Operational Units in Proposed Toxics Rule, 2015

	Average Age (years)	Average Capacity (MW)	Average Capacity Factor in Base
Retired Units	51	109	56%
Operational Units	44	278	71%

Source: Integrated Planning Model run by EPA, 2011.

Figure 8-8. Geographic Distribution of Incremental Retirements from Proposed Toxics Rule, 2015



Total operational capacity is lower in the policy scenario, primarily as a result of increases in coal retirements. Since most regions are projected to have excess capacity above their target reserve margins, most of these retirements are absorbed by a reduction in excess reserves. Operational capacity changes from the base case in 2015 are shown in Table 8-9.

Table 8-9. Total Generation Capacity by 2015 (GW)

	2010	Base Case	Toxics Rule
Pulverized Coal	317	309	299
Natural Gas Combined Cycle	201	272	280
Other Oil/Gas	253	236	236
Non-Hydro Renewables	31	78	79
Hydro	99	99	99
Nuclear	102	102	103
Other	5	7	7
Total	1,009	1,104	1,103

Source: 2010 data from EPA's NEEDS v.4.10_PTox. Projections from Integrated Planning Model run by EPA.
Note: "Non-Hydro Renewables" include biomass, geothermal, solar, and wind electric generation capacity. 2015 capacity reflects plant closures planned to occur prior to 2015.

The policy case analyzed maintains resource adequacy in each region experiencing coal unit retirements by using excess reserve capacity within the region, reversing base case retirements of non-coal capacity, building new capacity, or by importing excess reserve capacity from other regions. Although any closure of a large generation facility will need to be studied to determine potential local reliability concerns, EPA analysis suggests that projected retirements

under the proposed Toxics Rule could have little to no overall impact on electric reliability. Not only are projected retirements under the proposed Toxics Rule limited in scope, but the existing state of the power sector is also characterized by substantial excess capacity. The weighted average reserve margin at the national level is projected to be approximately 25% in the base case, while the North American Electric Reliability Corporation (NERC) recommends a margin of 15%. EPA projects that the proposed Toxics Rule would only reduce total operational capacity by less than one percent in 2015.

Moreover, projected coal retirements are distributed throughout the power grid with limited effect at the regional level, such that any potential impacts should not adversely affect reserve margins and should be manageable through the normal industry processes. For example, the coal-fired generating areas in western Pennsylvania, West Virginia, Ohio, and Indiana all have significant excess generation resources: these areas combined see a decrease of less than 2% in their reserve margins in the policy case and retain an overall reserve margin of over 20%. Furthermore, subregions may share each other's excess reserves to ensure adequate reserve margins within a larger reliability region. EPA's IPM modeling accommodates such transfers of reserves within the assumed limits of reliability of the inter-regional bulk power system. For these reasons, the projected closures of coal plants are not expected to raise broad reliability concerns.

8.7 Projected Capacity Additions

Due in part to a low growth rate anticipated for future electricity demand levels in the latest EIA forecast, EPA analysis indicates that there is sufficient excess capacity through 2015 to compensate for capacity that is retired from service under the proposed Toxics Rule. In the short-term, most new capacity is projected as a mix of wind and natural gas in response to low fuel prices and other energy policies (such as tax credits and state renewable portfolio standards). In addition, future electricity demand expectations have trended downwards in recent forecasts, reducing the need for new capacity in the 2015 timeframe (see Chapter 7 for more discussion on future electricity demand).

Table 8-10. Total Generation Capacity by 2030 (GW)

	2010	Base Case	Toxics Rule	Change
Pulverized Coal	316	309	299	-10.0
Natural Gas Combined Cycle	201	272	280	7.6
Other Oil/Gas	253	236	236	0.4
Non-Hydro Renewables	31	78	79	0.8
Hydro	78	99	99	0.0
Nuclear	102	102	103	0.8
Other	26	7	7	0.0
Total	1,009	1,104	1,103	-0.5

Source: 2010 data from EPA's NEEDS v.4.10_PTox. Projections from Integrated Planning Model run by EPA.
Note: "Non-Hydro Renewables" include biomass, geothermal, solar, and wind electric generation capacity.

8.8 Projected Coal Production for the Electric Power Sector

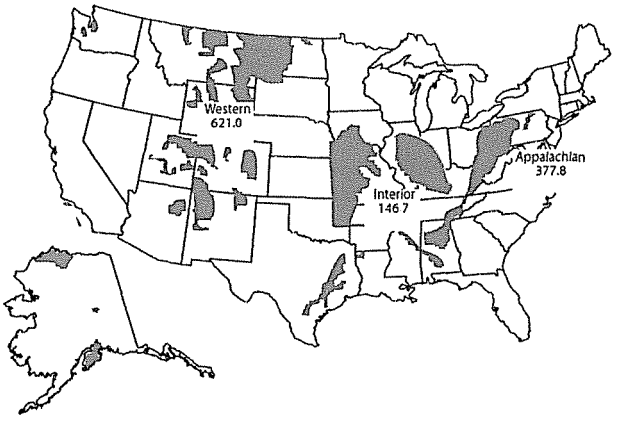
Coal production for electricity generation under the proposed Toxics Rule is expected to decline modestly relative to the base case without the rule. The reductions in emissions from the power sector will be met through the installation and operation of pollution controls for HAP removal. Many available pollution controls achieve emissions removal rates of up to 99 percent (e.g., HCl removal by new scrubbers), which allows industry to rely more heavily on local bituminous coal in the eastern and central parts of the country that has higher contents of HCl and sulfur, and it is less expensive to transport than western subbituminous coal. Generally, the demand for bituminous coals increases under the proposed rule, while demand for subbituminous and lignite coals is reduced slightly (see Tables 8-10 and 8-11). The trend reflects the reduced demand for lower-sulfur coal under the proposed Toxics Rule, where nearly all units are operating with a post-combustion emissions control. In this case, because of the additional pollution controls, many of these units no longer find it economic to pay a transportation premium to purchase lower-sulfur subbituminous coals. Instead, EGUs are generally shifting consumption towards nearby bituminous coal, which can achieve low emissions when combined with post-combustion emissions controls. This explains the increase in coal supplied from the Interior region, which is located in relatively close proximity to many coal-fired generators subject to this proposed rule. The decline in lignite use reflects a decrease in generation from lignite-fired boilers, as well as a general shift toward subbituminous for boilers which were burning lignite coal in the base case.

Table 8-11. 2015 Coal Production for the Electric Power Sector with the Base Case and the Proposed Toxics Rule (Million Tons)

Supply Area	2009	2015 Base	2015 Toxics Rule	Change in 2015
Appalachia	246	183	168	-8%
Interior	129	227	233	2%
West	553	551	543	-2%
Waste Coal	14	14	13	-5%
Imports		30	30	0%
Total	942	1,006	987	-2%

Source: Source: Production: U.S. Energy Information Administration (EIA), *Coal Distribution -- Annual (Final)*, web site http://www.eia.doe.gov/cneaf/coal/page/coaldistrib/a_distributions.html (posted February 18, 2011); Waste Coal: U.S. EIA, *Monthly Energy Review, January 2011 Edition,* Table 6.1 Coal Overview, web site http://www.eia.doe.gov/emeu/mer/coal.html (posted January 31, 2011). All projections from Integrated Planning Model run by EPA, 2011.

Figure 8-9. Total Coal Production by Coal-Producing Region, 2007 (Million Short Tons)



Note: Regional totals do not include refuse recovery

Source: EIA Annual Coal Report, 2007

Table 8-12. 2015 Power Sector Coal Use with the Base Case and the Proposed Toxics Rule, by Coal Rank (TBtu)

Coal Rank	Base	Toxics Rule	Change
Bituminous	11,450	11,628	2%
Subbituminous	7,762	7,668	-1%
Lignite	904	609	-33%
Total	20,116	19,905	-1%

Source: Integrated Planning Model run by EPA, 2011.

8.9 Projected Retail Electricity Prices

EPA's analysis projects a near-term increase in the average retail electricity price of 3.7% in 2015 falling to 2.6% by 2020 under the proposed Toxics Rule in the contiguous U.S. The projected price impacts vary by region and are provided in Table 8-13 (see Figure 8-10 for regional classifications).

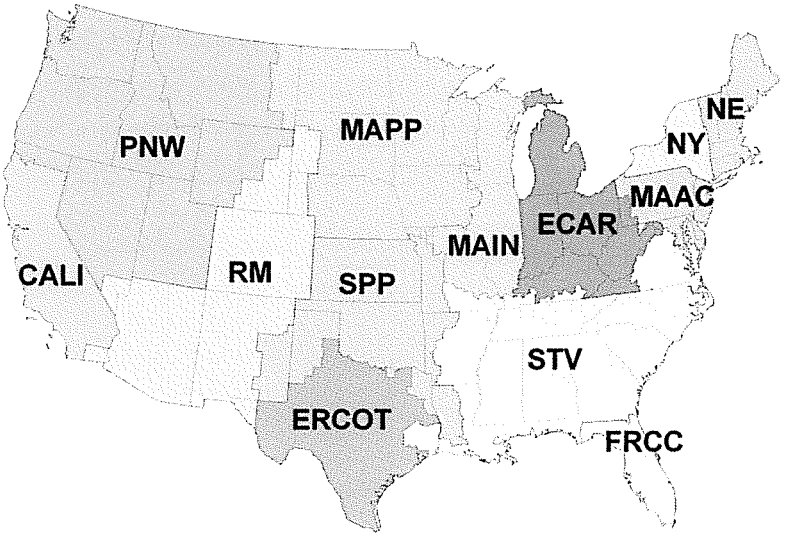
Table 8-13. Projected Contiguous U.S. and Regional Retail Electricity Prices with the Base Case and with the Proposed Toxics Rule (2007 cents/kWh)

	Base Case			Proposed Toxics Rule			Percent Change		
	2015	2020	2030	2015	2020	2030	2015	2020	2030
ECAR	8.1	8.2	9.6	8.5	8.5	9.9	5.5%	4.0%	3.4%
ERCOT	8.9	8.7	11.3	9.3	8.8	11.3	5.3%	1.5%	0.2%
MAAC	9.5	10.3	12.7	9.8	10.4	12.7	3.2%	0.5%	0.4%
MAIN	8.0	8.4	9.7	8.3	8.6	10.0	4.0%	2.7%	3.2%
MAPP	8.0	7.9	8.6	8.5	8.3	8.9	5.4%	5.6%	4.5%
NY	13.7	13.3	16.5	14.1	13.4	16.5	2.6%	0.8%	0.4%
NE	12.3	11.8	13.8	12.7	12.4	14.1	2.8%	5.2%	2.2%
FRCC	10.2	9.7	11.0	10.5	9.9	11.1	2.8%	2.1%	1.0%
STV	7.9	7.8	8.4	8.2	8.0	8.6	3.6%	2.8%	2.3%
SPP	7.7	7.4	8.0	8.2	7.8	8.5	7.1%	6.4%	5.5%
PNW	7.1	6.8	7.3	7.3	7.0	7.4	2.5%	2.1%	1.6%
RM	9.2	9.4	10.9	9.4	9.6	11.0	2.3%	2.0%	1.1%
CALI	13.0	12.5	12.5	13.2	12.6	12.6	1.4%	0.7%	0.1%
Contiguous U.S. Average	9.0	8.9	10.2	9.3	9.2	10.4	3.7%	2.6%	1.9%

Source: EPA's Retail Electricity Price Model, 2011.

Regional retail electricity prices are projected to range from 1 to 7 percent higher with the proposed Toxics Rule in 2015. The extent of regional retail electricity increases correlates with states that have considerable coal-fired generation that is less well-controlled (such as in the ECAR, MAAC, and SPP regions). EPA has not presented an analysis of the economy-wide impacts from projected electricity price changes but will consider doing so in the final rule.

Figure 8-10. Retail Price Model Regions



8.10 Projected Fuel Price Impacts

The impacts of the proposed Toxics Rule on coal and natural gas prices before shipment are shown below in Tables 8-14 and 8-15. Overall, average coal price changes are related to changes in demand for a wide variety of coals based upon a number of parameters (e.g., chlorine or mercury content, heat content, proximity to the power plant, etc.). Generally, the demand for bituminous coals increases under the proposed rule, while demand for subbituminous and lignite coals is reduced slightly. This is reflected in the projected average minemouth price of coal, which goes up slightly (about 1 percent) even though total demand for coal is reduced slightly (1 percent reduction). Notwithstanding the projected "mine-mouth" coal price changes, many units may in fact be realizing overall fuel cost savings by switching to more local coal supplies (which reduces transportation costs) after installing additional pollution control equipment. Gas price changes are directly related the projected increase in natural gas consumption under the proposed rule. This increase in demand is met by producing additional natural gas at some increase in regional costs, resulting over time in a small price increase.

IPM modeling of natural gas prices uses both short- and long-term price signals to balance supply of and demand in competitive markets for the fuel across the modeled time horizon. As such, it should be understood that the pattern of IPM natural gas price projections over time is not a forecast of natural gas prices incurred by *end-use consumers* at any particular point in time. The natural gas market in the United States has historically experienced significant price volatility from year to year, between seasons within a year, and even sees major price swings during short-lived weather events (such as cold snaps leading to short-run spikes in

heating demand). These short-term price signals are fundamental for allowing the market to successfully align immediate supply and demand needs; however, end-use consumers are typically shielded from experiencing these rapid fluctuations in natural gas prices by retail rate regulation and by hedging through longer-term fuel supply contracts. IPM assumes these longer-term price arrangements take place "outside of the model" and on top of the "real-time" shorter-term price variation necessary to align supply and demand. Therefore, the model's natural gas price projections should not be mistaken for traditionally experienced consumer price impacts related to natural gas, but a reflection of expected average price changes over the time period 2015 to 2030.

For this analysis, in order to represent a natural gas price evolution that end-use consumers can anticipate under retail rate regulation and/or typical hedging behavior, EPA is displaying the weighted average of IPM's natural gas price projections for the 2015-2030 time horizon (see Table 8-15). In that framework, consumer natural gas price impacts are anticipated to range from 0.6% to 1.3% based on consumer class in response to the proposed Toxics Rule. EPA has not presented an analysis of the economy-wide impacts from projected fuel price changes but will consider doing so in the final rule.

Table 8-14. Average Minemouth and Delivered Coal Prices with the Base Case and with the Proposed Toxics Rule (2007$/MMBtu)

		2015			2030		
	2007	Base Case	Policy Case	Percent Change from Base	Base Case	Policy Case	Percent Change from Base
Minemouth	1.27	1.36	1.38	0.9%	1.53	1.58	3.1%
Delivered	1.76	2.12	2.13	0.5%	2.31	2.34	1.3%

Source: Historical data from EIA AEO 2010 Reference Case Table 15 (Coal Supply, Distribution, and Prices); projections from the Integrated Planning Model run by EPA, 2011.

Table 8-15. 2015-2030 Weighted Average Henry Hub (spot) and Delivered Natural Gas Prices with the Base Case and with the Proposed Toxics Rule (2007$/MMBtu)

	Base Case	Policy Case	Percent Change from Base
Henry Hub	5.28	5.35	1.3%
Delivered - Electric Power	5.55	5.62	1.3%
Delivered - Residential	10.93	11.00	0.6%

Source: Projections from the Integrated Planning Model run by EPA (2011) adjusted to Henry Hub prices using historical data from EIA AEO 2011 reference case to derive residential prices.

8.11 Key Differences in EPA Model Runs for the Toxics Rule Modeling

The 2015 base case EGU emissions projections of mercury, hydrogen chloride, SO_2, and PM were obtained from an interim version 4.10 of the Integrated Planning Model (IPM) (http://www.epa.gov/airmarkt/progsregs/epa-ipm/index.html). The IPM is a multiregional, dynamic, deterministic linear programming model of the U.S. electric power sector. Version 4.10 reflects state rules and consent decrees through December 1, 2010, and incorporates information on existing controls collected through the Utility MACT ICR. Units with SO_2 or NO_X advanced controls (e.g., scrubber, SCR) that were not required to run for compliance with Title IV, New Source Review (NSR), state settlements, or state-specific rules were allowed in IPM to decide on the basis of economic efficiency whether to operate those controls. Units with advanced mercury controls (e.g., ACI) were assumed to operate those controls in states with mercury requirements. Note that this base case includes the proposed Transport Rule, which will be finalized in June, 2011. Further details on the EGU emissions inventory used for this proposal can be found in the IPM Documentation.

The length of time required to conduct emissions and photochemical modeling precluded the use of IPM version 4.10_PTox (Proposed Toxics Rule). Thus the air quality modeling for the Toxics Rule relied on EGU emission projections from an interim IPM platform that was subsequently updated during the rulemaking process for the scenario summarized in this chapter. The IPM update reflects additional information obtained primarily from the 2010 ICR and from comments submitted on an IPM Notice of Data Availability (NODA) in October 2010. Notably, this IPM update included the addition of over 20 GW of existing ACI reported to EPA via the ICR, which explains the majority of the difference in interim and final base case EGU mercury projections. This update also includes additional unit-level updates that were made based on the ICR and public comments on the IPM NODA which identified additional existing pollution controls which affect base case projections of multiple pollutants, including mercury emissions

incidentally captured through operation of post-combustion controls (such as scrubbers). Additionally, the IPM update corrected an erroneous natural gas $PM_{2.5}$ emission factor from the interim platform which overestimated EGU $PM_{2.5}$ emissions by about 85,000 tons. Other updates includes adjustments to assumptions regarding the cost and performance of acid gas control technologies, a correction to mercury removal from new fabric filters, new costs imposed on fuel-switching (e.g., bituminous to subbituminous), correction of lignite availability to some plants, incorporation of additional planned retirements, a more inclusive implementation of the scrubber upgrade option, and the availability of a scrubber retrofit to waste coal-fired fluidized bed combustion units without an existing scrubber.

The interim policy case modeling of EGU emissions for air quality modeling, presented in Chapter 3, was conducted before EPA completed a comprehensive review of ICR data to inform the proposed Toxics Rule emissions limits. This interim policy case reflected more stringent HCl and mercury emission reduction requirements than are being proposed in today's action.

8.12 Projected Primary PM Emissions from Power Plants

IPM does not endogenously model primary PM emissions from power plants. These emissions are calculated as a function of IPM outputs, emission factors and control configuration. IPM-projected fuel use (heat input) is multiplied by PM emission factors (based in part on the presence of PM-relevant pollution control devices) to determine PM emissions. Primary PM emissions are calculated by adding the filterable PM and condensable PM emissions.

Filterable PM emissions for each unit are based on historical information regarding existing emissions controls and types of fuel burned and ash content of the fuel burned, as well as the projected emission controls (e.g., scrubbers and fabric filters).

Condensable PM emissions are based on plant type, sulfur content of the fuel, and SO_2/HCl and PM control configurations. Although EPA's analysis is based on the best available emission factors, these emission factors do not account for the potential changes in condensable PM emissions due to the installation and operation of SCRs. The formation of additional condensable PM (in the form of SO_3 and H_2SO_4) in units with SCRs depends on a number of factors, including coal sulfur content, combustion conditions and characteristics of the catalyst used in the SCR, and is likely to vary widely from unit to unit. SCRs are generally designed and operated to minimize increases in condensable PM. This limitation means that IPM post-processing is potentially underestimating condensable PM emissions for units with SCRs. In

contrast, it is possible that IPM post-processing overestimates condensable PM emissions in a case where the unit is combusting a low-sulfur coal in the presence of a scrubber.

EPA plans to continue improving and updating the PM emission factors and calculation methodologies. For a more complete description of the methodologies used to post-process PM emissions from IPM, see "IPM ORL File Generation Methodology" (March, 2011).

8.13 Illustrative End-use Energy Efficiency Policy Sensitivity

To explore the possible impacts of this rule under an alternative baseline with increased federal and state energy efficiency policies and resultant lower levels of electricity generation, EPA developed an end-use energy efficiency policy scenario and analyzed the associated effects. By reducing electricity demand, energy efficiency avoids emissions of all pollutants associated with electricity generation, including emissions of toxic air pollutants targeted by this rule. This "energy efficiency sensitivity" illustrates a possible alternative future where use of energy efficiency policies lead to increased investment in cost-effective energy end-use technologies beyond what is reflected in the reference electricity demand forecast used for EPA's core analysis (i.e., the analysis described in the preceding subsections of this chapter). This sensitivity does not represent an EPA forecast of electricity demand.

EPA based the energy efficiency sensitivity on two policies: implementation of federal appliance standards for products required under existing statutes and the possible increased use of ratepayer-funded energy efficiency programs consistent with recent state policy trends. The projected electricity demand impacts of the federal appliance standards were provided by the U.S. Department of Energy's (DOE) Office of Energy Efficiency and Renewable Energy and are an estimate of the incremental effects, relative to the AEO 2010 reference case forecast, of DOE's statutorily mandated appliance standards rulemakings (appliance standards that have been implemented are in the base case). The projected electricity demand impacts of the increased use of ratepayer-funded energy efficiency programs are derived from a 2009 analysis by the Lawrence Berkeley National Lab (LBNL).[1] For both of these energy efficiency policies (federal appliance standards and ratepayer-funded energy efficiency), EPA assumed the continued use of the policies at similar levels of effectiveness, and estimated the associated impacts, through 2050 (the final year for EPA's regulatory analysis.

After developing the basis for the energy efficiency sensitivity, EPA derived annual electricity demand impacts, estimated associated costs, and analyzed impacts on the electricity

[1] Lawrence Berkeley National Laboratory, "The Shifting Landscape of Ratepayer Funded Energy Efficiency in the U S ," (October 2009), Galen Barbose et al , LBNL-2258E, (http://eetd.lbl gov/ea/ems/reports/lbnl-2258e pdf).

generation sector. The total U.S. electricity demand reductions in 2015, 2020, 2030, 2040, and 2050, for the energy efficiency scenario represent 2.9%, 5.3%, 6.6%, 6.1% and 5.8% of U.S. electricity demand in those years, respectively. These reductions lower annual average electricity demand growth (from 2009 historic data) through 2020 from the reference forecast from 1.04% to 0.55%. Similarly, through 2030 the reduction is from 0.97% to 0.64%, and through 2050 the reduction is from 0.91% to 0.77%. These reductions in demand growth are substantially lower than recent estimates of available, cost-effective energy efficiency potential. Costs associated with the two policy strategies were estimated based upon historical studies specific to each policy. For more information on the construction of the EE sensitivity, see Appendix D.

EPA analyzed the impacts of the energy efficiency sensitivity on the electricity generation sector by conducting alternative scenarios using the Integrated Planning Model. The results from those modeling runs and the associated costs are summarized in Tables 8-16, 8-17, and 8-18.[1]

The effects of the Toxics Rule under the Energy Efficiency Scenario on total electricity generating costs of the power sector are shown below in Table 8-16. In this table we also see the projected costs in the Base and Toxics Rule Cases with and without energy efficiency. In this analysis, the costs of additional energy efficiency investments to ratepayers and consumers are treated as a component of the cost of generating electricity and are imbedded in the costs seen in this table. Under the Energy Efficiency Scenario, the incremental costs of the Toxics Rule are moderately reduced in 2015, 2020, and 2030, by $0.3 billion, $1.1 billion, and $0.8 billion, respectively. When comparing the Toxics Rule Case without energy efficiency to the Toxics Rule Case with energy efficiency, the analysis suggests that these energy efficiency policies could mitigate the cost of the Toxics Rule such that the overall system costs are reduced by $2.3 billion in 2015, $6.0 billion in 2020, and $11.4 billion in 2030.

[1] EPA's analysis may not capture the full spectrum of behavior effects associated with energy efficiency policies that can mitigate projected reductions in energy demand. These effects can include: "rebound effect" (increased use of energy efficient product as a result of perceived energy savings, increased consumption of other energy consuming products -- from disposable income freed up by energy savings, increased production that might occur as a result of cost savings from energy efficient technologies, or changes in energy efficient product utility that lead to increased use). This analysis does, however, account for many of these effects in the estimation of the energy demand reductions associated with the federal appliance standards for products.

Table 8-16. Electric System Generation & Energy Efficiency Costs (billions of 2007$)

	2015	2020	2030
Total Costs			
Base Case	$144.3	$155.2	$200.4
Base Case w/ Energy Efficiency (EE)	$142.3	$150.3	$189.8
Toxics Rule Case	$155.2	$165.3	$210.3
Toxics Rule Case w/ Energy Efficiency (EE)	$152.9	$159.3	$198.9
Incremental Costs			
Base to Base w/EE	-$2.0	-$4.9	-$10.6
Toxics Rule to Toxics Rule w/EE	-$2.3	-$6.0	-$11.4
Base to Toxics Rule	$10.9	$10.1	$10.0
Base with EE to Toxics Rule w/EE	$10.5	$9.0	$9.1
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-$0.3	-$1.1	-$0.8

Source: Integrated Planning Model run by EPA, 2011.

The effects of the Toxics Rule under the Energy Efficiency Scenario on retail electricity prices for the continental U.S. are shown in Table 8-17. In this table we also see the projected retail electricity prices in the Base and Toxics Rule Cases with and without energy efficiency. The costs of the energy efficiency investments that would be borne by ratepayers are included in these retail electricity prices for the energy efficiency cases. Under the Energy Efficiency Scenario, the incremental impacts of the Toxics Rule on retail electricity prices are to increase them in 2015, 2020, and 2030, by 0.36 cents/KWh, 0.26 cents/KWh , and 0.21 cents/KWh, respectively. When comparing the Toxics Rule Case without energy efficiency to the Toxics Rule Case with energy efficiency, the analysis suggests that these energy efficiency policies could mitigate the impacts of the Toxics Rule such that the retail electricity prices are reduced by 0.04 cents/KWh in 2015, 0.38 cents/KWh in 2020, and 0.42 cents/KWh in 2030.

Table 8-17. Projected Contiguous U.S. Electricity Prices Including Energy Efficiency Costs (2007 cents/kWh)

	2015	2020	2030
Base Case	9.01	8.94	10.16
Base Case w/ Energy Efficiency (EE)	8.95	8.54	9.72
Toxics Rule Case	9.35	9.17	10.35
Toxics Rule Case w/ Energy Efficiency (EE)	9.31	8.80	9.93
Incremental Price Changes			
Base to Base w/EE	-0.07	-0.40	-0.44
Toxics Rule to Toxics Rule w/EE	-0.04	-0.38	-0.42
Base to Toxics Rule	0.33	0.23	0.19
Base with EE to Toxics Rule w/EE	0.36	0.26	0.21
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.03	0.02	0.02

Source: Integrated Planning Model run by EPA, 2011.

The effects of the Toxics Rule under the Energy Efficiency Scenario on new capacity additions are shown in Table 8-18. Under the Energy Efficiency Scenario, the incremental impacts of the Toxics Rule on new capacity additions are to increase them in 2015, 2020, and 2030, by 0.1 GW, 0.2 GW, and 5.1 GW, respectively. When comparing the Toxics Rule Case without energy efficiency to the Toxics Rule Case with energy efficiency, the analysis suggests that these energy efficiency policies reduce the need for new capacity by 0.3 GW in 2015, 8.5 GW in 2020, and 39.8 GW in 2030.

Table 8-18. New Capacity Additions Including Energy Efficiency Cases (Cumulative GW)

	2015	2020	2030
Base Case	29.6	36.3	108.0
Base Case w/ Energy Efficiency (EE)	29.3	30.6	71.2
Toxics Rule Case	29.7	39.2	116.0
Toxics Rule Case w/ Energy Efficiency (EE)	29.4	30.8	76.3
Incremental Capacity Additions			
Base to Base w/EE	-0.4	-5.7	-36.9
Toxics Rule to Toxics Rule w/EE	-0.3	-8.5	-39.8
Base to Toxics Rule	0.1	3.0	8.0
Base with EE to Toxics Rule w/EE	0.1	0.2	5.1
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.1	-2.8	-2.9

Source: Integrated Planning Model run by EPA, 2011.

For a discussion of the approach taken to define and analyze the energy efficiency scenario, and an expanded set of IPM results for the scenario, including costs, energy prices, retirements, emission controls, and air emissions, see Appendix D.

8.14 Limitations of Analysis

EPA's modeling is based on expert judgment of various input assumptions for variables whose outcomes are in fact uncertain. Assumptions for future fuel supplies and electricity demand growth deserve particular attention because of the importance of these two key model inputs to the power sector. As a general matter, the Agency reviews the best available information from engineering studies of air pollution controls to support a reasonable modeling framework for analyzing the cost, emission changes, and other impacts of regulatory actions.

The annualized cost estimates of private compliance costs provided in this analysis are meant to show the increase in production (generating) costs to the power sector in response to the proposed Toxics Rule. To estimate these annualized costs, EPA uses a conventional and widely-accepted approach that applies a capital recovery factor (CRF) multiplier to capital investments and adds that to the annual incremental operating expenses. The CRF is derived from estimates of the cost of capital (private discount rate), the amount of insurance coverage required, local property taxes, and the life of capital. The private compliance costs presented earlier are EPA's best estimate of the direct private compliance costs of the Proposed Toxics Rule.

The annualized cost of the proposed Toxics Rule, as quantified here, is EPA's best assessment of the cost of implementing the rule. These costs are generated from rigorous economic modeling of changes in the power sector due to the proposed Toxics Rule. This type of analysis using IPM has undergone peer review, and federal courts have upheld regulations covering the power sector that have relied on IPM's cost analysis.

The direct private compliance cost includes, but is not limited to, capital investments in pollution controls, operating expenses of the pollution controls, investments in new generating sources, and additional fuel expenditures. EPA believes that the cost assumptions used for the proposed Toxics Rule reflect, as closely as possible, the best information available to the Agency today. The relatively small cost associated with monitoring emissions, reporting, and record keeping for affected sources is not included in these annualized cost estimates, but EPA has done a separate analysis and estimated the cost to be approximately $49 million annually (see Section 10.3, Paperwork Reduction Act).

Cost estimates for the proposed Toxics Rule are based on results from ICF's Integrated Planning Model. The model minimizes the costs of producing electricity (including abatement costs) while meeting load demand and other constraints (full documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm and in the "Documentation Supplement for EPA Base Case v.4.10_PTox – Updates for Proposed Toxics Rule.". IPM assumes "perfect foresight" of market conditions over the time horizon modeled; to the extent that utilities and/or energy regulators misjudge future conditions affecting the economics of pollution control, costs may be understated as well.

In the policy case modeling, EPA assumes that a subset of covered units might require a retrofit fabric filter (also known as a baghouse) in order to meet at least one of the proposed emissions standards. Based on ICR data and existing pollution controls, EPA estimates that approximately 54 GW of existing capacity without fabric filters may not require this retrofit for compliance with any of the proposed Toxics Rule emissions standards. It is possible that this assumption is conservative, and that more EGUs may be able to comply with the proposed Toxics Rule standards without constructing a new fabric filter.

Additionally, this modeling analysis does not take into account the potential for advancements in the capabilities of pollution control technologies as well as reductions in their costs over time. In addition, EPA modeling cannot anticipate in advance the full spectrum of compliance strategies that the power sector may innovate to achieve the required emission reductions under the proposed Toxics Rule, which would potentially reduce overall compliance

costs. Where possible, EPA designs regulations to assure environmental performance while preserving flexibility for affected sources to design their own solutions for compliance. Industry will employ an array of responses, some of which regulators may not fully anticipate and will generally lead to lower costs associated with the rule than modeled in this analysis. For example, unit operators may find opportunities to improve or upgrade existing pollution control equipment without requiring as many new retrofit devices (i.e., meeting the PM standard with an existing ESP without requiring installation of a new fabric filter). With that in mind, the Toxics Rule establishes emission rates on key HAPs, and although this analysis projects a specific set of technologies and behaviors as EPA's judgment of least-cost compliance, the power sector is free to adopt alternative technologies and behaviors to achieve the same environmental outcome EPA has deemed in the public interest as laid out in the Clean Air Act. Such regulation serves to promote innovation and the development of new and cheaper technologies. As an example, cost estimates of the Acid Rain SO_2 trading program by Resources for the Future (RFF) and MIT's Center for Energy and Environmental Policy Research (CEEPR) have been as much as 83 percent lower than originally projected by the EPA (see Carlson et al., 2000; Ellerman, 2003). It is important to note that the original analysis for the Acid Rain Program done by EPA also relied on an optimization model like IPM. Ex ante, EPA cost estimates of roughly \$2.7 to \$6.2 billion[1] in 1989 were an overestimate of the costs of the program in part because of the limitation of economic modeling to perfectly anticipate technological improvement of pollution controls and economic improvement of other compliance options such as fuel switching. Ex post estimates of the annual cost of the Acid Rain SO_2 trading program range from \$1.0 to \$1.4 billion.

In recognition of this historic pattern of overestimated regulatory cost, EPA's mobile source program uses adjusted engineering cost estimates of pollution control equipment and installation costs.[2] To date, and including this analysis, EPA has not incorporated a similar approach into IPM modeling of EGU compliance with environmental constraints. As a result, this analysis may overstate costs where such cost savings from as-yet untapped improvements to pollution control technologies may occur in the future. Considering the broad and complex suite of generating technologies, fuels, and pollution control strategies available to the power sector, as well as the fundamental role of operating cost in electricity dispatch, it is not possible to apply a single technology-improving "discount" transformation to the cost projections in this analysis. The Agency will consider additional methodologies in the future which may inform the amount

[1] 2010 Phase II cost estimate in \$1995.

[2] See regulatory impact analysis for the Tier 2 Regulations for passenger vehicles (1999) and Heavy-Duty Diesel Vehicle Rules (2000).

by which projected compliance costs could be overstated regarding further technological development in analyses of power sector regulations.

EPA's latest update of IPM incorporates state rules or regulations and various NSR settlements adopted through December of 2010. Documentation for IPM can be found at http://www.epa.gov/airmarkets/progsregs/epa-ipm and in the TSD "Updates to EPA Base Case v.4.10 Using the Integrated Planning Model."

As configured in this application, IPM does not take into account demand response (i.e., consumer reaction to electricity prices). The increased retail electricity prices shown in Table 8-13 would prompt end users to increase investment in energy efficiency and/or curtail (to some extent) their use of electricity and encourage them to use substitutes.[1] Those responses would lessen the demand for electricity, resulting in electricity price increases slightly lower than IPM predicts, which would also reduce generation and emissions. Demand response would yield certain unquantified cost savings from requiring less electricity to meet the quantity demanded. To some degree, these saved resource costs will offset the additional costs of pollution controls and fuel switching that EPA anticipates from the proposed Toxics Rule. Although the reduction in electricity use is likely to be small, the cost savings from such a large industry[2] are not insignificant. EIA analysis examining multi-pollutant legislation in 2003 indicated that the annualized costs of the Toxics Rule may be overstated substantially by not considering demand response, depending on the magnitude and coverage of the price increases.[3]

EPA's IPM modeling of the proposed Toxics Rule reflects the Agency's authority to allow facility-level compliance with the HAP emission standards rather than require each affected unit at a given facility to meet the standards separately. This flexibility would offer important cost savings to facility owners in situations where a subset of affected units at a given facility could be controlled more cost-effectively such that their "overperformance" would compensate for any "underperformance" of the rest of the affected units. EPA's modeling in this analysis required the average emission rate across all affected units at a given facility to meet the standard. This averaging flexibility has the potential to offer further cost savings beyond this

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[1] The degree of substitution/curtailment depends on the costs and performance of the goods that substitute for more energy consuming goods, which is reflected in the demand elasticity.

[2] Investor-owned utilities alone accounted for nearly $300 billion in revenue in 2008 (EIA).

[3] See "Analysis of S. 485, the Clear Skies Act of 2003, and S. 843, the Clean Air Planning Act of 2003." Energy Information Administration. September, 2003. EIA modeling indicated that the Clear Skies Act of 2003 (a nationwide cap and trade program for SO_2, NO_X, and mercury), demand response could lower present value costs by as much as 47% below what it would have been without an emission constraint similar to the Transport Rule.

analysis if particular units find ways to achieve superior pollution control beyond EPA's assumptions of retrofit technology performance at the modeled costs (which could then reduce the need to control other units at the same facility).

Additionally, EPA has chosen to express most of the control requirements here as engineering performance standards (e.g., lbs/MMBtu of heat input), which provide power plant operators goals to meet as they see fit in choosing coals with various pollutant concentrations and pollutant control technologies that they adopt to meet the requirements. Historically, such an approach encourages industry to engineer cheaper solutions over time to achieve the pollution controls requirements.

EPA's IPM modeling is based on retrofit technology cost assumptions which reflect the best available information on current and foreseeable market conditions for pollution control deployment. In the current economic environment, EPA does not anticipate (and thus this analysis does not reflect) significant near-term price increases in retrofit pollution control supply chains in response to the proposed Toxics Rule. To the extent that such conditions may develop during the sector's installation of pollution control technologies under the proposed Toxics Rule, this analysis may understate the cost of compliance.

8.15 Significant Energy Impact

The Proposed Toxics Rule would have a significant impact according to *E.O. 13211: Actions that Significantly Affect Energy Supply, Distribution, or Use*. Under the provisions of this proposed rule, EPA projects that approximately 9.9 GW of coal-fired generation (roughly 3 percent of all coal-fired capacity and 1% of total generation capacity in 2015) may be removed from operation by 2015. These units are predominantly smaller and less frequently-used generating units dispersed throughout the area affected by the rule. If current forecasts of either natural gas prices or electricity demand were revised in the future to be higher, that would create a greater incentive to keep these units operational.

EPA also projects fuel price increases resulting from the proposed Toxics Rule. Average retail electricity price are shown to increase in the contiguous U.S. by 3.7 percent in 2015. This is generally less of an increase than often occurs with fluctuating fuel prices and other market factors. Related to this, the average delivered coal price increases by less than 1 percent in 2015 as a result of shifts within and across coal types. As discussed above in section 8.10, EPA also projects that electric power sector-delivered natural gas prices will increase by about 1.3% percent over the 2015-2030 timeframe and that natural gas use for electricity generation will increase by less than 300 billion cubic feet (BCF) over that horizon. These impacts are well

within the range of price variability that is regularly experienced in natural gas markets. Finally, the EPA projects coal production for use by the power sector, a large component of total coal production, will decrease by 20 million tons in 2015 from base case levels, which is less than 2 percent of total coal produced for the electric power sector in that year. The EPA does not believe that this rule will have any other impacts (e.g., on oil markets) that exceed the significance criteria.

8.16 References

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APPENDIX D.
ILLUSTRATIVE END-USE ENERGY EFFICIENCY POLICY SENSITIVITY

This appendix summarizes the approach taken to define and analyze an illustrative energy efficiency policy sensitivity ("energy efficiency sensitivity"), and presents the results of the analysis. This appendix provides the basis for the discussion of the energy efficiency sensitivity provided in the rule preamble (Section M) and the RIA (Section 8.13). For completeness some discussion is repeated from Section 8.13.

D.1 Basis for the Energy Efficiency Sensitivity, Electricity Demand Impacts, and Associated Energy Efficiency-related Costs

To explore the possible impacts of this rule under an alternative baseline with increased federal and state energy efficiency policies and resultant lower levels of electricity generation, EPA developed an illustrative end-use energy efficiency policy scenario and analyzed the associated effects. By possibly reducing electricity demand, this illustrative energy efficiency scenario avoids emissions of all pollutants associated with electricity generation, including emissions of toxic air pollutants targeted by this rule. This energy efficiency sensitivity illustrates a possible alternative future where increased use of well designed and implemented energy efficiency policies lead to increased investment in cost-effective energy end-use technologies beyond what is reflected in the reference electricity demand forecast used for EPA's core analysis. This sensitivity does not represent an EPA forecast of electricity demand.

EPA based the energy efficiency sensitivity on two policies: implementation of federal appliance standards for products required under existing statutes and the increased possible use of ratepayer-funded energy efficiency programs consistent with recent state policy trends. The projected electricity demand impacts of the federal appliance standards were provided by the U.S. Department of Energy's (DOE) Office of Energy Efficiency and Renewable Energy and are an estimate of the incremental effects, relative to the AEO 2010 reference case forecast, of DOE's statutorily mandated appliance standards rulemakings (appliance standards that have been implemented are in the base case). The projected electricity demand impacts of the increased use of ratepayer-funded energy efficiency programs are derived from a 2009 analysis by the Lawrence Berkeley National Lab (LBNL).[1] For both policy approaches (federal appliance standards and ratepayer-funded energy efficiency), EPA assumed the continued use of the policies at similar levels of effectiveness, and estimated the associated impacts, through 2050

[1] Lawrence Berkeley National Laboratory, "The Shifting Landscape of Ratepayer Funded Energy Efficiency in the U.S.," (October 2009), Galen Barbose et. al., LBNL-2258E, (http://eetd.lbl.gov/ea/ems/reports/lbnl-2258e.pdf).

(the final year for EPA's regulatory analysis), which is beyond the timeframes of DOE's and LBNL's analyses (2035 and 2020, respectively).

After developing the basis for the energy efficiency sensitivity, EPA derived annual electricity demand impacts, estimated associated costs, and analyzed impacts on the electricity generation sector. The projected electricity impacts are summarized in Table D-1 and information is provided to put these reductions in context relative to the reference case forecast. The total U.S. electricity demand reductions in 2015, 2020, 2030, 2040, and 2050, for the energy efficiency scenario represent 2.9%, 5.3%, 6.6%, 6.1% and 5.8% of U.S. electricity demand in those years, respectively. These reductions lower annual average electricity demand growth (from 2009 historic data) through 2020 from the reference forecast from 1.04% to 0.55%. Similarly, through 2030 the reduction is from 0.97% to 0.64%, and through 2050 the reduction is from 0.91% to 0.77%. These reductions are substantially less than recent estimates of available, cost-effective energy efficiency potential[1].

[1] For example, McKinsey & Company, "Unlocking Energy Efficiency in the U.S. Economy," (July 2009), Hannah Choi Granade, Jon Creyts, Anton Derkach, Philip Farese, Scott Nyquist, and Ken Ostrowski (http://www.mckinsey.com/clientservice/electricpowernaturalgas/downloads/us_energy_efficiency_full_report.pdf) and Electric Power Research Institute, "Assessment of Achievable Potential of Energy Efficiency and Demand Response Programs in the U.S. (2010-2030)," (January 2009), (http://www edisonfoundation net/iee/reports/EPRI_SummaryAssessmentAchievableEEPotential0109 pdf)

Table D-1. Energy Efficiency Sensitivity Impacts on U.S. Electricity Demand (TWh)

	2009	2012	2015	2020	2030	2040	2050
Ratepayer-funded EE Programs		59	110	174	198	198	198
% of U.S. Demand		1.50%	2.70%	4.10%	4.20%	3.90%	3.60%
Federal Appliance Standards		0	6	52	112	114	124
% of U.S. Demand		0.00%	0.20%	1.20%	2.40%	2.20%	2.20%
Total EE Demand Reductions		59	117	226	310	312	322
% of U.S. Demand		1.50%	2.90%	5.30%	6.60%	6.10%	5.80%
U.S. Electricity Demand (EPA Reference)	3,838	4,043	4,086	4,302	4,703	5,113	5,568
Average Annual Growth Rate (2009 to 20xx)			1.05%	1.04%	0.97%	0.93%	0.91%
Net Demand after EE	3,838	3,984	3,969	4,076	4,392	4,801	5,246
Average Annual Growth Rate (2009 to 20xx)			0.56%	0.55%	0.64%	0.73%	0.77%

Costs associated with the two policy strategies discussed above were estimated based upon historical studies specific to each policy. For the electricity savings resulting from federal appliance standards a "cost of saved energy" of $33.70/MWh (2007$) is used based on a study by Resources for the Future.[1] This figure represents the costs associated with energy savings resulting from federally mandated appliance standards and is based upon their analysis of historical data. These costs are borne by appliance manufacturers to produce products meeting the mandated energy standards and may be reflected in prices for those products paid by consumers. For the electricity savings resulting from ratepayer-funded energy efficiency programs, a value of $46/MWh for the cost of saved energy is used based upon a national survey

[1] Resources for the Future, "Retrospective Examination of Demand-Side Energy Efficiency Policies," (June 2004), Kenneth Gillingham, Richard G. Newell, and Karen Palmer (http://www.rff.org/Documents/RFF-DP-04-19rev.pdf). RFF Study concluded that appliance standards cost of saved energy was $28/MWh in 2000$ which we inflated to $33.70/MWh in 2007$.

conducted by the American Council for an Energy Efficient Economy of program evaluations conducted by states and/or utilities[1].

D.2 Application within IPM and Summary Results

The annual estimated electricity demand impacts for the energy efficiency sensitivity were used to develop two additional IPM runs, one using the adjusted demand within the EPA base case and the other using the adjusted demand within the EPA Toxics Rule case. The results from these runs are summarized in Tables D-2 through D-11 and show the effects of the energy efficiency sensitivity on the following key outputs: total costs (Table D-2), retail electricity prices (Table D-3), required new generation capacity (Table D-4), CO2 emissions (Table D-5), SO2 emissions (Table D-6), NOx emissions (Table D-7), Hg emissions (Table D-8), total retirements (Table D-9), coal retirements (Table D-10), and required FGD retrofits (Table D-11). Tables D-2 and D-3 reflect the estimated costs associated with energy efficiency policies as discussed above.

Table D-2. Electric System Generation & Energy Efficiency Costs (billions of 2007$)

	2015	2020	2030
Total Costs			
Base Case	$144.3	$155.2	$200.4
Base Case w/ Energy Efficiency (EE)	$142.3	$150.3	$189.8
Toxics Rule Case	$155.2	$165.3	$210.3
Toxics Rule Case w/ Energy Efficiency (EE)	$152.9	$159.3	$198.9
Incremental Costs			
Base to Base w/EE	-$2.0	-$4.9	-$10.6
Toxics Rule to Toxics Rule w/EE	-$2.3	-$6.0	-$11.4
Base to Toxics Rule	$10.9	$10.1	$10.0
Base with EE to Toxics Rule w/EE	$10.5	$9.0	$9.1
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-$0.3	-$1.1	-$0.8

Source: Integrated Planning Model run by EPA, 2011, and EPA estimates of energy efficiency policy costs.

[1] American Council for an Energy-Efficient Economy, "Saving Energy Cost-Effectively: A National Review of Cost of Energy Saved Through Utility-Sector Energy Efficiency Programs," (September 2009), Report Number U092, Katherine Friedrich, Maggie Eldridge, Dan York, Patti Witte, and Marty Kushler (http://www.aceee.org/sites/default/files/publications/researchreports/U092.pdf). The sub-components of this value are the costs borne by utilities and/or their ratepayers of $25/MWh and costs borne by program participants of $21/MWh.

Table D-3. Projected Contiguous U.S. Electricity Prices Including Energy Efficiency Costs (2007 cents/kWh)

	2015	2020	2030
Base Case	9.01	8.94	10.16
Base Case w/ Energy Efficiency (EE)	8.95	8.54	9.72
Toxics Rule Case	9.35	9.17	10.35
Toxics Rule Case w/ Energy Efficiency (EE)	9.31	8.80	9.93
Incremental Price Changes			
Base to Base w/EE	-0.07	-0.40	-0.44
Toxics Rule to Toxics Rule w/EE	-0.04	-0.38	-0.42
Base to Toxics Rule	0.33	0.23	0.19
Base with EE to Toxics Rule w/EE	0.36	0.26	0.21
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.03	0.02	0.02

Source: Integrated Planning Model run by EPA, 2011, EPA's Retail Electricity Price Model, and EPA estimates of energy efficiency policy costs.

Table D-4. New Capacity Additions Including Energy Efficiency Cases (Cumulative GW)

	2015	2020	2030
Base Case	29.6	36.3	108.0
Base Case w/ Energy Efficiency (EE)	29.3	30.6	71.2
Toxics Rule Case	29.7	39.2	116.0
Toxics Rule Case w/ Energy Efficiency (EE)	29.4	30.8	76.3
Incremental Capacity Additions			
Base to Base w/EE	-0.4	-5.7	-36.9
Toxics Rule to Toxics Rule w/EE	-0.3	-8.5	-39.8
Base to Toxics Rule	0.1	3.0	8.0
Base with EE to Toxics Rule w/EE	0.1	0.2	5.1
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.1	-2.8	-2.9

Source: Integrated Planning Model run by EPA, 2011

Table D-5. CO2 Emissions Impacts Including Energy Efficiency Cases (million metric tonnes)

	2015	2020	2030
Base Case	2243	2326	2484
Base Case w/ Energy Efficiency (EE)	2190	2222	2372
Toxics Rule Case	2219	2297	2449
Toxics Rule Case w/ Energy Efficiency (EE)	2144	2181	2321
Incremental Emissions Impacts			
Base to Base w/EE	-53	-103	-112
Toxics Rule to Toxics Rule w/EE	-74	-115	-128
Base to Toxics Rule	-24	-29	-35
Base with EE to Toxics Rule w/EE	-45	-41	-51
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-21	-12	-16

Source: Integrated Planning Model run by EPA, 2011

Table D-6. SO2 Emissions Impacts Including Energy Efficiency Cases (million tons)

	2015	2020	2030
Base Case	3.89	3.87	3.71
Base Case w/ Energy Efficiency (EE)	3.86	3.84	3.67
Toxics Rule Case	1.84	1.85	1.90
Toxics Rule Case w/ Energy Efficiency (EE)	1.80	1.78	1.85
Incremental Emissions Impacts			
Base to Base w/EE	-0.03	-0.03	-0.04
Toxics Rule to Toxics Rule w/EE	-0.04	-0.08	-0.05
Base to Toxics Rule	-2.05	-2.01	-1.81
Base with EE to Toxics Rule w/EE	-2.06	-2.06	-1.82
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-0.01	-0.04	-0.01

Source: Integrated Planning Model run by EPA, 2011

Table D-7. NOx Emissions Impacts Including Energy Efficiency Cases (million tons)

	2015	2020	2030
Base Case	2.02	2.07	2.15
Base Case w/ Energy Efficiency (EE)	1.97	1.98	2.10
Toxics Rule Case	1.88	1.94	2.01
Toxics Rule Case w/ Energy Efficiency (EE)	1.81	1.82	1.92
Incremental Emissions Impacts			
Base to Base w/EE	-0.05	-0.09	-0.05
Toxics Rule to Toxics Rule w/EE	-0.07	-0.11	-0.08
Base to Toxics Rule	-0.14	-0.13	-0.14
Base with EE to Toxics Rule w/EE	-0.16	-0.16	-0.17
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-0.02	-0.02	-0.03

Source: Integrated Planning Model run by EPA, 2011

Table D-8. Hg Emissions Impacts Including Energy Efficiency Cases (tons)

	2015	2020	2030
Base Case	26.69	27.08	27.34
Base Case w/ Energy Efficiency (EE)	26.20	26.29	26.66
Toxics Rule Case	8.72	8.86	9.05
Toxics Rule Case w/ Energy Efficiency (EE)	8.58	8.61	8.82
Incremental Emissions Impacts			
Base to Base w/EE	-0.49	-0.79	-0.68
Toxics Rule to Toxics Rule w/EE	-0.15	-0.26	-0.23
Base to Toxics Rule	-17.97	-18.21	-18.29
Base with EE to Toxics Rule w/EE	-17.62	-17.68	-17.85
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.34	0.53	0.45

Source: Integrated Planning Model run by EPA, 2011

Table D-9. Total Retirements Including Energy Efficiency Cases (GW)

	2015	2020	2030
Base Case	26.9	27.4	27.4
Base Case w/ Energy Efficiency (EE)	37.8	54.2	53.5
Toxics Rule Case	35.3	35.1	35.1
Toxics Rule Case w/ Energy Efficiency (EE)	46.6	60.1	59.5
Incremental Retirements			
Base to Base w/EE	10.9	26.7	26.0
Toxics Rule to Toxics Rule w/EE	11.3	25.1	24.4
Base to Toxics Rule	8.5	7.6	7.6
Base with EE to Toxics Rule w/EE	8.8	6.0	6.0
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	0.4	-1.7	-1.6

Source: Integrated Planning Model run by EPA, 2011

Table D-10. Coal Retirements Including Energy Efficiency Cases (GW)

	2015	2020	2030
Base Case	4.6	5.1	5.1
Base Case w/ Energy Efficiency (EE)	11.8	12.4	12.4
Toxics Rule Case	14.5	14.2	14.2
Toxics Rule Case w/ Energy Efficiency (EE)	24.7	24.3	24.3
Incremental Retirements			
Base to Base w/EE	7.2	7.3	7.3
Toxics Rule to Toxics Rule w/EE	10.2	10.1	10.1
Base to Toxics Rule	9.9	9.1	9.1
Base with EE to Toxics Rule w/EE	12.9	11.9	11.9
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	3.0	2.8	2.8

Source: Integrated Planning Model run by EPA, 2011

Table D-11. FGD Retrofits Including Energy Efficiency Cases (cumulative GW)

	2015	2020	2030
Base Case	12.4	18.4	18.7
Base Case w/ Energy Efficiency (EE)	11.3	16.2	16.5
Toxics Rule Case	33.2	33.6	34.0
Toxics Rule Case w/ Energy Efficiency (EE)	29.7	30.1	30.3
Incremental Retrofits			
Base to Base w/EE	-1.1	-2.2	-2.2
Toxics Rule to Toxics Rule w/EE	-3.5	-3.4	-3.6
Base to Toxics Rule	20.8	15.2	15.3
Base with EE to Toxics Rule w/EE	18.4	13.9	13.8
(Base to Toxics Rule) to (Base w/EE to Toxics Rule w/EE)	-2.4	-1.2	-1.5

Source: Integrated Planning Model run by EPA, 2011

Chapter 9
ECONOMIC AND EMPLOYMENT IMPACTS

9.1 Partial Equilibrium Analysis (Multiple Markets)

Our partial equilibrium analysis uses a market model that simulates how stakeholders (consumers and industries) might respond to the additional regulatory program costs. In this section, we provide an overview of the economic model and the results for a short-run economic impact analysis (in this case, for 2016, the analysis year for this RIA). More details on the economic model, the results, and data used by the model can be found in Appendix E.

9.1.1 Overview

Although several tools are available to estimate social costs, current EPA guidelines suggest that multimarket models "...are best used when potential impacts on related markets might be considerable" and modeling using a computable general equilibrium model is not available or practical (EPA, 2010, p. 9-21). Other guides for environmental economists offer similar advice (Berck and Hoffmann, 2002; Just, Hueth, and Schmitz, 2004). Multimarket models focus on "short-run" time horizons and measure a policy's near-term or transition costs (EPA, 1999). Our multimarket model contains the following features:

- Industry sectors and benchmark data set
  - 100 industry sectors
  - multiple benchmark years
- Economic behavior
  - industries respond to regulatory costs by changing production rates
  - market prices rise and fall to reflect higher energy and other non-energy material costs and changes in demand
  - customers respond to price increases and consumption falls
- Model scope
  - 100 sectors are linked with each other based on their use of energy and other non-energy materials. For example, the construction industry is linked with the petroleum, cement, and steel industries and is influenced by price changes that occur in each sector. The links allow EPA to account for indirect effects the regulation has on related markets.
  - production adjustments influence employment levels
  - international trade (imports/exports) responds to domestic price changes

- Model time horizon ("short run") for a single period (2015)[1]
  - fixed production resources (e.g., capital) lead to an upward-sloping industry supply function
  - firms cannot alter certain input mixes; there is no substitution among intermediate production inputs
  - there is no explicit labor market (a real wage and labor supply is not determined within the model)
  - investment and government expenditures are fixed.

    Although the model is intended to examine transition or short-term effects of this rulemaking, the results may be muted due to the use of annualized capital cost as an input to the model rather than the total capital cost.

9.1.2 Economic Impact Analysis Results

Market-Level Results

Market-level impacts include price and quantity adjustments including the changes in international trade (Table 9-1). Under the Toxics rule, the Agency's economic model suggests the average national price increase for energy is 0.8%. Higher energy costs result in subsequent manufacturing sector price increases nationwide of 0.1% or less. Imports also slightly rise because of higher U.S. prices. The one exception is transportation services; since sectors using transportation services are producing less, the demand for transportation services declines. The size of the transportation services demand shift outweighs any supply side cost increases that place upward pressure on service prices (e.g. higher electricity and refined petroleum prices). As a result, the average transportation services price falls.

Social Cost Estimates Toxics Rule

In the short run, the Agency's partial equilibrium multi-market model suggests that industries are able to pass on $8.4 billion (2007$) of the Toxic Rule's costs to U.S. households in the form of higher prices (Table 9-2). Existing U.S. industries' surplus falls by $2.6 billion and the net U.S. loss in aggregate, is $11.0 billion (2007$). This is slightly higher than the annualized nationwide compliance cost estimate of the proposal as shown in Chapter 8 of the RIA because it excludes gains to other countries discussed below.

[1] For this analysis, we use 2015 as our analysis year and as a proxy for 2016. This allows us to maintain consistency with the results of the analysis using IPM (found in Chapter 8) that serve as inputs to this economic impact analysis.

Table 9-1. Short-Term Market-Level Changes within the U.S. Economy in 2015

Industry Sector	U.S. Prices	U.S. Production	Imports	U.S. Consumption	Exports
Energy	0.769%	−0.120%	0.035%	−0.072%	−0.120%
Coal	−0.078%	−0.215%	−0.167%	−0.214%	0.008%
Crude Oil Extraction	0.018%	−0.234%	0.068%	−0.011%	0.000%
Electric generation	3.770%	−0.261%	0.000%	−0.261%	−0.592%
Natural Gas	0.018%	−0.142%	0.217%	−0.075%	−0.005%
Refined Petroleum	0.011%	−0.011%	0.010%	−0.007%	−0.001%
Nonmanufacturing	0.003%	−0.012%	0.005%	−0.010%	−0.003%
Manufacturing					
Food, beverages, and textiles	0.018%	−0.023%	0.025%	−0.013%	−0.014%
Lumber, paper, and printing	0.035%	−0.023%	0.035%	−0.017%	−0.024%
Chemicals	0.009%	−0.024%	0.010%	−0.017%	−0.009%
Plastics and Rubber	0.026%	−0.026%	0.029%	−0.017%	−0.026%
Nonmetallic Minerals	0.048%	−0.029%	0.043%	−0.018%	−0.040%
Primary Metals	0.031%	−0.041%	0.028%	−0.024%	−0.030%
Fabricated Metals	0.026%	−0.016%	0.028%	−0.011%	−0.013%
Machinery and Equipment	0.003%	−0.015%	0.002%	−0.010%	−0.004%
Electronic Equipment	0.003%	−0.017%	0.004%	−0.008%	−0.007%
Transportation Equipment	0.004%	−0.011%	0.005%	−0.007%	−0.009%
Other	0.011%	−0.027%	0.017%	−0.011%	−0.014%
Wholesale and Retail Trade	0.007%	−0.008%	0.005%	−0.008%	−0.005%
Transportation Services	−0.012%	−0.015%	−0.011%	−0.014%	0.010%
Other Services	0.007%	−0.008%	0.003%	−0.007%	−0.005%

Note: Approximated using the IPM cost analysis. For example, with the $11 billion increase in compliance costs for the electric power sector, IPM projects a 3.77 percent increase in the retail price of electricity. All other energy market-level changes are determined within the multimarket model. Appendix F provides additional details.

As U.S. prices rise, other countries are affected through international trade relationships. The price of goods produced in the United States increase, domestic exports decline, and domestic production is replaced to a certain degree by imports; the model estimates a net gain of about $0.1 billion for other countries. The net change in total surplus is *lower* than the annualized nationwide compliance cost estimate of the proposal as shown in Chapter 8 of the RIA. Our estimate of social costs for the proposal incorporates the net change in total surplus, and this estimate is $10.9 billion (2007 dollars) as shown in Table 9-2, or nearly identical to the compliance costs.[2] Compliance costs based on the pre-policy output levels would be overstated if we do not consider the new lower levels of consumption as a result of higher market prices.[3]

[2] The same is true for many recent rulemakings, including the Boiler MACT.

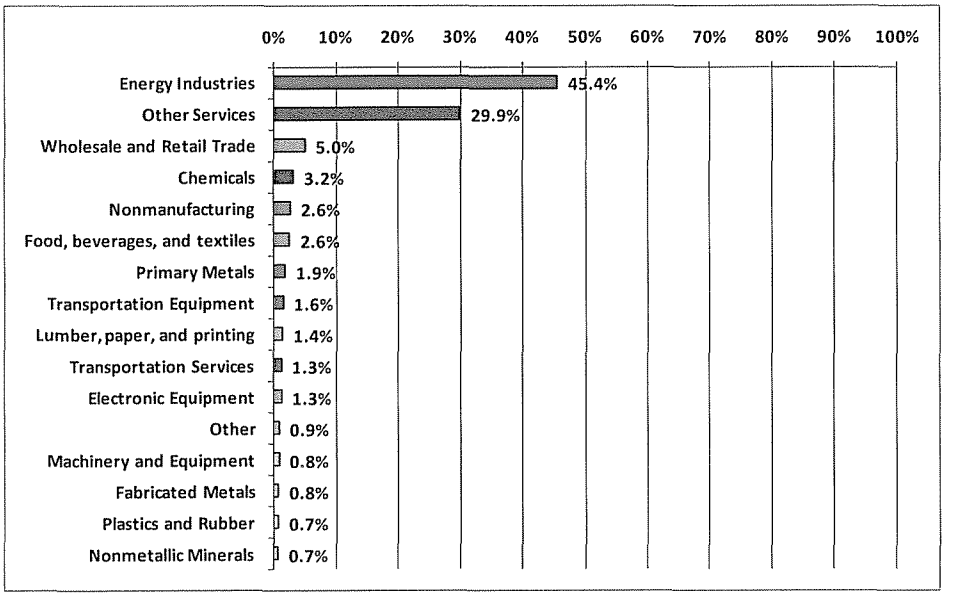
[3] There are small additional losses associated with the foregone benefits associated with reduced consumption (e.g. deadweight loss). However, in a perfectly competitive market without pre-existing distortions, the costs represent only a small fraction of total social costs. A more detail discussion of the economic costs of regulation are discussed in Chapter 8 of EPA (2010).

Table 9-2. Distribution of Social Costs (billions, 2007$): 2015

Change in U.S. consumer surplus	−$8.4
Change in U.S. producer surplus	−$2.6
Net Change in U.S. Surplus	**−$11.0**
Net change in rest of world surplus	$0.1
Net change in Total Surplus	**−$10.9**

As shown in Figure 9-1, the surplus losses are concentrated in the electric generation sector (45.4 percent) and other services (29.9 percent). Other services include information, finance and insurance, real estate, professional services, management, administrative services, education, health care, arts, accommodations, and public services. Although electricity costs represent a small share of total service industry production costs, the service sectors represent a significant economic sector within the U.S. economy and use a large amount of electricity. The transition or short-term evaluation using a partial equilibrium model does not allow for resources to be allocated according to price changes. So the results of the model does not capture any distortions in the economy that may results as the price of electricity changes. If the distortions are significant, the "true" social cost would be higher than the compliance cost and the results of this partial equilibrium model.

Figure 9-1. Distribution of Total Surplus Change ($10.9 billion) by Sector



9.1.3 Alternative Approach to Estimating Social Cost

In the Transport Rule proposed last summer, EPA used a different model to estimate the social cost of the regulatory approach than applied in this RIA. That model, EPA's Economic Model for Policy Analysis (EMPAX), is a computable general equilibrium model (CGE) which dynamically cascades the cost of a regulation through the entire economy. However, since that rule was proposed, an updated version of EMPAX was used to estimate the social cost of the Clean Air Act in a new EPA report entitled "The Benefits and Costs of the Clean Air Act from 1990 to 2020. This report is available at http://www.epa.gov/air/sect812/feb11/fullreport.pdf.

This updated version of EMPAX added in the benefit-side effects (incorporating labor-force and health care expenditures) which significantly changed the social cost estimate from the previous edition. In December 2010, EPA's Science Advisory Board (SAB) found that "The inclusion of benefit-side effects (reductions in mortality, morbidity, and health-care expenditures) in a computable general equilibrium (CGE) model represents a significant step forward in benefit-cost analysis." http://yosemite.epa.gov/sab/sabproduct.nsf/1E6218DE3BFF682E852577FB005D46F1/$File/EPA-COUNCIL-11-001-unsigned.pdf. A description of the changes to the model and implications are covered in detail in chapter 8 of the section 812 report. EPA has determined that it needs to update the EMPAX model version used for RIAs to add this benefit-side effect prior to use in any additional regulatory analysis. EPA plans to use the updated version of EMPAX for the final RIA.

9.2 Employment Impacts for the Proposed Toxics Rule

In addition to addressing the costs and benefits of the proposed Utility Air Toxics Rule (Toxics Rule), EPA has estimated preliminary impacts of this rulemaking on labor demand, which are presented in this section.[4] While a standalone analysis of employment impacts is not included in a standard cost-benefit analysis, such an analysis is of particular concern in the current economic climate of sustained unemployment. Executive Order 13563, states, "Our regulatory system must protect public health, welfare, safety, and our environment while promoting economic growth, innovation, competitiveness, and job creation" (emphasis added). Therefore, we have provided this analysis to inform the discussion of labor demand and job impacts. We provide an estimate of the employment impacts on the regulated industry over time. We also provide the short-term employment impacts (increase in labor demand) associated

[4] See TSD as part of the Toxics Rule Docket: "Employment Estimates of Direct Labor in Response to the Proposed Toxics Rule in 2015."

with the construction of needed pollution control equipment until the compliance date of the regulation.

We have not quantified the rule's effects on all labor in other sectors not regulated by this proposal, or the effects induced by changes in workers' incomes. What follows is an overview of the various ways that environmental regulation can affect employment, followed by a discussion of the estimated impacts of this rule. EPA continues to explore the relevant theoretical and empirical literature and to seek public comments in order to ensure that such estimates are as accurate, useful and informative as possible.

From an economic perspective labor is an input into producing goods and services; if regulation requires that more labor be used to produce a given amount of output, that additional labor is reflected in an increase in the cost of production. Moreover, when the economy is at full employment, we would not expect an environmental regulation to have an impact on overall employment because labor is being shifted from one sector to another. On the other hand, in periods of high unemployment, an increase in labor demand due to regulation may result in a net increase in overall employment. With significant numbers of workers unemployed, the opportunity costs associated with displacing jobs in other sectors are likely to be much smaller.

To provide a partial picture of the employment consequences of this rule, EPA takes two approaches. First, the analysis uses the results of Morgenstern, Pizer, and Shih (2002) to estimate the effects of the regulation on the regulated industry, the electric power industry in this case. This approach has been taken by EPA previously in Regulatory Impact Analyses. (See, for example, the Regulatory Impact Analysis for the recently finalized Industrial Boilers and CISWI rulemakings, promulgated on February 21, 2011). Second, EPA uses information derived from its cost estimation documentation for the IPM model. Historically, EPA has only reported employment impacts on a few regulations. EPA is interested in public comments on the merits of including information derived in this fashion for assessing the employment consequences of regulations.

Section 9.3 discusses the estimates of the employment consequences in the electricity sectors, using the Morgenstern, et al. approach. Section 9.4 estimates the employment consequences in the environmental protection sector, using the new approach.

9.3 Employment Impacts primarily on the regulated industry: Morgenstern, Pizer, and Shih (2002)

EPA examined possible employment effects within the electric utility sector using a peer-reviewed, published study that explores historical relationships between industrial employment and environmental regulations (Morgenstern, Pizer, and Shih, 2002). The fundamental insight of Morgenstern, et al. is that environmental regulations can be understood as requiring regulated firms to add a new output (environmental quality) to their product mixes. Although legally compelled to satisfy this new demand, regulated firms have to finance this additional production with the proceeds of sales of their other (market) products. Satisfying this new demand requires additional inputs, including labor, and may alter the relative proportions of labor and capital used by regulated firms in their production processes.

Thus, Morgenstern et al. decompose the overall effect of a regulation on employment into the following three subcomponents:

- The "Demand Effect": higher production costs raise market prices, reducing consumption (and production), thereby reducing demand for labor within the regulated industry [5];
- The "Cost Effect": As production costs increase, plants use more of all inputs, including labor, to maintain a given level of output. For example, in order to reduce pollutant emissions while holding output levels constant, regulated firms may require additional labor;
- The "Factor-Shift Effect": Regulated firms' production technologies may be more or less labor intensive after complying with a regulation (i.e., more/less labor is required per dollar of output).
- Decomposing the overall employment impact of environmental regulation into three subcomponents clarifies the conceptual relationship between environmental regulation and employment in regulated sectors, and permitted Morgenstern, et al. to provide an empirical estimate of the net impact. For present purposes, the net effect is of particular interest, and is the focus of our analysis.

Using plant-level Census information between the years 1979 and 1991, Morgenstern et al. estimate the size of each effect for four polluting and regulated industries (petroleum, plastic

[5] The Morgenstern et al. results rely on industry demand and supply elasticities to determine cost pass-through and reductions in output.

material, pulp and paper, and steel). On average across the four industries, each additional $1 million ($1987) spending on pollution abatement results in a (statistically insignificant) net increase of 1.55 (+/- 2.24) jobs. As a result, the authors conclude that increases in pollution abatement expenditures do not necessarily cause economically significant employment changes. The conclusion is similar to Berman and Bui (2001) who found that increased air quality regulation in Los Angeles did not cause in large employment changes[6].

Since the Morgenstern, et al. parameter estimates are expressed in jobs per million ($1987)[7] of environmental compliance expenditures, their study offers a transparent and simple way to transfer estimates for other employment analysis. For each of the three job effects outlined above, EPA used the Morgenstern et al. four industry average parameters and standard errors along with the estimated private compliance costs to provide a range of electricity sector employment effects associated with the proposed Toxics Rule.

By applying these estimates to pollution abatement costs for the proposed rule for the electric power sector, we estimated each effect. The results are

- Demand effect: −45,000 to +2,500 jobs in the directly affected sector with a central estimate of −21,000;
- Cost effect: +4,700 to +24,000 jobs in the directly affected sector with a central estimate of +14,000; and
- Factor-shift effect: +200 to +32,000 jobs in the directly affected sector with a central estimate of +16,000.
- EPA estimates the net employment effect to range from 17,000 to +35,000 jobs in the directly affected sector with a central estimate of +9,000.[8,9]

These estimates are shown in Table 9-3.

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[6] For alternative views, see Henderson (1996) and Greenstone (2002).

[7] The Morgenstern et al. analysis uses "production worker" as defined in the US Census Bureau's Annual Survey of Manufactures (ASM) in order to define a job. This definition can be found on the Internet at http://www.census.gov/manufacturing/asm/definitions/index.html.

[8] Since Morgenstern's analysis reports environmental expenditures in $1987, we make an inflation adjustment the IPM costs using the ratio of the consumer price index, U.S. city, all items reported by the U.S. Bureau of Labor Statistics: $CPI_{1987}/ CPI_{2007} = (113.6/207.3) = 0.55$

[9] Net employment effect = 1.55× $10,900 million × 0.55. This estimated net result is not statistically different from zero.

Table 9-3. Employment Impacts Using Peer-Reviewed Study

	Estimates using Morgenstern et al. (2002)			
	Demand Effect	**Cost Effect**	**Factor Shift Effect**	**Net Effect**
Change in Full-Time Jobs per Million Dollars of Environmental Expenditure[a]	−3.56	2.42	2.68	1.55
Standard Error	2.03	1.35	0.83	2.24
EPA estimate for Toxics Rule[b]	−21,000 −45,000 to +2,500	+14,000 +4,700 to +24,000	+16,000 +200 to +32,000	+9,000 −17,000 to +35,000

[a] Expressed in 1987 dollars. See footnote 8 for inflation adjustment factor used in the analysis.

[b] According to the 2007 Economic Census, the electric power generation, transmission and distribution sector (NAICS 2211) had approximately 510,000 paid employees. Both the midpoint and range for each effect are reported in the last row of the table.

All ranges for these job changes are based on the 95th percentile of results. EPA recognizes there may be other employment effects which are not considered in the Morgenstern et al. study. For example, employment in environmental protection industries may increase as firms purchase more pollution control equipment and services to meet the proposed rule's requirements. EPA does provide such an estimate of employment change later in this section in a separate analysis. On the other hand, industries that use electricity will face higher electricity prices as the result of the toxics rule, reduce output, and demand less labor. We do not currently have sufficient information to quantify these as potential employment gains or losses.

9.3.1 Limitations

Although the Morgenstern et al. paper provides information about the potential job effects of environmental protection programs, there are several caveats associated with using those estimates to analyze the final rule. First, the Morgenstern et al. estimates presented in Table 9-3 and used in EPA's analysis represent the weighted average parameter estimates for a set of manufacturing industries (pulp and paper, plastics, petroleum, and steel). Morgenstern, et al. present those industries' estimates separately, and they range from -1.13 jobs per $1 million (in 1987 dollars) of environmental expenditures for pulp and paper, to +6.90 jobs for plastics. Only two of the total jobs estimates are statistically significantly different from zero, and the

overall weighted average used here, 1.55 jobs per $1 million, is not statistically significant. Moreover, here we are applying the estimate to the electricity generating industry.

Second, relying on Morgenstern et al. implicitly assumes that estimates derived from 1979–1991 data are still applicable. Third, the methodology used in Morgenstern et al. assumes that regulations affect plants in proportion to their total costs. In other words, each additional dollar of regulatory burden affects a plant by an amount equal to that plant's total costs relative to the aggregate industry 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 or larger plants.

9.4 Employment Impacts of the Proposed Toxics Rule-Environmental Protection Sector Approach by 2015 [10]

Regulations set in motion new orders for pollution control equipment and services. New categories of employment have been created in the process of implementing regulations to make our air safer to breathe. When a regulation is promulgated, the first response of industry is to order pollution control equipment and services in order to comply with the regulation when it becomes effective. Revenue and employment in the environmental technology industry have grown steadily between 2000 and 2008, reaching an industry total of approximately $300 billion in revenues and 1.7 million employees in 2008.[11] While these revenues and employment figures represent gains for the environmental technologies industry, they are costs to the regulated industries required to install the equipment. Moreover, it is not clear the 1.7 million employees in 2008 represent anything other than workers diverted from other productive employment as opposed to new additional employment.

Regulated firms hire workers to operate and maintain pollution controls. Once the equipment is installed, regulated firms hire workers to operate and maintain the pollution control equipment – much like they hire workers to produce more output. A study by Resources for the

[10] EPA expects that the installation of retrofit control equipment in response to the requirements of this proposal will primarily take place within 3 years of the effective date of the final rule, but there may be a possibility that some installations may occur within 4 years of the effective date.

[11] In 2008, the industry totaled approximately $315 billion in revenues and 1.9 million employees including indirect employment effects, pollution abatement equipment production employed approximately 4.2 million workers in 2008. These indirect employment effects are based on a multiplier for indirect employment = 2.24 (1982 value from Nestor and Pasurka - approximate middle of range of multipliers 1977-1991). Environmental Business International (EBI), Inc., San Diego, CA. Environmental Business Journal, monthly (copyright). http://www.ebiusa.com/ EBI data taken from the Department of Commerce International Trade Administration Environmental Industries Fact Sheet from April 2010: http://web.ita.doc.gov/ete/eteinfo.nsf/068f3801d047f26e85256883006ffa54/4878b7e2fc08ac6d85256883006c452c?OpenDocument

Future examined how regulated industries respond to regulation. They found that on average, employment goes up in regulated firms.[12,13] Of course, these firms may also reassign existing employees to do these activities.

Environmental regulations support employment in many basic industries. In addition to the increase in employment in the environmental protection industry (increased orders for pollution control equipment), environmental regulations also support employment in industries that provide intermediate goods to the environmental protection industry. For example, $1 billion in capital expenditures to reduce air pollution involves the purchase of abatement equipment. The equipment manufacturers, in turn, order steel, tanks, vessels, blowers, pumps, and chemicals to manufacture and install the equipment.

A study (2008) by Bezdek, Wendling, and DiPernab found that investments in environmental protection industries create jobs and displace jobs, but the net effect on employment is positive."[14]

The focus of this part of the employment analysis is on short-term jobs related to the compliance actions of the affected entities. This analysis estimates of the employment impacts due to the increased demand for pollution control retrofits.[15] Results indicate that the Toxics Rule has the potential to result in a net increase of labor in these industries, driven by the high demand for new pollution controls. Overall, the preliminary results of the environmental protection sector approach indicate that the Toxics Rule could support an increase of about 31,000 job-years[16] by 2015.

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[12] A recent study Bezdek, Wendling, and DiPernab shows that "investments in EP create jobs and displace jobs, but the net effect on employment is positive." *Environmental protection, the economy, and jobs: National and regional analyses,* Roger H. Bezdek, Robert M. Wendling and Paula DiPerna, Journal of Environmental Management Volume 86, Issue 1, January 2008, Pages 63-79.

[13] Environmental Business International (EBI), Inc., San Diego, CA. Environmental Business Journal, monthly (copyright). http://www.ebiusa.com/ EBI data taken from the Department of Commerce International Trade Administration Environmental Industries Fact Sheet from April 2010: http://web.ita.doc.gov/ete/eteinfo.nsf/068f3801d047f26e85256883006ffa54/4878b7e2fc08ac6d85256883006c452c?OpenDocument.

[14] Environmental protection, the economy, and jobs: National and regional analyses, Roger H. Bezdek, Robert M. Wendling and Paula DiPerna, Journal of Environmental Management Volume 86, Issue 1, January 2008, Pages 63-79.

[15] For more detail on methodology, approach, and assumptions, see TSD as part of the Toxics Rule Docket: "Employment Estimates of Direct Labor in Response to the Proposed Toxics Rule in 2015."

[16] Numbers of job years are not the same as numbers of individual jobs, but represents the amount of work that can be performed by the equivalent of one full-time individual for a year (or FTE). For example, 25 job years may be equivalent to five full-time workers for five years, twenty-five full-time workers for one year, or one full-time worker for twenty-five years.

9.4.1 Overall Approach and Methodology for Environmental Protection Sector Approach

EPA commissioned ICF International to provide estimates for the Environmental Protection Sector, and the analysis utilizes a bottom-up engineering based methodology combined with macroeconomic data on industrial output and productivity, to estimate employment impacts. It relies heavily on the cost analysis from the IPM model which uses labor and capital estimates to derive control costs. The approach also relies upon prior EPA studies on similar issues, and in particular uses data and information from an extensive resource study conducted in 2002, which was updated for purposes of the proposed rule to reflect more recent information.[17] The approach involves using IPM projected results from the proposed Toxics Rule analysis for the set of pollution control technologies expected to be installed to comply with the rule, along with data from secondary sources, to estimate the job impacts using this approach.[18] This will cover the labor needed to design, manufacture and install the needed pollution control equipment over the 3 to 4 years leading up to compliance in 2015.

For construction labor, the labor needs are derived from the 2002 EPA resource analysis for installing various retrofits (FGD – Flue Gas Desulfurization scrubbers, SCR- selective catalytic reduction, ACI – activated carbon injection, DSI - dry sorbent injection, and FF 0-Fabric Filters) and are further classified into different labor categories, such as boilermakers, engineers and a catch-all "other installation labor." For the inputs needed (e.g., steel), the 2002 resource study was used to determine the steel demand for each MW of additional pollution control and combined with labor productivity data from the Economic Census and BLS for relevant industries.

More detail on methodology, assumptions, and data sources can be found in the TSD "Employment Estimates of Direct Labor in Response to the Proposed Toxics Rule in 2015."

Projections from IPM were used to estimate the incremental retrofit capacities projected in response to the proposed rule. These additional pollution controls are shown in Table 9-4 below, and reflect the added pollution controls needed to meet the requirements of the rule. Additional information on the power sector impacts can be found in Chapter 8 of the RIA.

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[17] Engineering and Economic Factors Affecting the Installation of Control Technologies for Multipollutant Strategies EPA-600/R-02/073 (2002).

[18] Detailed results from IPM for the proposed Toxics Rule can be found in Chapter 8 of the RIA.

Table 9-4. Increased Retrofit Demand due to the Toxics Rule, by 2015 (GW)

Retrofit Type	IPM Projected Additional Pollution Control
FGD	21
SCR	3
ACI	93
DSI	56
FF[19]	107

9.4.2 Summary of Employment Estimates from Environmental Protection Sector Approach

Table 9-5 presents additional detail on the estimated employment impacts using the environmental protection sector approach resulting from the proposed Toxics Rule. Results for the Environmental Protection Sector Approach indicate the proposed Toxics Rule could support or create roughly 31,000 one-time job-years of increased cost of direct labor, driven by the need to build the pollution control retrofits.

Table 9-5. Employment Effects Using the Environmental Protection Sector Approach for the Proposed Toxics Rule (in Job-Years)

Employment	Incremental Employment
One-Time Employment Changes for Construction	
1. Boilermakers	13,400
2. Engineers	3,270
3. General Construction	13,770
4. Steel Manufacturing	430
	30,870

9.4.3 Other Employment Impacts of the Proposed Toxics Rule

We expect ongoing employment impacts on regulated and non-regulated entities for a variety of reasons. These include labor changes in the regulated entities resulting from shifts in demand for fuel changes, increased demand for materials to operate pollution control equipment, changes in employment resulting from coal retirements, and changes in other industries due to changes in the price of electricity and natural gas. We provide preliminary estimates of some of

[19] In the policy case modeling, EPA assumes that a fabric filter (also known as a baghouse) is necessary for coal- and solid-oil derived fuel-fired EGUs to meet the total PM standard. The estimate for FFs include here is for stand-alone FFs, and does not include some additional FFs that may be installed in conjunction with other pollution controls (e.g., in combination with a dry scrubber).

these effects below. The most notable of the ones we are unable to estimate are the impacts on employment as a result of the increase in electricity and other energy prices in the economy. Because of this inability to estimate all the important employment impacts, EPA neither sums the impacts that the Agency is able to estimate for the ongoing non-regulated group or make any inferences of whether there is a net gain or loss of employment for the non-regulated group. These other ongoing employment impacts are found in Table 9-6.

Table 9-6. Employment Impacts for Entities Not Regulated by the Proposed Toxics Rule

Employment Changes for Ongoing Annual Operation	
Employment Changes from Changes to Demand in Materials	
1. Limestone (FGD)	2,020
2. Ammonia (SCR)	20
3. Catalyst (SCR)	100
4. Activated Carbon (ACI)	90
5. Sodium Bicarbonate (DSI)	2,940
6. Baghouse material (FF)	60
Sub-Total:	**5,230**
Employment Changes for Ongoing Annual Retrofit Operation	**5,500**
Employment Annual Changes due to Coal Capacity Retirements	**(5,630)**
Annual Employment Changes due to Changes in Fuel Use	
Coal	(2,200)
Natural Gas	1,090
New Natural Gas Pipeline	300

9.5 Summary

The three approaches use different analytical techniques and are applied to different industries during different time periods, and they use different units of analysis. These estimates should not be summed because of the different metrics, length and methods of analysis. The Morgenstern estimates are used for the ongoing employment impacts for the regulated entities (the electric power sector). The short term estimates for employment needed to design, construct, and install the control equipment in the three or four year period leading up to the compliance date are also provided. Finally some of the other types of employment impacts that will be ongoing are estimated.

In Table 9-7, we show the employment impacts of the Toxics Rule as estimated by the environmental protection sector approach and by the Morgenstern approach.

Table 9-7. Estimated Employment Impact Table

	Annual (reoccurring)	**One time (construction during compliance period)**
Environmental Protection Sector approach*	Not Applicable	30,870
Net Effect on Electric Utility Sector Employment from Morgenstern et al. approach***	****9,000** **-17, 000 to +35,000******	Not Applicable

*These one-time impacts on employment are estimated in terms of job-years.

**This estimate is not statistically different from zero.

***These annual or recurring employment impacts are estimated in terms of production workers as defined by the US Census Bureau's Annual Survey of Manufacturers (ASM).

**** 95% confidence interval

9.6 References

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APPENDIX E.
OAQPS MULTIMARKET MODEL TO ASSESS THE ECONOMIC IMPACTS OF ENVIRONMENTAL REGULATION

E.1 Introduction

An economic impact analysis (EIA) provides information about a policy's effects (i.e., social costs); emphasis is also placed on how the costs are distributed among stakeholders (EPA, 2010). In addition, large-scale policies that may affect a large number of industries or a substantial part of the whole economy require additional analysis to better understand how costs are passed across the economy. Although several tools are available to estimate social costs, current EPA guidelines suggest that multimarket models "...are best used when potential economic impacts and equity effects on related markets might be considerable" and modeling using a computable general equilibrium model is not available or practical (EPA, 2010, p. 9-21). Other guides for environmental economists offer similar advice (Berck and Hoffmann, 2002; Just, Hueth, and Schmitz, 2004).

Multimarket models focus on "short-run" time horizons and measure a policy's near term or transition costs (EPA, 1999). Recent studies suggest short-run analyses can complement full dynamic general equilibrium analysis.

The multimarket model described in this appendix is a new addition to the Office of Air Quality Planning and Standards' (OAQPS's) economic model tool kit; it is designed to be used as a transparent tool that can respond quickly to requests about how stakeholders in 100 U.S. industries might respond to new environmental policy. It was used to analyze the economic impacts of the industrial boiler NESHAP and CISWI final rules recently signed by EPA. Next, we provide an overview of the model, data, and parameters.

E.2 Multimarket Model

The multimarket model contains the following features:

- Industry sectors and benchmark data set
  - 100 industry sectors
  - a single benchmark year (2015)[1]

[1] As mentioned in Chapter 9, we use 2015 as a proxy for 2016 in order to maintain consistency between the analysis from this model and the IPM outputs (generated for 2015, as mentioned in Chapter 8) that serve as inputs to this analysis.

- Economic behavior
  - industries respond to regulatory costs by changing production rates
  - market prices rise to reflect higher energy and other nonenergy material costs
  - customers respond to these price increases and consumption falls
- Model scope
  - 100 sectors are linked with each other based on their use of energy and other nonenergy materials. For example, the construction industry is linked with the petroleum, cement, and steel industries and is influenced by price changes that occur in each sector. The links allow EPA to account for indirect effects the regulation has on related markets.
  - Links come from input-output information from IMPLAN as used in OAQPS's computable general equilibrium (CGE) model, the Economic Model for Policy Analysis (EMPAX)
  - production adjustments influence employment levels
  - international trade (imports/exports) behavior considered
- Model time horizon ("short-run")
  - fixed production resources (e.g., capital) leads to an upward-sloping industry supply function

    firms cannot alter input mixes; there is no substitution among intermediate production inputs
  - investment and government expenditures are fixed.
- Labor and Capital Markets and Pre-existing Distortions in Other Markets
  - Unlike CGE models, our multimarket model does not include a national labor or capital market. As a result, we do not estimate real wage changes, changes in labor /leisure choices, or savings and investment decisions within the model. Therefore we do not consider whether policies interact with existing distortions, particularly tax distortions in a ways that increase or decrease estimates of the social cost. Since savings and investment decisions are not modeled, social costs associated with capital stock changes are also not considered.

E.2.1 Industry Sectors and Benchmark Data Set

The multimarket model includes 100 industries. For the benchmark year, the model uses information from OAQPS's computable general equilibrium model's balanced social accounting matrix (SAM) and the following accounting identity holds:

$$\text{Output} + \text{Imports} = \text{Consumption} + \text{Investment} + \text{Government} + \text{Exports} \qquad \text{(E.1)}$$

If we abstract and treat each industry as a national market, the identity represents the prepolicy (baseline) market-clearing condition, or benchmark "equilibrium"; supply equals demand in each market. In Table E-1, we identify the 100 industries for the multimarket model; Table E-2 provides the 2015 benchmark data set. Since the benchmark data are reported in value terms, we also use the common "Harberger convention" and choose units where are all prices are one in the benchmark equilibrium (Shoven and Whalley, 1995).

Table E-1. Industry Sectors Included in Multimarket Model

Industry Label	*Description*	*Representative NAICS*[a]
Energy Industries		
COL	Coal	2121
CRU	Crude Oil Extraction	211111 (exc. nat gas)
ELE	Electric Generation	2211
GAS	Natural Gas	211112 2212 4862
OIL	Refined Petroleum	324
Nonmanufacturing		
AGR	Agricultural	11
MIN	Mining	21 less others
CNS	Construction	23
Manufactured Goods		
Food, beverages, and textiles		
ANM	Animal Foods	3111
GRN	Grain Milling	3112
SGR	Sugar	3113
FRU	Fruits and Vegetables	3114
MIL	Dairy Products	3115
MEA	Meat Products	3116
SEA	Seafood	3117
BAK	Baked Goods	3118
OFD	Other Food Products	3119
BEV	Beverages and Tobacco	312
TEX	Textile Mills	313
TPM	Textile Product Mills	314
WAP	Wearing Apparel	315
LEA	Leather	316
Lumber, paper, and printing		
SAW	Sawmills	3211
PLY	Plywood and Veneer	3212
LUM	Other Lumber	3219
PAP	Pulp and Paper Mills	3221
CPP	Converted Paper Products	3222
PRN	Printing	323
Chemicals		
CHM	Chemicals and Gases	3251
RSN	Resins	3252
FRT	Fertilizer	3253
MED	Drugs and Medicine	3254
PAI	Paints and Adhesives	3255
SOP	Soap	3256
OCM	Other Chemicals	3259

(continued)

Table E-1. Industry Sectors Included in Multimarket Model (continued)

Industry Label	*Description*	*Representative NAICS*[a]
Plastics and Rubber		
PLS	Plastic	3261
RUB	Rubber	3262
Nonmetallic Minerals		
CLY	Clay	3271
GLS	Glass	3272
CEM	Cement	3273
LIM	Lime and Gypsum	3274
ONM	Other Non-Metallic Minerals	3279
Primary Metals		
I_S	Iron and Steel	3311 3312 33151
ALU	Aluminum	3313 331521 331524
OPM	Other Primary Metals	3314 331522 331525 331528
Fabricated Metals		
FRG	Forging and Stamping	3321
CUT	Cutlery	3322
FMP	Fabricated Metals	3323
BOI	Boilers and Tanks	3324
HRD	Hardware	3325
WIR	Springs and Wires	3326
MSP	Machine Shops	3327
EGV	Engraving	3328
OFM	Other Fabricated Metals	3329
Machinery and Equipment		
CEQ	Construction and Agricultural Equipment	3331
IEQ	Industrial Equipment	3332
SEQ	Service Industry Equipment	3333
HVC	HVAC Equipment	3334
MEQ	Metalworking Equipment	3335
EEQ	Engines	3336
GEQ	General Equipment	3339
Electronic Equipment		
CPU	Computers	3341
CMQ	Communication Equipment	3342
TVQ	TV Equipment	3343
SMI	Semiconductor Equipment	3344
INS	Instruments	3345
MGT	Magnetic Recording Equipment	3346
LGT	Lighting	3351
APP	Appliances	3352

(continued)

Table E-1. Industry Sectors Included in Multimarket Model (continued)

Industry Label	*Description*	*Representative NAICS*[a]
Electronic Equipment (continued)		
ELQ	Electric Equipment	3353
OEQ	Other Electric Equipment	3359
Transportation Equipment		
M_V	Motor Vehicles	3361
TKB	Truck Bodies	3362
MVP	Motor Vehicle Parts	3363
ARC	Aircraft	3364
R_R	Rail Cars	3365
SHP	Ships	3366
OTQ	Other Transport Equipment	3369
Other		
FUR	Furniture	337
MSC	Miscellaneous Manufacturing	339
Services		
Wholesale and Retail Trade		
WHL	Wholesale Trade	42
RTL	Retail Trade	44–45
Transportation Services		
ATP	Air Transportation	481
RTP	Railroad Transportation	482
WTP	Water Transportation	483
TTP	Freight Truck Transportation	484
PIP	Pipeline Transport	486
OTP	Other Transportation Services	485 487 488
Other Services		
INF	Information	51
FIN	Finance and Insurance	52
REL	Real Estate	53
PFS	Professional Services	54
MNG	Management	55
ADM	Administrative Services	56
EDU	Education	61
HLT	Health Care	62
ART	Arts	71
ACM	Accommodations	72
OSV	Other Services	81
PUB	Public Services	92

[a] NAICS = North American Industry Classification System. Industry assignments are based on data used in the EMPAX-modeling system, which relies on the commodity code system used in IMPLAN.

Table E-2. 2015 Benchmark Data Set (billion 2007$)

Industry Label	*Industry Description*	*Output*	*Imports*	*Consumption*	*Investment and Government*	*Exports*
ACM	Accommodations	$940	$7	$919	$20	$8
ADM	Administrative Services	$923	$39	$885	$72	$5
AGR	Agricultural	$349	$71	$390	$6	$25
ALU	Aluminum	$81	$21	$88	$4	$10
ANM	Animal Foods	$50		$41	Less than $1	$10
APP	Appliances	$34	$26	$48	$8	$4
ARC	Aircraft	$257	$57	$68	$116	$129
ART	Arts	$286		$276	$3	$7
ATP	Air Transportation	$174	$34	$98	$30	$80
BAK	Baked Goods	$68	$4	$69	$3	Less than $1
BEV	Beverages and Tobacco	$157	$62	$217	$1	$1
BOI	Boilers and Tanks	$35	$3	$22	$10	$5
CEM	Cement	$74		$68	$4	$3
CEQ	Construction and Agricultural Equipment	$95	$31	$61	$42	$23
CHM	Chemicals and Gases	$355	$124	$409	$12	$58
CLY	Clay	$12	$6	$14	$1	$3
CMQ	Communication Equipment	$96	$43	$60	$57	$23
CNS	Construction	$1,393	$107	$816	$684	Less than $1
COL	Coal	$48	$2	$46		$4
CPP	Converted Paper Products	$60	$2	$48	$7	$7
CPU	Computers	$193	$85	$171	$52	$54
CRU	Crude Oil Extraction	$75	$213	$289		
CUT	Cutlery	$13	$6	$11	$6	$3
EDU	Education	$1,122		$296	$810	$15
EEQ	Engines	$46	$18	$37	$8	$20
EGV	Engraving	$26		$11	$6	$9
ELE	Electric Generation	$339	Less than $1	$304	$35	Less than $1
ELQ	Electric Equipment	$46	$21	$31	$22	$14
FIN	Finance and Insurance	$2,345	$157	$2,308	$51	$144
FMP	Fabricated Metals	$85	$4	$77	$9	$3
FRG	Forging and Stamping	$25	Less than $1	$22	$1	$2
FRT	Fertilizer	$53	$6	$40	$5	$14
FRU	Fruits and Vegetables	$82	$14	$85	$5	$6
FUR	Furniture	$78	$42	$104	$14	$2

(continued)

Table E-2. 2015 Benchmark Data Set (billion 2007$) (continued)

Industry Label	*Industry Description*	*Output*	*Imports*	*Consumption*	*Investment and Government*	*Exports*
GAS	Natural Gas	$150	$34	$170	$7	$7
GEQ	General Equipment	$72	$41	$62	$31	$20
GLS	Glass	$37	Less than $1	$21	$3	$12
GRN	Grain Milling	$86	$10	$83	$2	$11
HLT	Health Care	$2,154		$2,108	$22	$24
HRD	Hardware	$10	$5	$6	$4	$4
HVC	HVAC Equipment	$46	$12	$36	$13	$8
I_S	Iron and Steel	$156	$53	$181	$12	$16
IEQ	Industrial Equipment	$35	$18	$21	$18	$15
IFN	Information	$1,502	$84	$1,409	$162	$13
INS	Instruments	$145	$47	$89	$64	$38
LEA	Leather	$4	$25	$28	Less than $1	$1
LGT	Lighting	$16	$15	$23	$7	$2
LIM	Lime and Gypsum	$9		$2	$1	$7
LUM	Other Lumber	$57	$3	$45	$12	$3
M_V	Motor Vehicles	$304	$180	$346	$86	$52
MEA	Meat Products	$193	$11	$189	$5	$10
MED	Drugs and Medicine	$318	$131	$379	$22	$49
MEQ	Metalworking Equipment	$30	$13	$20	$17	$6
MGT	Magnetic Recording Equipment	$19	$2	$15	$3	$4
MIL	Dairy Products	$96	$4	$94	$5	$2
MIN	Mining	$65	$3	$39	$15	$13
MNG	Management	$560	$10	$453	Less than $1	$116
MSC	Miscellaneous Manufacturing	$213	$134	$221	$60	$65
MSP	Machine Shops	$48	$2	$40	$7	$4
MVP	Motor Vehicle Parts	$246	$92	$254	$19	$64
OCM	Other Chemicals	$56	$2	$28	$11	$19
OEQ	Other Electric Equipment	$43	$22	$38	$10	$16
OFD	Other Food Products	$102	$9	$102	$2	$7
OFM	Other Fabricated Metals	$71	$35	$64	$27	$15
OIL	Refined Petroleum	$650	$171	$728	$19	$74
ONM	Other Non-Metallic Minerals	$18	$7	$22	$1	$3
OPM	Other Primary Metals	$51	$34	$66	$3	$16
OSV	Other Services	$2,628		$1,676	$602	$351

(continued)

Table E-2. 2015 Benchmark Data Set (billion 2007$) (continued)

Industry Label	*Industry Description*	*Output*	*Imports*	*Consumption*	*Investment and Government*	*Exports*
OTP	Other Transportation Services	$350		$300	$27	$23
OTQ	Other Transport Equip	$26	$9	$17	$11	$7
PAI	Paints and Adhesives	$44	$2	$36	$3	$7
PAP	Pulp and Paper Mills	$151	$25	$154	$6	$16
PFS	Professional Services	$2,439	$87	$2,002	$490	$34
PIP	Pipeline Transport	$44	$101	$50	Less than $1	$95
PLS	Plastic	$173	$19	$169	$5	$17
PLY	Plywood and Veneer	$26	$11	$35	$1	$2
PRN	Printing	$57	$1	$39	$11	$7
PUB	Public Services	$1,248	$54	$406	$896	Less than $1
R_R	Rail Cars	$13	$2	$7	$3	$5
REL	Real Estate	$3,165	$2	$2,975	$111	$81
RSN	Resins	$133	$29	$117	$7	$38
RTL	Retail Trade	$1,688	$58	$1,652	$82	$12
RTP	Railroad Transportation	$79	Less than $1	$49	$7	$23
RUB	Rubber	$45	$24	$43	$17	$10
SAW	Sawmills	$40	$12	$49	$1	$3
SEA	Seafood	$14	$4	$16	$1	$1
SEQ	Service Industry Equipment	$38	$31	$30	$31	$9
SGR	Sugar	$38	$7	$40	$2	$3
SHP	Ships	$43	$6	$15	$25	$8
SMI	Semiconductor Equipment	$188	$85	$197	$14	$61
SOP	Soap	$100	$6	$89	$4	$14
TEX	Textile Mills	$28	$11	$32	$1	$6
TKB	Truck Bodies	$67	$12	$39	$25	$15
TPM	Textile Product Mills	$26	$18	$37	$3	$4
TTP	Freight Truck Transportation	$337	$49	$295	$37	$54
TVQ	TV Equipment	$24	$46	$63	$4	$3
WAP	Wearing Apparel	$23	$90	$112	$1	Less than $1
WHL	Wholesale Trade	$1,535	$37	$1,219	$174	$178
WIR	Springs and Wires	$7		$2	$1	$3
WTP	Water Transportation	$50		$15	$13	$22

E.2.2 Economic Behavior

E.2.2.1 U.S. Supply

In a postpolicy scenario (e.g. a MACT or NSPS such as those in the Toxics Rule), industry responds to changes in the new market-clearing "net" price for the good or service sold:

$$\%\Delta\text{"net" price} = \%\Delta \text{ market price} - \%\Delta \text{ direct costs} - \%\Delta \text{ indirect costs} \quad \text{(E.2)}$$

The %Δ direct costs are approximated using the IPM cost analysis and baseline value of output. For example, with the $11 billion increase in compliance costs for the electricity sector (ELE), IPM projects a 3.77 percent increase in the retail price of electricity as mentioned in Chapter 8 of this RIA. For the electric power sector (EPS), percentage change in direct costs would be represented in the model as follows:

$$\%\Delta \text{ direct costs} = 3.77\% \quad \text{(E.3)}$$

To ensure the market-clearing electricity price matches IPM results, we adjust the supply elasticity to reflect a horizontal supply function (i.e. supply is infinitely elastic near market equilibrium).

The multimarket model simultaneously considers how the policy influences other industry supply functions (via changes in energy and other intermediate material prices). As a result, the multimarket model can provide additional information about how policy costs (higher electricity prices) are transmitted through the economy in the short run. As shown in Figure E-1, the higher electricity prices provide other industries with incentives to alter production rates at current market prices; market prices must rise to maintain the original prepolicy production levels (Q). As shown in Figure E-2, the other indirect cost change provides the industry with additional incentives to alter production rates at current market prices.

The %Δ indirect effects associated with each input are approximated using an input "use" ratio and the price change that occurs in the input market.

$$\%\Delta \text{ indirect costs} = \text{input use ratio} \times \%\Delta \text{ input price} \quad \text{(E.4)}$$

The social accounting matrix provides an internally consistent estimate of the use ratio and describes the dollar amount of an input that is required to produce a dollar of output. Higher ratios suggest strong links between industries, while lower ratios suggest weaker links. Given the short time horizon such as that for this analysis with a compliance year of 2015, we assume the

input use ratio is fixed and cannot adjust their input mix; this is a standard assumption in public and commercial input-output (IO) and SAM multiplier models (Berck and Hoffmann, 2002). Morgenstern and colleagues (2004) and Ho and colleagues (2008) also use this assumption when examining near-term effects of environmental policy.

Figure E-1. Higher Electricity Prices Reduce Other Sector Production Rates at Benchmark Prices

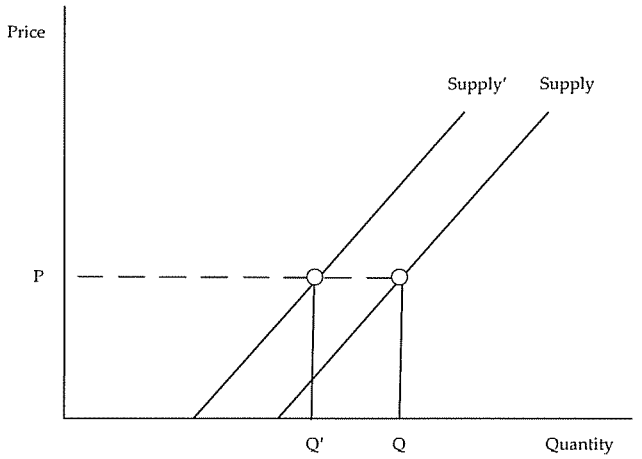
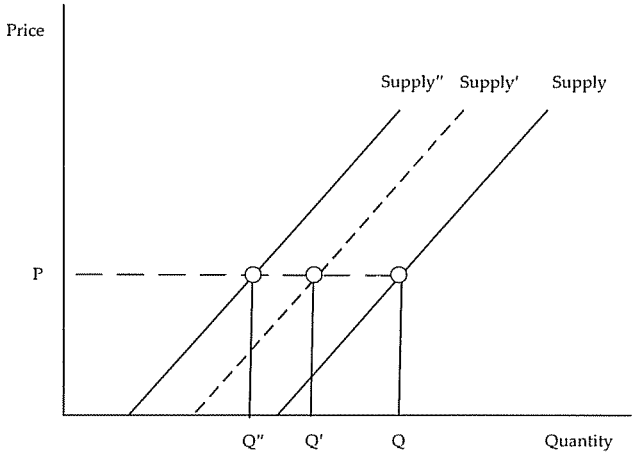


Figure E-2. Indirect Costs Further Reduce Production Rates at Benchmark Prices



Following guidance in the OAQPS economic resource manual (EPA, 1999), we use a general form for the U.S. industry supply function:

$$Q'_g = b\left(P'_g - t - \sum_{i=1}^{n} \alpha_{gi}(P'_i - P_i)\right)^{\varepsilon_g} \quad \text{(E.5)}$$

where

Q'_g = with-policy supply quantity (g)
b = calibrated scale parameter for the supply price relationship
P'_g = with-policy price for output (g)
t = direct compliance costs per unit of supply
α_{gi} = input use ratio (g using input i)
P'_i = with-policy input (i) price
P_i = benchmark input (i) price
ε_g = price elasticity of supply for output (g)

The key supply parameter that controls the industry production adjustments is the price elasticity of supply (ε_g). To our knowledge, there is no existing empirical work that estimates short-run supply elasticities for all industry groups used in the multimarket model. As a result, we assume the U.S. supply elasticities are a function of econometrically estimated rest-of-world (ROW) export supply elasticities (see discussion in the next section). We report the values currently available in the model in Table E-5.

E.2.2.2 International Competition

International competition is captured by a single ROW supply function:

$$Q'_g = c\left(P'_g\right)^{\varepsilon_g^{ROW}} \tag{E.6}$$

where

Q'_g = with-policy supply quantity (g)
c = calibrated scale parameter for the supply and price relationship
P'_g = with-policy U.S. price for output (g)
ε_g^{ROW} = price elasticity of supply of goods from the ROW to the United States (imports) (g)

The key supply parameter that controls the ROW supply adjustments is the price elasticity of supply (ε_g^{ROW}). We obtained these estimates for a variety of industry groups from a recently published article by Broda and colleagues (2008b).

E.2.2.3 Price Elasticity of Supply: Rest of World (ROW)

Broda and colleagues (2008a and 2008b) provide an empirical basis for the multimarket model supply elasticities. Broda et al. provide over 1,000 long-run trade elasticities that RTI organized to be comparable with the 100-sector model. The first step was to match the Harmonized Trade System (HS) elasticities estimated in the article to the appropriate NAICS

codes. Many of the HS codes correspond with a detailed NAICS codes (5- and 6-digit level), while the multimarket sector industries typically correspond with more aggregated sectors (NAICS 2-, 3-, or 4-digit levels). To adapt these labels to our model, we combined the 5- and 6-digit NAICS under their 3- and 4-digit codes and calculated an average elasticity value for codes that fell within the multimarket model's aggregate industrial sectors.[1] This gives a crude way to account for the variety of products detailed in the original data set. We also restricted the long-run elasticity sample to those that Broda et al. classify as "medium" and "low" long-run categories; these categories tend to have lower elasticity values that are more likely to be consistent with the multimarket model's modeling horizon (i.e., in the short run, importers are likely to have less flexibility to respond to price changes implying the elasticities are low rather than high).[2]

Our ideal preference was to use an exact 3- or 4-digit match from the medium category if one was available. If the multimarket model had a 4-digit code for which there was no direct match, we aggregated up a level and applied the relevant 3-digit elasticity. If a multimarket code was not covered in the medium set of elasticities, we used the low elasticity category. This method was sufficient for mapping the majority of the sectors in the model. After applying our inverse elasticity values to the multimarket sectors, we calculated the inverse of the value to arrive at the actual supply elasticity. Since Broda et al.'s article focused on industrial production goods, some of the multimarket sectors were not covered in the elasticity data. These sectors included mainly service industries, transportation, and energy sources.

[1] Given Broda et al.'s research design, the parameter estimates reported are inverse export supply elasticities. For example, a reported parameter estimate for inverse export supply elasticity of 1.6 would imply a ROW supply elasticity of 1/1.6, or 0.6. A one percent increase in the domestic price lead to an 0.6 increase in the volume of goods supplied (i.e., exported) to the U.S. by other countries (p. 2043).

[2] Broda et al.'s intent was to use these categories to describe or proxy for domestic market power.

Table E-5. Supply Elasticities

Industry Label	*Industry Description*	*Rest of World (ROW)*	*U.S.*
ACM	Accommodations	0.7	0.7
ADM	Administrative Services	0.7	0.7
AGR	Agricultural	1.0	1.0
ALU	Aluminum	0.8	0.5
ANM	Animal Foods	1.1	0.8
APP	Appliances	0.9	0.8
ARC	Aircraft	0.9	0.6
ART	Arts	0.7	0.7
ATP	Air Transportation	0.7	0.7
BAK	Baked Goods	0.8	0.7
BEV	Beverages and Tobacco	2.9	2.9
BOI	Boilers and Tanks	1.1	0.8
CEM	Cement	0.9	0.7
CEQ	Construction and Agricultural Equipment	0.8	0.6
CHM	Chemicals and Gases	1.1	0.8
CLY	Clay	0.8	0.6
CMQ	Communication Equipment	2.5	1.0
CNS	Construction	0.7	0.7
COL	Coal	2.2	2.2
CPP	Converted Paper Products	0.9	0.7
CPU	Computers	1.0	0.7
CRU	Crude Oil Extraction	3.7	3.7
CUT	Cutlery	1.4	1.1
EDU	Education	0.7	0.7
EEQ	Engines	1.2	1.0
EGV	Engraving	1.1	0.8
ELE	Electric Generation	[a]	[a]
ELQ	Electric Equipment	0.8	0.6
FIN	Finance and Insurance	0.7	0.7
FMP	Fabricated Metals	1.2	1.1
FRG	Forging and Stamping	1.6	1.5

(continued)

Table E-5. Supply Elasticities (continued)

Industry Label	*Industry Description*	*Rest of World (ROW)*	*U.S.*
FRT	Fertilizer	1.0	0.7
FRU	Fruits and Vegetables	1.0	0.7
FUR	Furniture	1.9	1.9
GAS	Natural Gas	12.2	12.2
GEQ	General Equipment	1.0	0.7
GLS	Glass	0.8	0.6
GRN	Grain Milling	1.7	1.5
HLT	Health Care	0.7	0.7
HRD	Hardware	1.1	0.8
HVC	HVAC Equipment	0.9	0.6
I_S	Iron and Steel	1.0	0.6
IEQ	Industrial Equipment	0.9	0.6
INF	Information	0.7	0.7
INS	Instruments	0.9	0.6
LEA	Leather	0.9	0.7
LGT	Lighting	1.1	0.7
LIM	Lime and Gypsum	0.9	0.7
LUM	Other Lumber	0.9	0.7
M_V	Motor Vehicles	1.3	0.7
MEA	Meat Products	1.2	3.9
MED	Drugs and Medicine	1.3	1.0
MEQ	Metalworking Equipment	0.7	0.5
MGT	Magnetic Recording Equipment	1.0	0.7
MIL	Dairy Products	1.1	0.9
MIN	Mining	2.2	2.2
MNG	Management	0.7	0.7
MSC	Miscellaneous Manufacturing	1.0	0.8
MSP	Machine Shops	1.1	0.8
MVP	Motor Vehicle Parts	0.9	0.6
OCM	Other Chemicals	1.1	0.6
OEQ	Other Electric Equipment	1.0	0.7
OFD	Other Food Products	1.1	0.7

(continued)

Table E-5. Supply Elasticities (continued)

Industry Label	*Industry Description*	*Rest of World (ROW)*	*U.S.*
OFM	Other Fabricated Metals	0.9	0.6
OIL	Refined Petroleum	1.0	0.7
ONM	Other Non-metallic Minerals	1.5	0.7
OPM	Other Primary Metals	0.7	0.5
OSV	Other Services	0.7	0.7
OTP	Other Transportation Services	0.7	0.7
OTQ	Other Transport Equipment	1.0	0.7
PAI	Paints and Adhesives	1.0	0.7
PAP	Pulp and Paper Mills	1.1	0.7
PFS	Professional Services	0.7	0.7
PIP	Pipeline Transport	2.0	2.0
PLS	Plastic	1.0	0.7
PLY	Plywood and Veneer	1.3	1.3
PRN	Printing	1.0	0.7
PUB	Public Services	0.7	0.7
R_R	Rail Cars	1.8	0.7
REL	Real Estate	0.7	0.7
RSN	Resins	1.0	0.7
RTL	Retail Trade	0.7	0.7
RTP	Railroad Transportation	0.7	0.7
RUB	Rubber	1.3	1.1
SAW	Sawmills	0.8	0.6
SEA	Seafood	1.1	0.8
SEQ	Service Industry Equipment	0.8	0.6
SGR	Sugar	1.1	0.8
SHP	Ships	1.0	0.7
SMI	Semiconductor Equipment	1.2	1.0
SOP	Soap	0.8	0.6
TEX	Textile Mills	1.0	0.7
TKB	Truck Bodies	3.2	3.1
TPM	Textile Product Mills	0.8	0.6
TTP	Freight Truck Transportation	0.7	0.7
TVQ	TV Equipment	5.8	5.4

(continued)

Table E-5. Supply Elasticities (continued)

Industry Label	*Industry Description*	*Rest of World (ROW)*	*U.S.*
WAP	Wearing Apparel	1.2	0.8
WHL	Wholesale Trade	0.7	0.7
WIR	Springs and Wires	1.9	0.8
WTP	Water Transportation	0.7	0.7

[a] For this analysis, EPA adjusted the domestic supply elasticity parameter to approximate a horizontal market supply function. This allows the multi-market model to replicate the predicted retail price changes estimated by IPM.

Note: RTI mapped Broda et al. data for their industry aggregation to the multimarket model's 100 industries. Domestic supply elasticities are typically assumed to be within one standard deviation of the sample of supply elasticities used for the ROW. In selected cases where this information is not available, the U.S. supply elasticity is set equal to the ROW.

Source: Broda, C., N. Limao, and D. Weinstein. 2008a. "Export Supply Elasticities." http://faculty.chicagobooth.edu/christian.broda/website/research/unrestricted/TradeElasticities/TradeElasticities.html. Accessed September 2009.

In order to fill these gaps, we turned to the source substitution elasticities from Purdue University's Global Trade Analysis Project (GTAP).[1] Although the elasticities in the GTAP model are a different type of international trade elasticity and cannot be directly applied in the multimarket model (e.g., they are based on the Armington structure[2]), the parameters provide us with some additional information about the relative trade elasticity differences between industry sectors. To use the GTAP information to develop assumptions about the multimarket model sectors with missing elasticities, we chose a base industrial sector (iron and steel) for which we had parameter value from Broda et al. Next, we developed industry-specific ratios for missing industries using the corresponding GTAP sector trade elasticities and the GTAP iron and steel sector. We multiplied the resulting ratio by the Broda et al. iron and steel parameter (1.0). For example, the GTAP trade elasticity for coal (6.10) is approximately 2.2 times the trade elasticity for iron and steel (2.95). As a result, the multimarket import supply elasticity for coal is computed as 2.2 (2.2 x 1.0).

F.2.2.4 Price Elasticity of Supply: United States

We also used Broda et al.'s elasticities to derive a set of domestic supply elasticities for the model. We have assumed that a product's domestic supply would be equal to or less elastic

[1] See Chapter 14 of the GTAP 7 Database Documentation for the full description of the parameters at https://www.gtap.agecon.purdue.edu/resources/download/4184.pdf; see Table 14.2 for elasticities.

[2] Detailed documentation of the entire GTAP 7 Database is available at https://www.gtap.agecon.purdue.edu/databases/v7/v7_doco.asp. The GTAP also uses a unique system of categorizing commodities that does not match the NAICS or HS system exactly.

than other countries' supply of imports. When we aggregated and averaged the original elasticities to the 3- and 4-digit NAICS level for our foreign supply elasticities, we also calculated the standard deviation of each 3- and 4-digit NAICS sample. By adding the standard deviation to the corresponding foreign supply and then taking the inverse, we were able to calculate a domestic supply elasticity for each sector that was lower than its foreign counterpart while maintaining the structure of the original elasticities. For sectors in which no standard deviation was available,[1] we used professional judgment to apply the closest available substitute from a similar industry. Without a comparable way of scaling our foreign elasticities for the sectors in which we used the GTAP elasticities, we elected to keep the domestic and foreign supply elasticities the same.

E.2.2.5 Demand

Uses for industry output are divided into three groups: investment/government use, domestic intermediate uses, and other final use (domestic and exports). Given the short time horizon, investment/government does not change. Intermediate use is determined by the input use ratios and the industry output decisions.

$$Q_i' = \alpha_{gi} Q_g' \tag{E.7}$$

Q_i' = with-policy input demand quantity (i)
α_{gi} = input use ratio (g using input i)
Q_g' = with-policy output quantity (g)

Other final use does respond to market price changes. Following guidance in the OAQPS economic resource manual (EPA, 1999), we use a general form for the U.S. industry demand function:

$$Q_g' = a\left(P_g'\right)^{\eta_g} \tag{E.8}$$

where

Q_g' = with-policy demand quantity (g)
a = calibrated scale parameter for the demand and price relationship
P_g' = with-policy price for output (g)
η_g = price elasticity of demand (g)

[1]No standard deviations were calculated for the 3- and 4-digit codes that had only one observation (i.e., Broda et al.'s model used the exact 3- or 4-digit code).

The key parameter that controls consumption adjustments is the price elasticity of demand (η_g). To approximate the response, we use demand elasticities reported in Ho, Morgenstern, and Shih (2008). To estimate the demand elasticities, Ho, Morgenstern, and Shih used a CGE model[1] and simulate the effects of placing a small tax on output and recording the quantity change. The general equilibrium quantity change associated with the tax considers all price and income changes that led to the quantity change. Table E-6 reports the values taken from Ho, Morgenstern, and Shih (2008) that are currently used for demand responses of other final uses (domestic and exports).

The current version of the multimarket model does not currently consider the consequences of exogenous demand shocks to the scale parameter (a) that the policy may bring about. For example, IPM explicitly models changes in fuel use (a switch from coal to natural gas) that utilities may use to meet the proposed Toxics Rule. Increases in natural gas demand subsequently lead to price increases for natural gas.[2] As a result of higher natural gas prices, industries with more intensive natural gas use may shrink while those with less intensive natural gas use may expand. A similar story with opposite effects occurs in the coal market; reduced demand lowers coal prices and may result in surplus loss for the coal industry. The proposed Toxics Rule may also increase the demand for materials for retrofits and increases in the prices of those goods as well as the demand for retrofit equipment; the demand increase will lead to relative expansion and contraction of industries. EPA acknowledges that the current multimarket model does not account for these types of changes in the market demand curves.

E.2.2.6 Model Scope

The multimarket model includes 100 sectors covering energy, manufacturing, and service applications. Each sector's production technology requires the purchase of energy and other intermediate goods made by other sectors included in the model. Linking the sectors in this manner allows the model to trace direct and indirect policy effects across different sectors. Therefore, it is best used when potential economic impacts and equity effects on related markets might be important to stakeholders not directly affected by an environmental policy. However, the model can also be run in single-market partial equilibrium mode to support and provide insights for other types of environmental policies.

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[1] The authors use the Adkins–Garbaccio CGE Model (Adkins, 2006).

[2] However, IPM treats the natural price increase as a cost to the electricity sector, but does not simultaneously consider that higher prices may result in a surplus gain to owners of natural gas reserves.

E.2.2.7 Model Time Horizon

The model is designed to address short-run and transitional effects associated with environmental policy. Production technologies are fixed; the model does not assess substitution among production inputs (labor, energy intermediates, and other intermediates) and assumes each investment cannot be changed during the time frame of the analysis. These issues are better addressed using other frameworks such as CGE modeling. Similarly, government purchases from each sector do not adjust in response to changes in goods/service prices. Although, employment levels (number of jobs) adjust as production levels change, wages are assumed to be fixed.

Table E-6. U.S. Demand Elasticities

Industry Label	*Industry Description*	*Demand Elasticity* η_g
ACM	Accommodations	−0.7
ADM	Administrative Services	−0.7
AGR	Agricultural	−0.8
ALU	Aluminum	−1.0
ANM	Animal Foods	−0.6
APP	Appliances	−2.6
ARC	Aircraft	−2.5
ART	Arts	−0.7
ATP	Air Transportation	−0.8
BAK	Baked Goods	−0.6
BEV	Beverages and Tobacco	−0.6
BOI	Boilers and Tanks	−0.5
CEM	Cement	−0.8
CEQ	Construction and Agricultural Equipment	−1.7
CHM	Chemicals and Gases	−1.0
CLY	Clay	−0.8
CMQ	Communication Equipment	−2.6
CNS	Construction	−0.8
COL	Coal	−0.1
CPP	Converted Paper Products	−0.7
CPU	Computers	−2.6
CRU	Crude Oil Extraction	−0.3
CUT	Cutlery	−0.5
EDU	Education	−0.7
EEQ	Engines	−1.7
EGV	Engraving	−0.5
ELE	Electric Generation	−0.2
ELQ	Electric Equipment	−2.6
FIN	Finance and Insurance	−0.7
FMP	Fabricated Metals	−0.5
FRG	Forging and Stamping	−0.5
FRT	Fertilizer	−1.0
FRU	Fruits and Vegetables	−0.6
FUR	Furniture	−0.7

(continued)

Table E-6. U.S. Demand Elasticities (continued)

Industry Label	*Industry Description*	*Demand Elasticity* η_g
GAS	Natural Gas	−0.3
GEQ	General Equipment	−1.7
GLS	Glass	−0.8
GRN	Grain Milling	−0.6
HLT	Health Care	−0.7
HRD	Hardware	−0.5
HVC	HVAC Equipment	−1.7
I_S	Iron and Steel	−1.0
IEQ	Industrial Equipment	−1.7
INF	Information	−0.7
INS	Instruments	−2.6
LEA	Leather	−1.1
LGT	Lighting	−2.6
LIM	Lime and Gypsum	−0.8
LUM	Other Lumber	−0.7
M_V	Motor Vehicles	−2.5
MEA	Meat Products	−0.6
MED	Drugs and Medicine	−1.0
MEQ	Metalworking Equipment	−1.7
MGT	Magnetic Recording Equipment	−2.6
MIL	Dairy Products	−0.6
MIN	Mining	−0.6
MNG	Management	−0.7
MSC	Miscellaneous Manufacturing	−1.7
MSP	Machine Shops	−0.5
MVP	Motor Vehicle Parts	−2.5
OCM	Other Chemicals	−1.0
OEQ	Other Electric Equipment	−2.6
OFD	Other Food Products	−0.6
OFM	Other Fabricated Metals	−0.5
OIL	Refined Petroleum	−0.1
ONM	Other Non-metallic Minerals	−0.8
OPM	Other Primary Metals	−1.0
OSV	Other Services	−0.7
OTP	Other Transportation Services	−0.8

(continued)

Table E-6. U.S. Demand Elasticities (continued)

Industry Label	*Industry Description*	*Demand Elasticity* η_g
OTQ	Other Transport Equip	−2.5
PAI	Paints and Adhesives	−1.0
PAP	Pulp and Paper Mills	−0.7
PFS	Professional Services	−0.7
PIP	Pipeline Transport	−0.8
PLS	Plastic	−1.0
PLY	Plywood and Veneer	−0.7
PRN	Printing	−0.7
PUB	Public Services	−0.7
R_R	Rail Cars	−2.5
REL	Real Estate	−0.7
RSN	Resins	−1.0
RTL	Retail Trade	−0.7
RTP	Railroad Transportation	−0.8
RUB	Rubber	−1.0
SAW	Sawmills	−0.7
SEA	Seafood	−0.6
SEQ	Service Industry Equipment	−1.7
SGR	Sugar	−0.6
SHP	Ships	−2.5
SMI	Semiconductor Equipment	−2.6
SOP	Soap	−1.0
TEX	Textile Mills	−1.1
TKB	Truck Bodies	−2.5
TPM	Textile Product Mills	−1.1
TTP	Freight Truck Transportation	−0.8
TVQ	TV Equipment	−2.6
WAP	Wearing Apparel	−2.4
WHL	Wholesale Trade	−0.7
WIR	Springs and Wires	−0.5
WTP	Water Transportation	−0.8

Note: RTI assigned an elasticity using the most similar industry from Ho and colleagues' industry aggregation.

Source: Ho, M. S, R. Morgenstern, and J. S. Shih. 2008. "Impact of Carbon Price Policies on USIndustry." RFF Discussion Paper 08-37. Http://Www.Rff.Org/Publications/Pages/Publicationdetails.Aspx?. Publicationid=20680. Accessed August 2009. Table B.6.

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Chapter 10
STATUTORY AND EXECUTIVE ORDER ANALYSES

10.1 Initial Regulatory Flexibility Analysis

The Regulatory Flexibility Act (RFA; 5 U.S.C.§ 601 et seq.), as amended by the Small Business Regulatory Enforcement Fairness Act (Public Law No. 104-121), provides that whenever an agency is required to publish a general notice of proposed rulemaking, it must prepare and make available an initial regulatory flexibility analysis (IRFA), unless it certifies that the proposed rule, if promulgated, will not have a significant economic impact on a substantial number of small entities (5 U.S.C. § 605[b]). Small entities include small businesses, small organizations, and small governmental jurisdictions. An IRFA describes the economic impact of the proposed rule on small entities and any significant alternatives to the proposed rule that would accomplish the objectives of the rule while minimizing significant economic impacts on small entities.

This IRFA has been prepared following EPA's guidance document for preparing initial and final regulatory flexibility analyses.[1]

10.1.1 Reasons why Action is Being Considered

In 2000, EPA made a finding that it was appropriate and necessary to regulate coal- and oil-fired electric utility steam generating units (EGUs) under Clean Air Act (CAA) section 112 and listed EGUs pursuant to CAA section 112(c). On March 29, 2005 (70 FR 15,994), EPA published a final rule (Section 112(n) Revision Rule) that removed EGUs from the list of sources for which regulation under CAA section 112 was required. That rule was published in conjunction with a rule requiring reductions in emissions of mercury from EGUs pursuant to CAA section 111 (Clean Air Mercury Rule (CAMR), May 18, 2005, 70 FR 28606). The Section 112(n) Revision Rule was vacated on February 8, 2008, by the U.S. Court of Appeals for the District of Columbia Circuit. As a result of that vacatur, CAMR was also vacated and EGUs remain on the list of sources that must be regulated under CAA section 112. This action provides EPA's proposed rule in response to the court's decisions. Under authority of CAA section 112, EPA is proposing a national emission standard for hazardous air pollutants (NESHAP) for EGUs that will regulate units at both major and area sources (henceforth referred to as the Toxics Rule).

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[1] See "EPA's *Action Development Process*: Final Guidance for EPA Rulewriters: Regulatory Flexibility Act as amended by the Small Business Regulatory Enforcement Fairness Act." OPEI Regulatory Development Series. November 2006. This can be found on the Internet at http://www.epa.gov/sbrefa/documents/rfaguidance11-00-06.pdf.

1.1) Statement of Objectives and Legal Basis for Proposed Rules

The proposed rule would protect air quality and promote public health by reducing emissions of the HAP. In the December 2000 regulatory determination, EPA made a finding that it was appropriate and necessary to regulate EGUs under CAA section 112. The February 2008 vacatur of the Section 112(n) Revision Rule reverted the status to that of the December 2000 regulatory determination. CAA section 112(n)(1)(A) and the 2000 determination do not differentiate between EGUs located at major versus area sources of HAP. Thus, the NESHAP for EGUs will regulate units at both major and area sources. Major sources of HAP are those that have the potential to emit at least 10 tons per year (tpy) of any one HAP or at least 25 tpy of any combination of HAP.

1.2) Description and Estimate of the Affected Small Entities

For the purposes of assessing the impacts of the proposed Toxics Rule on small entities, a small entity is defined as:

(1) A small business according to the Small Business Administration size standards by the North American Industry Classification System (NAICS) category of the owning entity. The NAICS affected by this proposed rule is 221122 (fossil fuel-fired electric utility steam generating units) and 921150 (fossil fuel-fired electric utility steam generating units in Indian country). The range of small business size standards for electric utilities is 4 billion kilowatt-hours (kWh) of production or less;

(2) A small government jurisdiction that is a government of a city, county, town, 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.

EPA examined the potential economic impacts to small entities associated with this rulemaking based on assumptions of how the affected entities will install control technologies in compliance with the Toxics Rule. This analysis does not examine potential indirect economic impacts associated with the proposed rule, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

EPA used Velocity Suite's Ventyx data as a basis for identifying plant ownership and compiling the list of potentially affected small entities.[1] The Ventyx dataset contains detailed ownership and corporate affiliation information. The analysis focused only on those EGUs affected by the proposed rule, which includes units burning coal, oil, petroleum coke, or coal refuse as the primary fuel, and excludes any combustion turbine units or EGUs burning natural gas. Also, because the rule does not affect combustion units with an equivalent electricity generating capacity up to 25 megawatts (MW), small entities that do not own at least one combustion unit with a capacity greater than 25 MW were removed from the dataset. For the affected units remaining, boiler and generator capacity, heat input, generation, and emissions data were aggregated by owner and then by parent company. Entities with more than 4 billion kWh of annual electricity generation were removed from the list, as were municipal-owned entities with a population greater than 50,000. For cooperatives, investor-owned utilities, and subdivisions that generate less than 4 billion kWh of electricity annually but which may be part of a large entity, additional research on power sales, operating revenues, and other business activities was performed to make a final determination regarding size. Finally, small entities for which the Integrated Planning Model (IPM) does not project generation in 2015 in the base case were omitted from the analysis because they are not projected to be operating and, thus, will not face the costs of compliance with the proposed rule. After omitting entities for the reasons above, EPA identified a total of 83 potentially affected small entities that are affiliated with 102 electric generating units.

1.3) Compliance Cost Impacts

This section presents the methodology and results for estimating the impact of the proposed rule on small entities in 2015 based on the following endpoints:

- annual economic impacts of the proposed Toxics Rule on small entities and
- ratio of small entity compliance cost impacts to revenues from electricity generation.[2]

10.1.2 Methodology for Estimating Impacts of the Toxics Rule on Small Entities

EPA estimated compliance costs of the proposed Toxics Rule as follows:

$$C_{Compliance} = \Delta C_{Operating+Capital} + \Delta C_{Fuel} + \Delta R$$

[1] For more information, please visit www.ventyx.com. This dataset was also a basis for identifying plant ownership and compiling the list of potentially affected small entities for the proposed Transport Rule issued by EPA in July, 2010.

[2] This methodology for estimating small entity impacts has been used in recent EPA rulemakings such as the proposed Transport Rule issued by EPA in July, 2010.

where C represents a component of cost as labeled, and Δ R represents the value of change in electricity generation, calculated as the difference in revenues between the base case and the proposed Toxics Rule.

Based on this formula, compliance costs for a given small entity could either be positive or negative (i.e., cost savings) based on their compliance choices and market conditions. Under the proposed Toxics Rule, some units will forgo some level of electricity generation (and, thus, revenues) to comply and this impact will be lessened on those entities by the projected increase in electricity prices under the MACT scenario (which raises their revenues from the remainder of their sales). On the other hand, some units may increase electricity generation, and coupled with the increase in electricity prices, will see an increase in electricity revenues resulting in lower net compliance costs. If entities are able to increase revenue more than an increase in retrofit and fuel costs, ultimately they will have negative net compliance costs (or savings). Because this analysis evaluates the total costs as a sum of the costs associated with compliance choices as well as changes in electricity revenues, it captures savings or gains such as those described. As a result, what EPA describes as a cost is really more of a measure of the net economic impact of the rule on small entities.

For this analysis, EPA used unit-level IPM parsed outputs to estimate costs based on the parameters above. These impacts were then summed for each small entity, adjusting for ownership share.[1] Net impact estimates were based on the following: changes in operating and capital costs, driven mainly by retrofit installations or upgrades, change in fuel costs, and change in electricity generation revenues under the proposed Toxics Rule relative to the base case. These individual components of compliance cost were estimated as follows:

(1) **Operating and capital costs:** Using the IPM parsed outputs for the base case and the proposed Toxics Rule policy case, EPA identified units that installed one or more pollution control technologies under the proposed rule. The equations for calculating operating and capital costs were adopted from technology assumptions used in EPA's version of IPM (version 4.10). The model calculates the capital cost (in $/MW); the fixed operation and maintenance (O&M) cost (in $/MW-year); and the variable O&M cost (in $/MWh).

[1] Unit-level cost impacts are adjusted for ownership shares for individual small entities, so as not to overestimate burden on each company. If an individual unit is owned by multiple small entities, total costs for that unit to meet the MACT obligations are distributed across all owners based on the percentage of the unit owned by each company. Ownership percentage was estimated based on the Ventyx database.

(2) **Fuel costs:** Fuel costs were estimated by multiplying fuel input (in million British thermal units, MMBtu) by region and fuel prices ($/MMBtu) from IPM. The incremental fuel expenditures under the proposed Toxics Rule were then estimated by taking the difference in fuel costs between the proposed Toxics Rule and the base case.

(3) **Value of electricity generated:** EPA estimated electricity generation by first estimating unit capacity factor and maximum fuel capacity. Unit capacity factor is estimated by dividing fuel input (MMBtu) by maximum fuel capacity (MMBtu). The maximum fuel capacity was estimated by multiplying capacity (MW) * 8,760 operating hours * heat rate (MMBtu/MWh). The value of electricity generated is then estimated by multiplying capacity (MW) * capacity factor * 8,760 * regional-adjusted retail electricity price ($/MWh), for all entities except those categorized as "Private" in Ventyx. For private entities, EPA used wholesale electricity price instead retail electricity price because most of the private entities are independent power producers (IPP). IPPs sell their electricity to wholesale purchasers and do not own transmission facilities and, thus, their revenue was estimated with wholesale electricity prices.

10.1.3 Results

The number of potentially affected small entities by ownership type and potential impacts of the proposed Toxics Rule are summarized in Table 10-1. All costs are presented in 2007 dollars. EPA estimated the annualized net compliance cost to small entities to be approximately $379 million in 2015.

Table 10-1. Projected Impact of the Toxics Rule on Small Entities in 2015

EGU Ownership Type	Number of Potentially Affected Entities	Number of Entities Retiring all Affected Units	Total Net Compliance Costs ($ millions)	Number of Small Entities with Compliance Cost > 1% of Generation Revenues	Number of Small Entities with Compliance Cost > 3% of Generation Revenues
Co-Op	20	2	176.1	15	12
IOU	8	0	51.3	5	5
Municipal	42	1	74.9	29	20
Sub-division	9	0	68.6	6	4
Private*	4	3	7.9	4	4
Total	**83**	**6**	**379**	**59**	**45**

Notes: The total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. About 20 of the 83 total potentially affected small entities are estimated to have cost savings under the MACT policy case (see text above for an explanation).

* Two of the four identified private entities exceed the 3% threshold of incremental costs as a percentage of revenues, while the other two, though not necessarily exceeding the threshold according to EPA's calculations, are considered to be significantly impacted as a result of having to shut down and are listed as such in the table.

Source: ICF International analysis based on IPM modeling results

EPA assessed the economic and financial impacts of the proposed rule using the ratio of compliance costs to the value of revenues from electricity generation, and our results focus on those entities for which this measure could be greater than 1 percent or 3 percent. Of the 83 small entities identified, EPA's analysis shows 59 entities may experience compliance costs greater than 1 percent of base generation revenues in 2015, and 45 may experience compliance costs greater than 3 percent of base revenues. Also, 6 small entities are estimated to have all of their affected units retire. The cost of a unit retiring is estimated as the base case profit that is forgone by not operating under the policy case. Because 45 of the 83 total units, or more than 50 percent, are estimated to incur compliance cost greater than 3 percent of base revenues, EPA has concluded that it cannot certify that there will be no SISNOSE for this proposed rule.[1]

[1] Results for small entities discussed here do not account for the reality that electricity markets are regulated in parts of the country. Entities operating in regulated or cost-of-service markets should be able to recover all of their costs of compliance through rate adjustments.

The separate components of annualized costs to small entities under the proposed Toxics Rule are summarized in Table 10-2. The most significant components of incremental costs to these entities are the increased capital and operating costs for retrofits, followed by changes in electricity revenues.

Table 10-2. Incremental Annualized Costs under the Toxics Rule Summarized by Ownership Group and Cost Category in 2015 ($2007 millions)

EGU Ownership Type	Capital+ Operating Costs ($MM)	Fuel Costs ($MM)	Change in Electricity Revenue ($MM)	Total
	A	B	C	=A+B-C
Co-Op	137.5	13.3	-25.3	**176.1**
IOU	32.5	-0.7	-19.5	**51.3**
Municipal	102.9	11.6	16.4	**74.9**
Sub-division	37.5	-1.0	32.2	**68.6**
Private	5.5	-0.0	-2.4	**7.9**
Total	**316**	**-0.1**	**-63.1**	

Note: Totals may not add due to rounding

Source: ICF International analysis based on IPM modeling results

Capital and operating costs increase across all ownership types, but the direction of changes in electricity revenues vary among ownership types. Municipals experience a net gain in electricity revenues under the proposed Toxics Rule, mainly due to higher electricity prices under the policy case. All other ownership types are estimated to experience a net loss in electricity revenue. The change in electricity revenue takes into account both the profit lost from units that do not operate under the policy case and the difference in revenue for operating units under the policy case. According to IPM results, an estimated 1.1 GW of capacity owned by small entities are considered uneconomical to operate under the policy case, resulting in a net loss of $258 MM (millions 2007$) in profits. On the other hand, many operating units actually increase their electricity revenue due to higher electricity prices under the proposed rule's policy scenario. Excluding retirements, small entities gain about $195 million in electricity revenues over the base case, resulting in a net loss in electricity revenue of about $63 MM across all ownership types

Federal Rules that May Overlap or Conflict with the Proposed Rule

As noted above, the "electric utility steam generating unit" source category includes those units that combust coal or oil for the purpose of generating electricity for sale and distribution through the national electric grid to the public. Because of the definition provided in

CAA section 112(a)(8), there should not be any EGU that is regulated under another CAA section 112 regulation.

Combustion units that burn fossil fuels but do not meet the size or electric distribution requirements of CAA section 112(a)(8) will be covered under the CAA section 112(d) rules for Industrial, Commercial, and Institutional Boilers (Area and Major Source Boiler NESHAPs), which were proposed on June 4, 2010 (75 FR 31896 and 75 FR 32006). Combustion units that burn a solid waste as defined by the Administrator will be covered as solid waste incineration units under CAA section 129. However, combustion units that burn a homogeneous solid waste and are qualifying units and are thus exempt from regulation under CAA section 129 under the provisions of CAA section 129(g)(1)(B) will be covered under the Utility NESHAP if they combust fossil fuel and meet the size and electric distribution requirements of CAA section 112(a)(8); otherwise they will likely be covered under one of the Boiler NESHAPs (final action required by February 21, 2011).

In 2007, EPA revised new source performance standards (NSPS) for EGUs having a heat input capacity greater than 250 million Btu per hour (40 CFR part 60, subpart Da). The NSPS regulates emissions of particulate matter (PM), sulfur dioxide (SO_2), and nitrogen oxides (NO_X) from boilers constructed after June 2007. EPA is currently working on additional revisions to the PM, SO_2, and NO_X emissions limits in subpart Da. Those revisions will be proposed and promulgated along with the Utility NESHAP on March 16, 2011 and November 16, 2011, respectively. Sources subject to the NSPS would also be subject to the Utility NESHAP because those rules regulate sources of HAP whereas the NSPS does not. However, in developing the NESHAP for EGUs, EPA will minimize the monitoring requirements, testing requirements, and recordkeeping requirements to avoid duplicating requirements to the extent possible.

On June 3, 2010 (75 FR 31514), EPA issued a final rule that establishes thresholds for greenhouse gas (GHG) emissions that define when permits under the New Source Review Prevention of Significant Deterioration (PSD) and title V Operating Permit programs are required for new and existing industrial facilities (the Tailoring Rule). The final rule addresses emissions of six GHGs: carbon dioxide (CO_2), methane (CH_4), nitrous oxide (N_2O), hydrofluorocarbons (HFCs), perfluorocarbons (PFCs), and sulfur hexafluoride (SF_6). As of January 2, 2011, large industrial sources, including power plants, are subject to permitting requirements for their GHG emissions if they otherwise are required to obtain a PSD or title V permit due to emissions of other air pollutants.

On December 23, 2010, EPA announced a settlement agreement, subject to CAA section 113(g) public comment, under which it would issue a proposed rule under CAA 111 (b) that includes standards of performance for GHGs for new and modified EGUs as well as issuing a proposed rule under CAA 111(d) that includes emissions guidelines for GHGs from existing EGUs. The rules would establish NSPS for new and modified EGUs and emission guidelines for existing EGUs. In addition to the NSPS requirements established for new and modified sources under section 111(b) of the CAA, for pollutants not regulated under other parts of the CAA, EPA must establish emission guidelines under CAA section 111(d) that States use to develop plans for reducing emissions from existing sources. The guidelines include targets based on demonstrated controls, emission reductions, costs and expected timeframes for installation and compliance, and can be less stringent than the requirements imposed on new sources. Under the agreement, EPA commits to issuing these proposed regulations by July 26, 2011 and, after considering any public comments received concerning the proposed rule(s), a final rule that takes final action with respect to the proposed rule(s) by May 26, 2012. At this time the Agency has not formulated a final approach for regulating GHGs from EGUs; however, there is the potential that compliance with requirements of the NESHAP could result in some existing sources becoming new sources for purposes of the NSPS.

On August 2, 2010 (75 FR 45210), EPA proposed a rule that would require 31 states and the District of Columbia (D.C.) to significantly improve air quality by reducing power plant emissions that contribute to ozone and fine particle pollution in other states (the Clean Air Transport Rule). Specifically, the proposal would require reductions in SO_2 and NO_X emissions that cross state lines. In response to a December 2008 court decision, the proposed Transport Rule would replace EPA's remanded 2005 Clean Air Interstate Rule (CAIR). The Transport Rule is expected to be finalized in July 2011. To the extent that EGUs are located in the final set of states or D.C., they would be subject to the Transport Rule.

Based on the findings from EPA's multi-year study of the Steam Electric Power Generating industry, EPA plans to revise the current effluent guidelines under the Clean Water Act (CWA) that apply to steam electric power plants. EPA evaluated waste streams generated at power plants, including wastewaters from wet flue gas desulfurization (FGD) air pollution control systems, fly ash and bottom ash handling, coal pile runoff, condenser cooling, equipment cleaning, and leachate from landfills and impoundments, but ultimately focused largely on discharges associated with coal ash handling operations and wastewater from FGD systems because these sources comprise a significant fraction of the pollutants discharged by steam

electric power plants. EPA is required by consent decree to propose revised effluent guidelines in July 2012 and to finalize the guidelines in January 2014.

Section 316(b) of the CWA requires EPA to establish best technology available standards to minimize adverse environmental impacts from cooling water intake structures. In developing these standards, EPA divided its effort into three rulemaking phases. Phase I, for new EGU plants using cooling water, was promulgated on December 18, 2001 (66 FR 65255). Minor revisions to the Phase I rule were finalized on June 19, 2003 (68 FR 36749). Phase II, for existing EGU plants that use at least 50 million gallons per day (MGD) of cooling water, was promulgated on July 9, 2004 (69 FR 41576). Those regulations were challenged, and several provisions of the Phase II rule were remanded on various grounds. EPA suspended most of the rule in response to the remand (72 FR 37107, July 9, 2007). On June 16, 2006 (71 FR 35005), EPA promulgated the Phase III regulations covering existing EGU plants using less than 50 MGD of cooling water. Those regulations also were challenged, and EPA requested, and was granted, a partial remand. EPA plans to issue regulations that address both Phase II and III facilities. EPA signed a settlement agreement that requires those regulations to be proposed by March 14, 2011, and promulgated by July 27, 2012.

On June 21, 2010 (75 FR 35128), EPA proposed national rules for the management of coal combustion residuals under the Resource Conservation and Recovery Act (RCRA). Coal combustion residuals, commonly known as coal ash, are residues from the combustion of coal in power plants and are captured by pollution control technologies, like scrubbers. The residues are disposed of in liquid form at surface impoundments and in solid form at landfills. EGUs will be subject to these coal ash specific regulations when they are issued. The date of final action has not yet been determined.

1.4) Description of Significant Alternatives

As required by RFA section 609(b), as amended by the Small Business Regulatory Fairness Act (SBREFA), EPA conducted outreach to small entities and convened a Small Business Advocacy Review (SBAR) Panel (composed of EPA, the Small Business Administration (SBA), and the Office of Management and Budget (OMB)) to obtain the advice and recommendations of small entity representatives (SERs) that potentially would be subject to the requirements of the proposed Toxics Rule. The outreach consisted of meeting with some organizations that represent and include small entities in their membership, including the American Public Power Association (APPA), Edison Electric Institute (EEI), the Utility Air Regulatory Group (UARG), the National Rural Electric Cooperative Association (NRECA), and

the Coal Utilization Research Council (CURC). As part of the SBAR Panel process, EPA conducted outreach with representatives from 18 various small entities that would be affected by the proposed Toxics Rule. The SBAR Panel convened on October 27, 2010. The Panel held a formal outreach meeting/teleconference with SERs on December 2, 2010.The final SBAR Panel report was completed on (February 16, 2011).

The Panel's most significant findings and discussion with respect to each of these items are summarized below. To read the full discussion of the Panel findings and recommendations, see Section 9 of the Panel Report.

Number and Types of Entities Affected

The estimated number of small entities that will be potentially subject to the Utility NESHAP includes 66 small State/local governments and 14 small non-government entities. These numbers reflect additions and deletions to the initial list of potentially impacted small entities as suggested by SERs as appropriate. For an estimate of the type and number of small entities to which the proposed rule will apply, see Section 5 of the Panel Report. The list of potentially affected small entities includes electricity generators. SERs believe that this list should also include distribution cooperatives that own electricity generation and transmission (G&T) cooperatives and that qualify as small entities. SERs stated that the Utility NESHAP will have a direct impact on all electric cooperatives generating and/or distributing coal-based power given the closely interwoven nature of the G&T cooperatives and the distribution cooperatives. The Panel acknowledges that small entity distribution cooperatives that own generation processes would be impacted in some way by the Utility NESHAP because generation processes will be regulated by the standards, but the extent to which small entity distribution cooperatives would be impacted is unclear without more detailed information on these entities.

Recordkeeping, Reporting, and Other Compliance Requirements

In general, SERs recommended that recordkeeping, reporting, and monitoring requirements should be minimized and simplified to the maximum extent possible.

EPA recommendations: EPA panel members recommend that the Agency consider proposing alternative monitoring approaches (e.g., parameter monitoring in lieu of requiring the use of mercury continuous emissions monitoring systems (CEMS), sorbent traps, periodic stack testing, etc.) and consider requiring particulate matter (PM) CEMS only for the largest EGUs or allow use of PM CEMS as an alternative to conducting opacity monitoring and periodic emissions testing. With respect to SERs' suggestion that if PM CEMS are required by the Utility NESHAP, opacity monitoring requirements of other Federal regulations should no longer apply,

EPA panel members recommend that the Agency consider the available alternatives and options to the current opacity provisions.

OMB recommendations: OMB recommends that alternative monitoring approaches (e.g., parameter monitoring in lieu of requiring the use of mercury CEMS, sorbent traps, periodic stack testing) be proposed for small entities and that EPA propose PM CEMS only for the largest EGUs or propose allowing use of PM CEMS as an alternative to conducting opacity monitoring and periodic emissions testing.

SBA recommendations: SBA agrees that EPA should consider relevant factors identified by the SERs in developing this rulemaking, but it does not believe that the Panel has sufficient information to make recommendations beyond EPA's existing obligations under the RFA or Paperwork Reduction Act. SBA agrees that these are flexibilities worthy of consideration, and perhaps proposal, but without information necessary to evaluate specific regulatory alternatives or the impacts of those decisions on particular small entities or small entities in general, SBA believes that the Panel can make no recommendations as to what specific regulatory options would "accomplish the stated objectives of applicable statutes and which minimize any significant economic impact of the proposed rule on small entities."

10.1.4 Related Federal Rules

SERs asked that EPA consider the impact of competing regulatory requirements and technologies when developing the Utility NESHAP. EPA is currently working on revisions to the PM, SO_2, and NO_X emissions limit in subpart Da. Sources subject to the NSPS would also be subject to the Utility NESHAP because those rules regulate sources of HAP whereas the NSPS does not.

In June 2010, EPA issued a final rule that establishes thresholds for GHG emissions that define when permits under the New Source Review PSD and title V Operating Permit programs are required for new and existing industrial facilities (the Tailoring Rule). Beginning in January 2011, large industrial sources, including power plants, became subject to permitting requirements for their GHG emissions.

On December 23, 2010, EPA announced a settlement agreement under which it would issue rules that will address GHG emissions from fossil fuel-fired power plants. The rules would establish NSPS for new and modified EGUs and emission guidelines for existing EGUs. Under the agreement, EPA commits to issuing proposed regulations by July 26, 2011 and final regulations by May 26, 2012.

In August 2010, EPA proposed a rule that would require 31 states and the District of Columbia (D.C.) to significantly improve air quality by reducing power plant emissions that contribute to ozone and fine particle pollution in other states (the Transport Rule). Specifically, the proposal would require reductions in SO_2 and NO_X emissions that cross state lines. The Transport Rule is expected to be finalized in July 2011. To the extent that EGUs are located in the final set of states or D.C., they would be subject to the Transport Rule. SERs expressed concern regarding what the impact of controlling SO_2 and NO_X emissions as a result of complying with the Transport Rule will do to the level of CO emissions.

Based on the findings from EPA's multi-year study of the Steam Electric Power Generating industry, EPA plans to revise the current effluent guidelines that apply to steam electric power plants. Revised effluent guidelines will be proposed in July 2012 and finalized in January 2014.

As required by section 316(b) of the Clean Water Act (CWA), EPA established best technology available standards to minimize adverse environmental impacts from cooling water intake structures. In developing these standards, EPA divided its effort into three rulemaking phases. Phase I standards, for new EGU plants using cooling water, were finalized in June 2003. Phases II and III standards, which address existing EGU plants that use cooling water, were promulgated in July 2004 and June 2006, respectively. Both regulations were challenged. Several provisions of the Phase II rule were remanded and EPA suspended most of the rule in response to the remand. EPA requested, and was granted, a partial remand of the Phase III rule. EPA signed a settlement agreement that requires regulations for Phase II and III facilities to be proposed by March 14, 2011, and promulgated by July 27, 2012.

In June 2010, EPA proposed national rules for the management of coal ash, which are residues from the combustion of coal in power plants that are captured by pollution control technologies, like scrubbers. EGUs will be subject to these coal ash specific regulations when they are issued.

SBA recommendations: SBA agrees that EPA should consider relevant factors identified by the SERs in development of this rulemaking, including the extent to which other recently proposed or finalized regulatory obligations imposed by EPA will impact small entities or make compliance with this rulemaking more difficult. SBA also agrees that EPA should always avoid duplication of requirements across programs. However, SBA does not believe that the Panel has information necessary make recommendations beyond a restatement of EPA's existing obligations or to evaluate specific regulatory decisions and the impacts of those decisions on

particular small entities or small entities in general. Therefore, SBA believes that the Panel can make no recommendations as to what specific regulatory options would "accomplish the stated objectives of applicable statutes and which minimize any significant economic impact of the proposed rule on small entities."

Panel recommendations: Although the requirements of section 112 of the CAA direct EPA to establish NESHAP for both major and area sources of HAP and prescribe the processes by which the standards are developed, the Panel recommends that the Agency consider the various flexibilities within its discretion in developing the proposed standards. The Panel recommends that the Agency investigate other potential surrogate pollutants for organic HAP in lieu of CO, given the NO_X-CO relationship (i.e., when NO_X emissions are reduced, CO emissions may increase). In developing the NESHAP for EGUs, the Panel recommends that the Agency avoid duplicating requirements to the fullest extent possible in order to minimize unnecessary costs.

10.1.5 Regulatory Flexibility Alternatives

MACT Floors and Variability

SERs raised four issues with respect to determining MACT floors and assessing variability: (1) pollutant-by-pollutant ranking approach, (2) pollutants to be regulated, (3) floor determination methodology for existing units, and (4) assessment of emissions variability, including periods of startup and shutdown, and fuel, performance, and load variability. A description of each of these issues along with the Panel recommendations is presented in succession below.

SERs stated that the end result of determining a MACT floor for each HAP or HAP surrogate (a pollutant-by-pollutant approach) for each subcategory of sources is a set of MACT floors that do not represent the emission levels achieved by an actual, best-performing EGU. SERs believe that this methodology for setting MACT floors is inconsistent with the requirements of CAA section 112(d)(3). It was suggested that MACT floors should be established using a facility-wide approach.

EPA recommendation: Consistent with EPA's legal interpretation, EPA panel members recommend that the Agency use the pollutant-by-pollutant approach for determining MACT standards for each HAP or HAP surrogate, while taking into account potential direct conflicts between pollution control technologies.

There are concerns with respect to the suggestion that MACT floors should be established using a facility-wide approach. Determining floors based on a facility-wide approach would lead to least common denominator floors – that is floors reflecting mediocre or no control, rather than performance which, for existing sources, is the average of what the best performing sources have achieved. For example, if the best performing 12 percent of facilities for HAP metals did not control organics as well as a different 12 percent of facilities, the floor for organics and metals would end up not reflecting best performance. This fact pattern has come up in every rule where EPA investigated a facility-wide approach. See, e.g. 75 FR at 54999 (Sept. 10, 2010). Thus, utilizing the single-facility theory proffered by the stakeholders would result in EPA setting the standards at levels that would, for some pollutants, actually be based on emissions limitations achieved by the *worst*-performing unit, rather than the *best*-performing unit, as required by the statute. Moreover, a single-facility approach would require EPA to make value judgments as to which pollutant reductions are most critical in working to identify the single facility that reduces emissions of HAP on an overall best-performing basis.

OMB and SBA recommendation: OMB and SBA recommend that in the proposed rule, EPA seek comment on reasonable alternative approaches to setting the MACT floor, which account for achievement in practice for control of all HAP.

SERs stated their belief that the Utility NESHAP should be limited to mercury control only. They explained that EPA has not determined that emissions of other HAP in the quantities emitted are detrimental to human health or the environment. SERs continue to support EPA's 2004 legal analysis that stated EPA believed it only had authority to set MACT standards for mercury under CAA section 112(d).

EPA recommendation: As to the comment that EPA should only regulate mercury from coal-fired EGUs and nickel from oil-fired EGUs consistent with the reasoning in the proposed NESHAP for these sources that was published on January 30, 2004, EPA panel members note that the Agency never finalized that proposed interpretation, and the Agency has determined that it must establish CAA section 112(d) standards for all HAP emitted from major source EGUs consistent with the statute and case law from the Court of Appeals for the D.C. Circuit. For these reasons, EPA rejects the proposed interpretation set forth in the 2004 proposed rule.

OMB and SBA recommendation: OMB and SBA recommend that in the proposed rule, EPA seek comment on the specific elements of the 2004 legal analysis and how subsequent court decisions affect that 2004 legal analysis.

Panel Recommendation: The Panel recommends that the analysis of impacts be able to distinguish the marginal costs and benefits of each required control technology, in order for the public to distinguish the impacts of regulating mercury from the impacts of regulating other HAPs. It should be noted that EPA cannot, at this point, estimate monetized benefits for HAP reductions other than Hg.

In addition, by focusing on one HAP at a time, SERs believe that the antagonistic effects a given HAP limit will have on other regulated pollutants are missed. Because production of CO during the combustion process is inversely related to NO_X production, it may be difficult to meet a CO limit if NO_X reductions also are required.

Panel Recommendation: The Panel recommends that the Agency investigate other potential surrogate pollutants for organic HAP (e.g., PAH, formaldehyde). The SERs' example of how a pollutant-by-pollutant approach could result in technical infeasibility with respect to CO and NO_X may argue against using CO as the surrogate pollutant for organic HAP.

SERs commented that the MACT floor for existing units should be determined using the entire inventory of EGUs and not using only the units for which EPA has test data.

EPA recommendation: The CAA requires the MACT floor for existing sources be based on the best performing sources. Thus, EPA must be able to show that the best performing units are in fact used to establish the MACT floor. To use the entire inventory of EGUs as the basis for determining the average of the best performing twelve percent of units, EPA must be confident that the EGUs for which data are available are the best performers. EPA panel members recommend that the Agency establish the MACT floors using all the available ICR data that was received to the maximum extent possible consistent with the CAA requirements.

OMB and SBA recommendations: OMB and SBA recommend that EPA establish MACT standards that minimize the burden on small entities. OMB and SBA also recommend that EPA consider, and present for comment, MACT floors based on the best performing 12 percent, rather than the best 12 percent of the data EPA collected. If EPA proposed the latter, OMB and SBA recommend that they clearly explain why the subset of sources for which they have data is representative of the entire set of sources.

SERs asked that EPA consider establishing percent reduction limits as an alternative to complying with an emissions limit as a means of providing small entities flexibility in complying with the NESHAP in addition to providing a means of potentially accounting for variability. SERs expressed concern that periods of startup and shutdown could present problems with

meeting emission limits and suggested that the emissions limits be based on a longer averaging time rather than basing limits on 3-run averages. SERs stated that the three-day stack sampling required by EPA's ICR provides a snapshot of a unit's HAP emissions and is not indicative or representative of the unit's emissions over longer periods of time. SERs pointed out that a critical question is how EPA plans to modify the stack emissions reported during the ICR to account for fuel, performance, and load variability. One SER suggested that use of a longer-term rolling average (i.e., a 12-month minimum rolling average) is necessary in order to account for varying levels of mercury in fuel. Additionally, one SER indicated that a *de minimis* exemption is a regulatory option/small entity flexibility that EPA should consider.

EPA recommendations: EPA is limited in its ability to establish percent reduction limits as an alternative to complying with an emissions limit. Even assuming that EPA can establish percent reduction standards under CAA section 112, to establish such standards, emissions data for the inlet to the EGU and for the stack are necessary. At this time, EPA does not have such data. EPA panel members recommend that the Agency consider the inclusion of percent reduction standards given the legal constraints and the lack of data necessary to establish such standards. Regarding the SERs' concerns with meeting emissions limits during periods of startup and shutdown, EPA panel members recommend that the Agency base the proposed emission limits on reasonable averaging times where appropriate. In determining reasonable averaging times, EPA panel members recommend that in addition to considering performance during periods of startup and shutdown, the Agency also consider fuel and load variability. In addition, EPA panel members recommend that the Agency use all data gathered through the ICR for EGUs that comprise the MACT floor, to the maximum extent possible consistent with the CAA requirements and as appropriate, in order to account for fuel, performance, and load variability. With regard to one SER's request that a *de minimis* exemption be considered, EPA must establish standards for all HAP emitted from major sources consistent with CAA section 112(d) and case law from the U.S. Court of Appeals for the D.C. Circuit.

Panel recommendations: The Panel recommends that EPA propose provisions for emissions averaging between units at a facility and long averaging times to address startup, shutdown, and fuel variability for the proposed emissions limit and, further, that the Agency solicit comment on an appropriate averaging time. The Panel recommends that EPA consider fuel variability when deriving the emissions standards. The Panel recommends that the Agency evaluate whether establishing work practice requirements during periods of startup and shutdown would be consistent with CAA section 112(h) and investigate whether there are technical bases

for establishing separate standards (e.g., work practices or subcategorization) for EGUs below a certain size and what that size threshold is.

Subcategorization

In general, SERs encouraged the broad use of subcategories. SERs commented that EPA should consider subcategorizing EGUs based on fuel type, boiler type, duty cycle, and size. Some SERs requested that EPA consider establishing a subcategory for combined heat and power (CHP) units that meet the definition of EGU (i.e., generate enough electricity). SERs explained that the duty cycles for some coal-fired EGUs are not primarily base-load, as in the past, but may alternate between operating as base-load units and peaking units. Similar comments were not made with regard to consideration of base-load oil-fired EGUs and peaking oil-fired EGUs as separate subcategories.

EPA recommendations: EPA recognizes subcategorization may be necessary and we will consider whether subcategorization is reasonable in light of the data and other information obtained in response to the ICR to the utility industry and the information from the SERs. SERs recommended that EPA consider adopting the following subcategories for EGUs:

- Fuel type
  - North Dakota lignite
  - Gulf Coast lignite
  - Bituminous coal
  - Sub-bituminous coal
  - Blended bituminous/sub-bituminous coal
  - Powder River Basin coal
  - Illinois Basin coal
- Boiler design
  - Units designed to burn coal
  - Units designed to burn oil
  - IGCC units
  - CHP units
  - Units designed to burn multiple fuels

- Unit type
  - Fluidized bed
  - Pulverized coal
  - Wall-fired
  - Tangentially-fired
- Duty cycle
  - Base-load oil-fired units
  - Peaking oil-fired units
  - Base-load coal-fired units
  - Coal-fired units that alternate operating as base-load and peaking
- Boiler class
  - Small entity non-profit providers

EPA and OMB recommendations: EPA panel members and OMB acknowledge that it may not be practicable to adopt all of the proposed subcategories, as there may be substantial overlap between the groups. EPA panel members and OMB recommend that EPA consider these subcategories and adopt a set of standards that is consistent with the CAA and which effectively reduces burden on small entities.

SBA recommendations: SBA agrees that EPA should consider various subcategorization options in developing this rulemaking, but it does not believe that the Panel has sufficient information to recommend a particular subcategorization option that would minimize the significant economic impact of the proposed rule on small entities. While a large number of subcategories may serve to establish standards that minimize the economic impacts on some particular small entities, it could also disadvantage small entities that would otherwise be among the best performing 12 percent of a larger subcategory.

Area Source Standards

SERs suggested that EPA establish separate emission standards for EGUs located at area sources of HAP and that the standards be based on generally available control technology (GACT) as allowed under section 112(d)(5) of the CAA. Specifically, SERs recommended that

EPA establish management practice standards for natural area source EGUs as well as synthetic area source EGUs.[1]

EPA recommendations: EPA panel members recommend that the Agency consider a regulatory approach for EGUs at area sources of HAP based on GACT. Further, EPA panel members recommend that the Agency consider establishing management practices for area source EGUs.

OMB recommendations: OMB recommends that EPA propose a regulatory approach for EGUs at area sources of HAP based on GACT and propose management practices for area source EGUs.

SBA recommendations: SBA agrees that EPA should consider the use of its authority to establish area sources standards for natural and synthetic area sources to the maximum extent permitted by statute, but does not believe that the Panel has sufficient information to recommend a particular regulatory option that would minimize the significant economic impact of the proposed rule on small entities.

Work Practice Standards. SERs recommended that EPA establish work practice standards for major source EGUs. A work practice standard, instead of MACT emission limits, may be proposed if it can be justified under section 112(h) of the CAA that it is not feasible to prescribe or enforce an emission standard (i.e., the application of measurement methodology to a particular class of sources is not practicable due to technological and economic limitations). Specifically, SERs believe it is not feasible to prescribe or enforce an emission standard for control of a HAP emitted at or below the detection limit of the method that was used to collect and analyze HAP emissions. A number of HAP, including a large percentage of the dioxin/furan and non-dioxin organics measurements, are emitted at or below detection limits.

EPA and OMB recommendation: EPA panel members and OMB recommend that the Agency evaluate the availability of work practice standards, in particular with regards to HAP that are emitted at or below the detection limit.

[1] Based on the 2010 national inventory derived from the 2010 ICR data, there are 141 natural or synthetic area source units. Of these 141 units, 23 units are owned in whole or in part by small entities (14 units are owned wholly by single small entities; 9 units are owned by a number of small entities holding small percentage ownership [less than 5%]).

SBA recommendation: SBA recommends that EPA propose work practices standards to the maximum extent permitted by statute. However, the Panel does not have sufficient information to specify which work practices standards can be proposed.

Health-Based Emissions Limits

SERs commented that health based emission limits (HBELs) should be used to the maximum extent possible when facts support their use. Specifically, SERs encouraged EPA to use its CAA section 112(d)(4) authority to set a HBEL for HCl based on its reference concentration for the entire EGU source category.

EPA recommendation: EPA panel members recommend that the Administrator consider her discretionary authority to propose a HBEL for acid gas HAP emissions as a regulatory flexibility option.

OMB and SBA recommendations: OMB and SBA recommend that in the proposed rule, EPA co-propose and seek comment on an HBEL for HAPs to the maximum extent permitted by statute, including, but not limited to, the acid gas HAP. OMB and SBA recommend that in the proposal EPA explain their method for deriving these limits, along with sample calculations.

Potential Adverse Economic Impacts

SERs commented on a number of concerns they have with respect to small entities' ability to comply with the potential requirements of the Utility NESHAP. SERs inquired as to EPA's authority to (1) move the effective date of the standards, (2) determine when implementation begins, (3) allow a phase-in of compliance, and (4) streamline the process for petitioning for a fourth year for purposes of complying with the standards. SERs asked that EPA consider the implications of EGU reliability versus compliance with the Utility NESHAP when establishing the rule's requirements. SERs expressed concern that, depending on the type and stringency of requirements, the regulations could be so expensive that they cause extensive plant retirements and job losses.

SBA recommendation: SBA recommends that EPA propose a streamlined process for granting a fourth year, including aiding small entities in gathering the information necessary to support such a petition, and recommends that EPA develop, in consultation with the Department of Defense and small entities affected by this rule, to develop the information necessary to support a recommendation under section 112(i)(4) of the CAA for consideration by the President.

Panel recommendations: The Panel recommends that the Agency weigh the potential burden of compliance requirements and consider various options for all regulated entities, especially small entities. With respect to dates, EPA does not have the authority to move the effective date of the standards (see CAA section 112(d)(10)), to initially provide more than three years for compliance (see CAA section112(i)), or to allow a phase-in of compliance. The Panel recommends that the Agency investigate the potential for streamlining the process for petitioning for a fourth year for purposes of compliance with the standards and consider the need to invoke the national security exemption under section 112(i)(4) of the CAA. Additionally, the Panel recommends that EPA seek comment in the proposed rule on the potential adverse economic impacts of the rule for small entities and recommendations for mitigating or eliminating these adverse economic impacts on small entities.

Concerns with the Small Business Advocacy Review Process

SERs stated that they do not believe they were provided the opportunity for effective participation in the Federal regulatory process as required by SBREFA. SERs indicated that they were not provided descriptions of significant alternatives to the proposed rule, differing compliance or reporting requirements or timetables that take into account the resources available to small entities. SERs further indicated that there was no pre-meeting to go over information on the rule, there was only one outreach meeting, and SERs were only provided 14 days to prepare written comments. SERs had various suggestions including that EPA schedule additional panel meetings once the Agency has progressed further in its rulemaking preparation, that EPA consider starting over with the SBREFA process, and that EPA request an extension to allow time to (1) adequately analyze lessons learned in the Boiler MACT rule development process, (2) thoroughly analyze the emissions data, (3) continue to meet with utility industry representatives, and (4) consider the range of possible emission control options that would allow for implementation to take place such that the integrity of the Grid, the national economy, and national security will be protected.

EPA recommendation: EPA appreciates the SERs' concerns, but believes it has fulfilled its statutory obligations under SBREFA and has afforded SERs sufficient opportunity to suggest regulatory alternatives, and thus, makes no recommendations to address these concerns. The time constraints of the small business advocacy review process with respect to the Utility NESHAP were explained at the beginning of the process. That is, due to the regulatory schedule there could only be one SER outreach meeting. The nature of the information to be provided was also outlined to the SERs at the start of the process. EPA panel members believe they provided sufficient information to allow SERs to make suggestions concerning regulatory

alternatives (e.g., regarding subcategories, HAP and HAP surrogates, monitoring requirements, control technologies potentially required to meet standards, CAA authorities to establish health-based emission limits and work practice standards) as part of the small business advocacy review process, and the SERs have in fact made many productive suggestions EPA will seriously consider as part of the rulemaking process.

OMB recommendation: Although OMB understands the time constraints imposed on this rulemaking process, we recommend that once EPA has drafted a set of emissions limits for EGUs, they convene another meeting with the SERs to gather insight on the feasibility and achievability of those limits for small entities. To the extent feasible, we recommend this meeting take place before the proposal is issued.

SBA recommendations: SBA agrees with the concerns raised by the SERs in their comments about the adequacy of the information provided to the Panel and the SERs and about the schedule for the Panel. SBA believes that more time is necessary for EPA to develop regulatory options and to share them with the SERs, so that the SERs could provide a more informed comment and better inform the Panel's recommendations.

SBA recommends that EPA request an extension of the regulatory deadlines imposed by the consent decree. The extension should provide enough time for EPA to:

- Analyze fully the results of the ICR and other data necessary to understand the emissions characteristics of the regulated entities;
- Develop a robust range of specific regulatory options;
- Consult with the SERs and provide an additional opportunity for the SERs to provide input on the regulatory options; and
- Allow for the full interagency review required by Executive Order 12866.

10.2 Unfunded Mandates Reform Act (UMRA) Analysis

Title II of the UMRA of 1995 (Public Law 104-4) (UMRA) establishes requirements for federal agencies to assess the effects of their regulatory actions on state, local, and Tribal governments and the private sector. Under Section 202 of the UMRA, 2 U.S.C. 1532, EPA generally must prepare a written statement, including a cost-benefit analysis, for any proposed or final rule that "includes any Federal mandate that may result in the expenditure by State, local, and Tribal governments, in the aggregate, or by the private sector, of $100,000,000 or more ... in

any one year." A "Federal mandate" is defined under Section 421(6), 2 U.S.C. 658(6), to include a "Federal intergovernmental mandate" and a "Federal private sector mandate." A "Federal intergovernmental mandate," in turn, is defined to include a regulation that "would impose an enforceable duty upon State, Local, or Tribal governments," Section 421(5)(A)(i), 2 U.S.C. 658(5)(A)(i), except for, among other things, a duty that is "a condition of Federal assistance," Section 421(5)(A)(i)(I). A "Federal private sector mandate" includes a regulation that "would impose an enforceable duty upon the private sector," with certain exceptions, Section 421(7)(A), 2 U.S.C. 658(7)(A).

Before promulgating an EPA rule for which a written statement is needed under Section 202 of the UMRA, Section 205, 2 U.S.C. 1535, of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and adopt the least costly, most cost-effective, or least burdensome alternative that achieves the objectives of the rule. Moreover, section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective or least burdensome alternative if the Administrator publishes an explanation why that alternative was not adopted.

In a manner consistent with the intergovernmental consultation provisions of Section 204 of the UMRA, EPA carried out consultations with the governmental entities affected by this rule. EPA held meetings with states and Tribal representatives in which the Agency presented its plan to develop a proposal and provided opportunities for participants to provide input as part of the rulemaking process. EPA has also analyzed the economic impacts of the proposed Toxics Rule on government entities and this section presents the results of that analysis. This analysis does not examine potential indirect economic impacts associated with the proposed rule, such as employment effects in industries providing fuel and pollution control equipment, or the potential effects of electricity price increases on industries and households.

Identification of Affected Government Entities

Using Ventyx data, EPA identified state- and municipality-owned utilities and subdivisions that would be affected by the proposed rule. EPA then used IPM parsed outputs to associate these entities with individual generating units. The analysis focused only on EGUs affected by the proposed rule, which includes units burning coal, oil, petroleum coke, or waste coal as the primary fuel, and excludes any combustion turbine units. Entities that did not own at least one unit with a generating capacity of greater than 25 MW were also removed from the dataset because of their exemption from the rule. Finally, government entities for which IPM does not project generation in 2015 under the base case were also exempted from this analysis,

because they are not projected to be operating and thus will not face the costs of compliance with the proposed rule. Based on this, EPA identified 96 state, municipal and sub-divisions affiliated with 169 electric generating units that are potentially affected by the proposed Toxics Rule.

Compliance Cost Impacts

After identifying the potentially affected government entities, EPA estimated the impact of the proposed rule in 2015 based on the following:

- total impacts of compliance on government entities and
- ratio of government entity impacts to revenues from electricity generation.

Methodology for Estimating Impacts of the Toxics Rule on Government Entities

EPA estimated compliance costs of the proposed Toxics Rulemaking as follows:

$$C_{Compliance} = \Delta C_{Operating+Capital} + \Delta C_{Fuel} + \Delta R$$

where C represents a component of cost as labeled, and Δ R represents the retail value of change in electricity generation, calculated as the difference in revenues between the base case and the Toxics Rule.

Based on this formula, compliance costs for a given government entity could either be positive or negative (i.e., cost savings) based on their compliance choices and market conditions. Under the Toxics Rule, some units will forgo some level of electricity generation (and thus revenues) to comply and this impact will be lessened on those entities by the projected increase in electricity prices under the MACT scenario. On the other hand, some units may increase electricity generation, and coupled with the increase in electricity prices, will see an increase in electricity revenues resulting in lower net compliance costs. If entities are able to increase revenue more than an increase in retrofit and fuel costs, ultimately they will have negative net compliance costs (or savings). Because this analysis evaluates the total costs as a sum of the costs associated with compliance choices as well as changes in electricity revenues, it captures savings or gains such as those described. As a result, what EPA describes as a cost is really more of a measure of the net economic impact of the rule on government entities.

For this analysis, EPA used unit-level IPM parsed outputs to estimate costs based on the parameters above. These impacts were then aggregated for each government entity, adjusting for ownership share. Compliance cost estimates were based on the following: changes in capital and

operating costs, change in fuel costs, and change in electricity generation revenues under the proposed rule relative to the base case. These components of compliance cost were estimated as follows:

(1) **Capital and operating costs:** Using the IPM parsed outputs for the base case and the Toxics Rule policy case, EPA identified units that install control technology under the proposed rule and the technology installed. The equations for calculating operating and capital costs were adopted from EPA's version of IPM (version 4.10). The model calculates the capital cost (in $/MW); the fixed operation and maintenance (O&M) cost (in $/MW-year); and the variable O&M cost (in $/MWh)

(2) **Fuel costs:** Fuel costs were estimated by multiplying fuel input (MMBtu) by region and fuel prices ($/MMBtu) from IPM. The change in fuel expenditures under the Toxics Rule was then estimated by taking the difference in fuel costs between the Toxics Rule and the base case.

(3) **Value of electricity generated:** EPA estimated electricity generation by first estimating the unit capacity factor and maximum fuel capacity. Unit capacity factor was estimated by dividing fuel input (MMBtu) by maximum fuel capacity (MMBtu). The maximum fuel capacity was estimated by multiplying capacity (MW) * 8,760 operating hours * heat rate (MMBtu/MWh). The value of electricity generated was then estimated by multiplying capacity (MW) * capacity factor * 8,760 * regional-adjusted retail electricity price ($/MWh).

Results

As was done for the small entities analysis, EPA assessed the economic and financial impacts of the rule using the ratio of compliance costs to the value of revenues from electricity generation, and our results focus on those entities for which this measure could be greater than 1 percent or 3 percent of base revenues. EPA projects that 55 government entities will have compliance costs greater than 1 percent of base generation revenue in 2015, and 37 may experience compliance costs greater than 3 percent of base revenues. Also, one government entity is estimated to have all of its affected units retire. Overall, 17 units owned by government entities retire. It is also worth noting that two-thirds of the net compliance costs shown above are due to lost profits from retirements. More than half of those lost profits arise from retiring two large units, according to EPA modeling.

The separate components of the annualized costs to government entities under the proposed Toxics Rule are summarized in Table 10-3 below. The most significant components of

incremental costs to these entities are the increased capital and operating costs, followed by changes in electricity revenues.

Table 10-3. Incremental Annualized Costs under the Toxics Rule Summarized by Ownership Group and Cost Category ($2007 millions) in 2015

EGU Ownership Type	Capital Costs + Operating Costs($MM)	Fuel Costs ($ MM)	Change in Revenue ($ MM)	Total
	A	B	C	=A+B-C
Sub-Division*	142.9	-2.8	53.3	86.8
State	109.1	-5.3	65.7	38.0
Municipal**	532.3	10.7	97.2	445.8
Total	**784.3**	**2.5**	**216.2**	**571**

Note: Totals may not add due to rounding.

* Sub-divisions are counties, municipalities, school districts, hospital districts, or any other political subdivision receiving electric service from an entity that has implemented customer choice, as defined in Section 31.002, Utilities Code.

** Municipal systems are owned by a unit of government, like a city, that purchases electricity at wholesale and distributes it to customers

Source: ICF International analysis based on IPM modeling results

The number of potentially affected government entities by ownership type and potential impacts of the Toxics Rule are summarized in Table 10-4. All costs are reported in $2007. EPA estimated the annualized net compliance cost to government entities to be approximately $571 million in 2015.

Table 10-4. Summary of Potential Impacts on Government Entities under the Toxics Rule in 2015

EGU Ownership Type	Number of Potentially Affected Entities	Number of Entities Retiring all Affected units	Total Net Costs of MACT compliance ($2007 MM)	Number of Government Entities with Compliance Cost > 1% of Generation Revenues	Number of Government Entities with Compliance Cost > 3% of Generation Revenues
Sub-Division	11	0	86.8	7	4
State	5	0	38.0	3	2
Municipal	80	1	445.8	45	31
Total	**96**	**1**	**571**	**55**	**37**

Note: The total number of entities with costs greater than 1 percent or 3 percent of revenues includes only entities experiencing positive costs. About 32 of the 96 total potentially affected government entities are estimated to have cost savings under the MACT policy case (see text above for an explanation).

Source: ICF International analysis based on IPM modeling results

Capital and operating costs increase over all ownership types. All ownership types, however, also experience a net gain in electricity revenue, mainly due to higher electricity prices under the policy case. As described in the small entity analysis, the change in electricity revenue takes into account both the profit lost from units that do not operate under the policy case and the difference in revenue for operating units under the policy case. According to EPA modeling, an estimated 2.1 GW of electricity generation is estimated to be uneconomical to operate under the policy case, accounting for about $416 MM in lost profits[1]. On the other hand, many operating units actually increase their electricity revenue due to higher electricity prices under the proposed rule's policy scenario. Excluding retirements, government entities gain about $632 MM in electricity revenue over the base case, resulting in a net gain in electricity revenue of about $216 MM across all ownership types.

10.3 Paperwork Reduction Act

The information collection requirements in this proposed rule will be submitted for approval to the OMB under the PRA, 44 U.S.C. 3501 et seq. An ICR document has been prepared by EPA (ICR No. 2028.05).

The information requirements are based on notification, recordkeeping, and reporting requirements in the NESHAP General Provisions (40 CFR part 63, subpart A), which are mandatory for all operators subject to national emission standards. These recordkeeping and reporting requirements are specifically authorized by CAA section 114 (42 U.S.C. 7414). All information submitted to EPA pursuant to the recordkeeping and reporting requirements for which a claim of confidentiality is made is safeguarded according to Agency policies set forth in 40 CFR part 2, subpart B.

This proposed rule would require maintenance inspections of the control devices but would not require any notifications or reports beyond those required by the General Provisions. The recordkeeping requirements require only the specific information needed to determine compliance.

The annual monitoring, reporting, and recordkeeping burden for this collection (averaged over the first 3 years after the effective date of the standards) is estimated to be $49.1 million. This includes 329,605 labor hours per year at a total labor cost of 27.0 million per year, and total non-labor capital costs of $22.1 million per year. This estimate includes initial and annual performance test, semiannual excess emission reports, maintenance inspections, developing a

[1] As mentioned before, two retiring EGUs owned by the same government entity account for $232 MM in lost profit of this amount.

monitoring plan, notifications, and recordkeeping. The total burden for the Federal government (averaged over the first 3 years after the effective date of the standard) is estimated to be 18,039 hours per year at a total labor cost of $877 million per year. All burden estimates are in 2007 dollars.

Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

An Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for our regulations are listed in 40 CFR part 9 and 48 CFR chapter 15.

To comment on EPA's need for this information, the accuracy of the provided burden estimates, and any suggested methods for minimizing respondent burden, including the use of automated collection techniques, EPA has established a public docket for this proposed rule, which includes this ICR, under the Docket ID numbers for the utility NESHAP and NSPS. Submit any comments related to the ICR to EPA and OMB. The final rule will respond to any OMB or public comments on the information collection requirements contained in this proposal.

10.4 Protection of Children from Environmental Health and Safety Risks

Executive Order 13045 (62 FR 19885, April 23, 1997) applies to any rule that: (1) is determined to be "economically significant" as defined under EO 12866, and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, the Agency must evaluate the environmental health or safety effects of this planned rule on children, and explain why this planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the Agency.

This proposed rule is subject to EO 13045 because it is an economically significant regulatory action as defined by Executive Order 12866, and we believe that the action concerns an environmental health risk which may have a disproportionate impact on children. Although

this proposed rule is based on technology performance, the standards are designed to protect against hazards to public health with an adequate margin of safety as described in Section XX "Hazard to Public Health of Adverse Environmental Effect" in the preamble. The protection offered by this rule may be especially important for children, especially the developing fetus. As referenced in Chapter 5 of the RIA, "Consideration of Health Risks to Children and Environmental Justice Communities" children are more vulnerable than adults to many HAPs emitted by EGUs due to differential behavior patterns and physiology. These unique susceptibilities were carefully considered in a number of different ways in the analyses associated with this rulemaking, and are summarized in Chapter 5 of the RIA.

The public is invited to submit comments or identify peer-reviewed studies and data that assess effects of early life exposure to this proposed rule.

10.5 Executive Order 13132, Federalism

Under EO 13132, EPA may not issue an action that has federalism implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by State and local governments, or EPA consults with State and local officials early in the process of developing the proposed action.

EPA has concluded that this action may have federalism implications, because it may impose substantial direct compliance costs on State or local governments, and the Federal government will not provide the funds necessary to pay those costs. Accordingly, EPA provides the following federalism summary impact statement as required by section 6(b) of EO 13132.

Based on the estimates in EPA's RIA for today's proposed rule, the proposed regulatory option, if promulgated, may have federalism implications because the option may impose approximately $666.3 million in annual direct compliance costs on an estimated 97 State or local governments. Specifically, we estimate that there are 81 municipalities, 5 States, and 11 political subdivisions (i.e., a public district with territorial boundaries embracing an area wider than a single municipality and frequently covering more than one county for the purpose of generating, transmitting and distributing electric energy) that may be directly impacted by today's proposed rule. Responses to EPA's 2010 ICR were used to estimate the nationwide number of potentially impacted State or local governments. As previously explained, this 2010 survey was submitted to all coal- and oil-fired EGUs listed in the 2007 version of DOE/EIA's "Annual Electric Generator Report," and "Power Plant Operations Report."

EPA consulted with State and local officials in the process of developing the proposed rule to permit them to have meaningful and timely input into its development. EPA met with 10 national organizations representing State and local elected officials to provide general background on the proposal, answer questions, and solicit input from State/local governments. The UMRA discussion in the preamble and the RIA includes a description of the consultation.

In the spirit of EO 13132, and consistent with EPA policy to promote communications between EPA and State and local governments, EPA specifically solicits comment on this proposed action from State and local officials.

10.6 Executive Order 13175, Consultation and Coordination with Indian Tribal Governments

Subject to EO 13175 (65 FR 67249, November 9, 2000) EPA may not issue a regulation that has Tribal implications, that imposes substantial direct compliance costs, and that is not required by statute, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by Tribal governments, or EPA consults with Tribal officials early in the process of developing the proposed regulation and develops a Tribal summary impact statement. EO 13175 requires EPA to develop an accountable process to ensure "meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications."

EPA has concluded that this action may have Tribal implications. However, it will neither impose substantial direct compliance costs on Tribal governments, nor preempt Tribal law. This proposed rule would impose requirements on owners and operators of EGUs. EPA is aware of three coal-fired EGUs located in Indian Country but is not aware of any EGUs owned or operated by tribal entities.

EPA offered consultation with Tribal officials early in the process of developing this proposed regulation to permit them to have meaningful and timely input into its development. Consultation letters were sent to 584 Tribal leaders. The letters provided information regarding EPA's development of NESHAP for EGUs and offered consultation. Three consultation meetings were held on December 7, 2011 with the Upper Sioux Community of Minnesota; December 13 with Moapa Band of Paiutes, Forest County Potawatomi, Standing Rock Sioux Tribal Council, Fond du Lac Band of Chippewa; January 5, 2011 with the Forest County Potawatomi, and a representative from the National Tribal Air Association. In these meetings, EPA presented the authority under the CAA used to develop these rules, and an overview of the industry and the industrial processes that have the potential for regulation. Tribes expressed

concerns about the impact of Utility Units on the reservations. Particularly, they were concerned about potential Hg deposition and the impact on the water resources of the Tribes, with particularly concern about the impact on subsistence lifestyles for fishing communities, the cultural impact of impaired water quality for ceremonial purposes, and the economic impact on tourism. In light of these concerns, the tribes expressed interested in an expedited implementation of the rule, they expressed concerns about how the Agency would consider variability in setting the standards, use Tribal-specific fish consumption data from the Tribes in our assessments, they were not supportive of using work practice standards as part of the rule, and asked the Agency to consider going beyond the floor to offer more protection for the Tribal communities. A more specific list of comments can be found in the Docket.

In addition, to these consultations, EPA also conducted outreach on this rule through presentations at the National Tribal Forum in Milwaukee, WI, and on National Tribal Air Association calls. EPA specifically requested tribal data that could support the appropriate and necessary analyses and the RIA for this rule. We will also hold additional meetings with Tribal environmental staff to inform them of the content of this proposal as wells as provide additional consultation with Tribal elected officials where it is appropriate.

EPA specifically solicits additional comment on this proposed rule from Tribal officials.

10.7 Environmental Justice

Our discussion of environmental justice and distributional impacts associated with the proposed Toxics Rule is found in Appendix C, the appendix to Chapter 6 of the benefits analysis.

10.8 Statement of Energy Effects

Our analysis to comply with EO 13211 (Statement of Energy Effects) can be found in section 8.15 of this RIA.

Chapter 11
COMPARISON OF BENEFITS AND COSTS

11.1 Comparison of Benefits and Costs

The estimated social costs to implement the proposed Toxics Rule, as described earlier in this document, are approximately $10.9 billion annually for 2016 (2007 dollars). Thus, the net benefits (social benefits minus social costs) of the program in 2016 are approximately $48 to 130 + B billion or $42 to 120 + B billion annually (2007 dollars, based on a discount rate of 3 percent and 7 percent for the benefits, respectively and rounded to three significant figures). (B represents the sum of all unquantified benefits and disbenefits of the regulation.) Therefore, implementation of this rule is expected, based purely on economic efficiency criteria, to provide society with a significant net gain in social welfare, even given the limited set of health and environmental effects we were able to quantify. Addition of acidification-, and eutrophication-related impacts would likely increase the net benefits of the rule. Table 11-1 presents a summary of the benefits, costs, and net benefits of the proposed Toxics rule.

Air quality modeling was not conducted for options other than that for the MACT floor for each HAP or its surrogate. Table 11-2 below presents the social costs and health benefits, including net social benefit, of the proposed rule.

As with any complex analysis of this scope, there are several uncertainties inherent in the final estimate of benefits and costs that are described fully in Chapters 5, 6, and 8.

Table 11-1. Summary of Annual Benefits, Costs, and Net Benefits of the Proposed Toxics Rule in 2016[a] (billions of 2007 dollars)*

Description	Estimate (3% Discount Rate)	Estimate (7% Discount Rate)
Social costs[b]	$10.9	$10.9
Social benefits[c,d]	$59 to $140 + B	$53 to $130 + B
Net benefits (benefits-costs)	$48 to $130	$42 to $120

[a] Estimates rounded to two significant figures and represent annualized benefits and costs anticipated for the year 2016.

[b] Note that costs are the annualized total social costs of reducing HAP in 2016.The social costs are estimated using the Multimarket Model. More information on the social costs and how they are estimated can be found in Chapter 9 and Appendix F of this RIA.

[c] Total benefits are comprised primarily of monetized PM-related health benefits. The reduction in premature fatalities each year accounts for over 90 percent of total monetized benefits. Benefits in this table are nationwide and are associated with directly emitted $PM_{2.5}$, NO_x, SO_2, and Hg reductions. The estimate of social benefits also includes CO_2-related benefits calculated using the social cost of carbon, discussed further in chapter 6.

[d] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. Estimates here are subject to uncertainties discussed further in the body of the document. Potential benefit categories that have not been quantified and monetized are listed in Table 1-4.

[e] Valuation assumes discounting over the SAB-recommended 20-year segmented lag structure. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

[f] Net benefits are rounded to three significant figures. Columnar totals may not sum due to rounding

* The 2016 compliance costs (incremental to the base case) for the proposed Toxics Rule are approximately $10.9 billion in 2007 dollars.

11.2 References

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