Benefit Analysis for the Section 112 Utility Rule

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SECTION 1

**QUALITATIVE ASSESSMENT OF BENEFITS
OF EMISSION REDUCTIONS**

The emission reductions achieved by the proposed action to reduce mercury and nickel emissions under CAA Section 111 or 112 will provide benefits to society by improving environmental quality. In this section, and the following section, information is provided on the types and levels of social benefits anticipated from the proposed action. This section discusses the health and welfare effects associated with mercury, nickel and other pollutants emitted by affected fossil-fuel fired electric utility steam generating units. The following section quantifies and places a monetary value on a portion of the benefits that are described here.

Results of this analysis are based on the costs and emissions reductions associated with a particular mercury control scenario that is consistent with the reduction in nationwide mercury emissions expected by implementation of the section 112 utility MACT standard in this proposal. The specific emissions control scenario is derived from application of the Integrated Planning Model (IPM), which EPA has used to assess the costs and emissions reductions associated with a number of regulations of the power sector. While the mercury reduction estimates in the scenario are consistent with the Agency's assessment of control technologies, EPA is aware that estimates of associated reductions in other pollutants, notably sulfur dioxide (SO_2) and nitrogen oxides (NOx) (co-benefits) may vary significantly with alternative assumptions about the application of particular control technologies and incentives created by the existence of other major regulatory programs affecting the power sector.   In particular, based on past EPA analyses of multi-pollutant strategies (e.g. Clear Skies Technical Support Document D, www.epa.gov/clearskies/ technical.html) and the analysis of the Interstate Air Quality Rule (IAQR; available in the docket) the control choices made pursuant to either a 112 or 111 based mercury program would likely be significantly affected by the requirements of the Interstate Air Quality Rule, which is intended to reduce the contribution of transported SO_2 and NOx emissions to violations of the PM2.5 and ozone NAAQS.   For these reasons, in addition to the findings of the analyses derived from the MACT only scenario, we also provide some rough estimates of the direction of costs and benefits under reasonably foreseeable alternative scenarios for implementing 112 and 111 standards that take such potential interactions into account.

The results of this analysis do not reflect any additional impacts, positive or negative, associated with additional mercury emission reductions beyond those that should result from sources meeting the emission limitations that make up the section 112 MACT floor alternative. The proposed actions are expected to reduce emissions of mercury, which can cause neurological damage and learning disorders, and nickel, which is classified as a probable human carcinogen based upon studies in animals. Due to the control technologies selected for analysis, the actions

to reduce mercury will also achieve reductions of NOx and SO2. Although not incorporated into the analyses, the actions to reduce nickel will also reduce direct emissions of particulate matter. The reduction of $PM_{2.5}$ formation, both from reductions in NO_x and SO_2, and reductions in directly emitted fine particles, may result in reduced fatalities, fewer cases of chronic bronchitis, asthma, and hospitalizations for respiratory diseases. The NO_x emissions also contribute to the formation of ground-level ozone. Reductions in ozone may result in reduced hospitalizations for respiratory diseases, reduced emergency room visits for asthma, fewer cases of acute respiratory illnesses, and fewer school absences due to illnesses. In addition, these emission reductions may also result in ecosystem and other welfare improvements, including effects on crops and other plant life, materials damage, soiling, reduced visibility impairment, and acidification of water bodies.

Due to technical, time, and resource limitations, the EPA is unable to model the impacts of the mercury and nickel emission reductions that may result from this regulation. Therefore, the EPA does not know the extent to which the adverse health effects described in this section occur in the populations surrounding these facilities. However, to the extent the adverse effects do occur, the rule will reduce emissions and subsequent exposures. For similar reasons, we are unable to quantify the co-benefits of reductions in ambient gaseous NO_x and SO_2, or ozone reductions resulting from reductions in NO_x emissions for this rule. Section 2 of this report presents an estimation of the health impacts and monetary co-benefits associated with reductions in $PM_{2.5}$ resulting from the reduction of NO_x and SO_2 emissions. Due to difficulties in quantifying emission reductions associated with application of control technologies, we are unable to quantify the benefits associated with reductions in directly emitted fine particles. A control technology specifically dedicated to mercury reductions, activated carbon injection (ACI), in many cases would require the use of another control technology, an added pulse-jet fabric filter. Similar to larger fabric filters currently used by power generation units, we expect that these pulse-jet fabric filters would achieve PM reductions in addition to the mercury reductions. However, at this time we do not have adequate test data to document the PM-removal efficiency associated with these devices. Without a PM-removal efficiency, no emission reductions of PM can be quantified, which leads us to our conclusion that we are unable to quantify the benefits associated with application of control technologies.

In 1997-98, EPA developed two studies of the emissions and health effects of pollutants emitted from electric utilities. According to the Mercury Study Report to Congress (EPA, 1997) and the Utility Air Toxics Report to Congress (EPA, 1998), mercury was identified as the toxic of greatest concern. These reports indicated that coal-fired power plants are the nation's largest source of mercury air emissions. Therefore, below we discuss the potential effects of mercury and then provide a description of potential effects from the emission reductions of nickel. In the last part of this section, we discuss the potential benefits of reducing NO_x and SO_2 emissions.

1.1 Benefits of Reducing Mercury Emissions

According to baseline emission estimates, the sources affected by this proposal currently emit approximately 44 tons of mercury per year nationwide. The proposed regulation will reduce approximately 15 tons of mercury (or 34%) at electric utility facilities that generate steam using fossil fuels (i.e., coal or oil fuels). For more information on the control technologies estimated to be used to comply with this rule and the calculation of emission reductions for Hg, please refer to the "Economic and Energy Impact Analysis for the Utility MACT Proposed Rulemaking" memo available in the docket for this proposal.

Mercury emitted from utilities and other natural and man-made sources is carried by winds through the air and eventually is deposited to water and land. Recent estimates (which are highly uncertain) of annual total global mercury emissions from all sources (natural and anthropogenic) are about 5,000 to 5,500 tons per year (tpy). Of this total, about 1,000 tpy are estimated to be natural emissions and about 2,000 tpy are estimated to be contributions through the natural global cycle of re-emissions of mercury associated with past anthropogenic activity. Current anthropogenic emissions account for the remaining 2,000 tpy. Point sources such as fuel combustion; waste incineration; industrial processes; and metal ore roasting, refining, and processing are the largest point source categories on a world-wide basis. Given the global estimates noted above, U.S. anthropogenic mercury emissions are estimated to account for roughly 3 percent of the global total, and U.S. utilities are estimated to account for about 1 percent of total global emissions. Mercury exists in three forms: elemental mercury, inorganic mercury compounds (primarily mercuric chloride), and organic mercury compounds (primarily methylmercury). Mercury is usually released in an elemental form and later converted into methylmercury by bacteria. Methylmercury is more toxic to humans than other forms of mercury, in part because it is more easily absorbed in the body (EPA, 1996).

If the deposition is directly to a water body, then the processes of aqueous fate, transport, and transformation begin. If deposition is to land, then terrestrial fate and transport processes occur first and then aqueous fate and transport processes occur once the mercury has cycled into a water body. In both cases, mercury may be returned to the atmosphere through resuspension. In water, mercury is transformed to methylmercury through biological processes and for exposures affected by this rulemaking, methylmercury is considered to be the form of greatest concern. Once mercury has been transformed into methylmercury, it can be ingested by the lower trophic level organisms where it can bioaccumulate in fish tissue (i.e., concentrations of mercury remain in the fish's system for a long period of time and accumulates in the fish tissue as predatory fish consume other species in the food chain). Fish and wildlife at the top of the food chain can, therefore, have mercury concentrations that are higher than the lower species, and they can have concentrations of mercury that are higher than the concentration found in the water body itself. In addition, when humans consume fish contaminated with methylmercury, the ingested methylmercury is almost completely absorbed into the blood and distributed to all tissues (including the brain); it also readily passes through the placenta to the fetus and fetal brain (EPA, 2001a).

Based on the findings of the National Research Council, EPA has concluded that benefits of Hg reductions would be most apparent at the human consumption stage, as consumption of fish is the major source of exposure to methylmercury. At lower levels, documented Hg exposure effects may include more subtle, yet potentially important, neurodevelopmental effects. Figure 1-1 shows how emissions of mercury can transport from the air to water and impact human health and ecosystems.
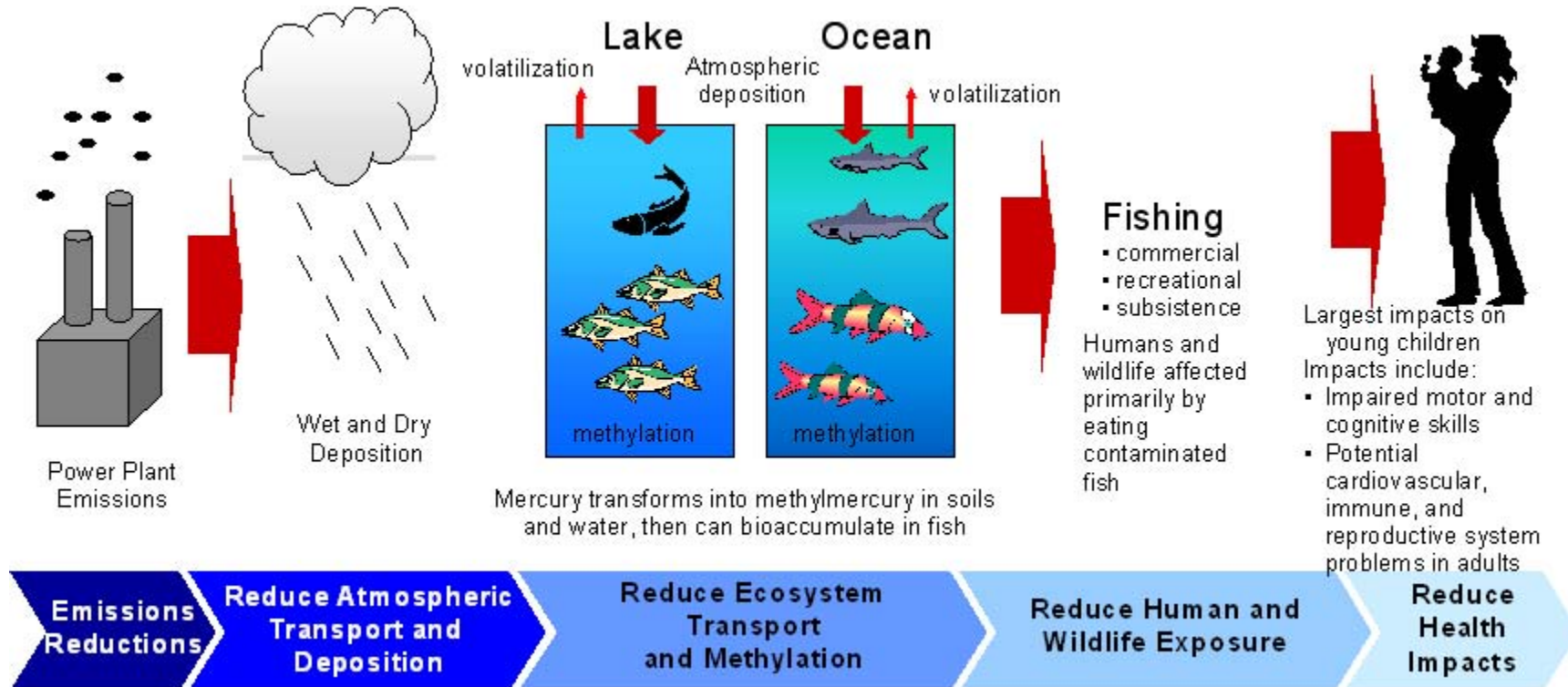
Figure 1-1: How Emissions of Mercury Can Impact Human Health and Ecosystems[1]

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[1] Cardiovascular, immune, and reproductive system problems in adults are potential effects as the literature is either contradictory or incomplete.

Some subpopulations in the U.S., such as: Native Americans, Southeast Asian Americans, and lower income subsistence fishers, may rely on fish as a primary source of nutrition and/or for cultural practices. Therefore, they consume larger amounts of fish than the general population and may be at a greater risk to the adverse health effects from Hg due to increased exposure. In pregnant women, methylmercury can be passed on to the developing fetus, and at sufficient exposure may lead to a number of neurological disorders in children. Thus, children who are exposed to low concentrations of methylmercury prenatally 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. The effects from prenatal exposure can occur even at doses that do not result in effects in the mother. Mercury may also affect young children who consume fish contaminated with Hg. Consumption by children may lead to neurological disorders and developmental problems, which may lead to later economic consequences.

Monitoring the concentrations of mercury in the blood of women of child-bearing age can help identify the proportion of children who may be at risk. EPA's reference dose (RfD) for methylmercury is 0.1 micrograms per kilogram body weight per day, which is approximately equivalent to a concentration of 5.8 parts per billion mercury in blood. Although the prenatal period is the most sensitive period of exposure, exposure to mercury during childhood also could pose a potential health risk (NAS, 2000).

Figure 1-2 shows reported concentrations of mercury in blood of women of childbearing age from the National Health and Nutrition Examination Survey (NHANES) (EPA, 2003b). The data presented are for total mercury, which includes methylmercury and other forms of mercury. Total blood mercury is a reasonable indicator of methylmercury exposure in people who consume fish and have no significant exposure to inorganic or elemental mercury (JAMA, April 2003). Thus the measured concentrations are a good indication of methylmercury concentrations. From this survey, about 8 percent of women of child-bearing age had at least 5.8 parts per billion of mercury in their blood in 1999-2000.
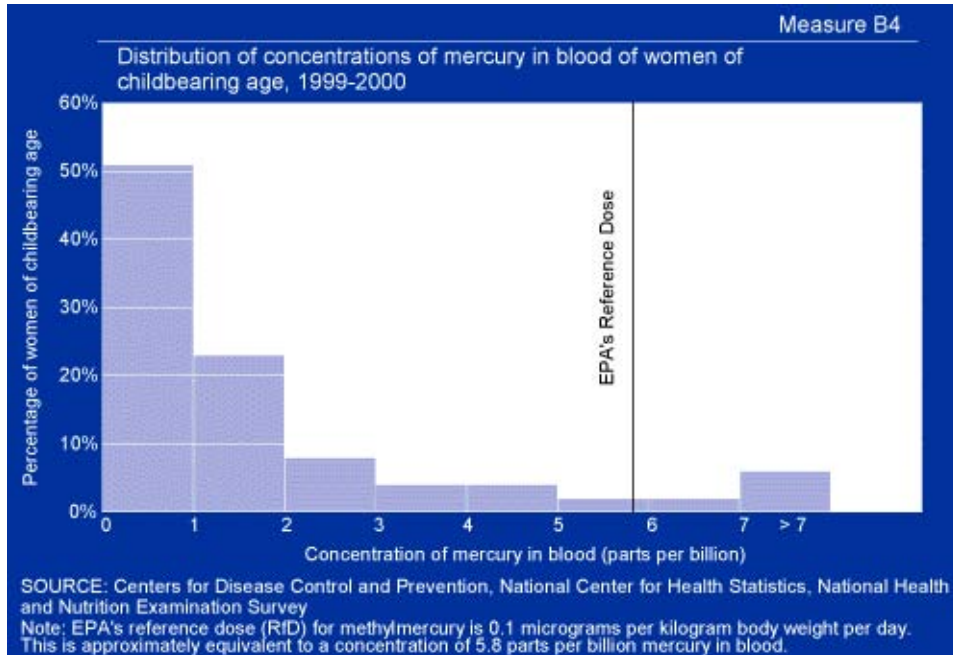
Figure 1-2: Concentrations of Mercury in Blood of Women of Childbearing Age

Figure 1-3 shows relative values of the BMD, BMDL and the RfD. The data show a Benchmark Dose (BMD) BMD at 85 ppb. The BMD is the dose or concentration that produced a doubling of the number of children with a response at the 5th percentile of the population.   In this case, the changes evaluated were changes on neuropsychological testing batteries (i.e. the Boston Naming Test). In determining the RfD, EPA started with the BMD (85 ppb) and then used the 95% lower confidence limit to arrive at the 58 ppb BMDL. EPA then applied a composite uncertainty factor of 10 to calculate a final RfD of 5.8 ppb. The uncertainty factor adjustment was used to account for pharmacokinetic and pharmacodynamic uncertainty and variability.
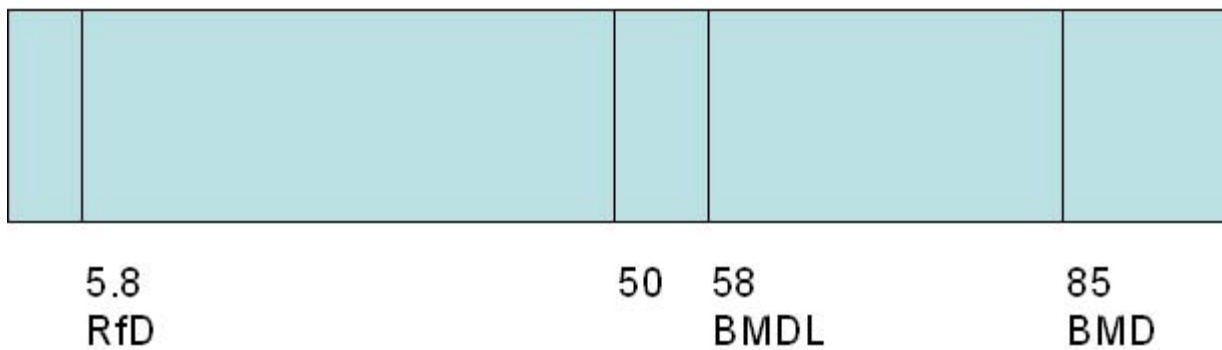

Figure 1-3: Relative Values of BMD, BMDL, and the RfD
(Values in ppb)

In response to potential risks of mercury-contaminated fish consumption, EPA and FDA have issued fish consumption advisories (FCA) which provide recommended limits on consumption of certain fish species for different populations. EPA and FDA are currently developing a joint advisory that has been released in draft form. This newest draft FDA-EPA fish advisory recommends that women and young children reduce the risks of mercury consumption in their diet by moderating their fish consumption, diversifying the types of fish they consume, and by checking any local advisories that may exist for local rivers and streams. This collaborative FDA-EPA effort will greatly assist in educating the most susceptible populations. Additionally, the reductions of mercury from this regulation may potentially lead to fewer fish consumption advisories (both from federal or state agencies), which will benefit the fishing community. As Figure 1-4 shows, currently 44 states have issued fish consumption advisories for non-commercial fish for some or all of their waters due to contamination of mercury. The scope of FCA issued by states varies considerably, with some warnings applying to all water bodies in a state and others applying only to individual lakes and streams. Note that the absence of a state advisory does not necessarily indicate that there is no risk of exposure to unsafe levels of mercury in recreationally caught fish. Likewise, the presence of a state advisory does not indicate that there is a risk of exposure to unsafe levels of mercury in recreationally caught fish, unless people consume these fish at levels greater than those recommended by the fish advisory. This figure also displays the change in mercury emissions that will result from implementation of this rule. Note that we are not able to predict whether these reductions in emissions will result in any changes in FCA in states where the emissions reductions are projected to occur.

Reductions in methylmercury concentrations in fish should reduce exposure, subsequently reducing the risks of mercury-related health effects in the general population, to children, and to certain subpopulations. Fish consumption advisories (FCA) issued by the States may also help to reduce exposures to potential harmful levels of methylmercury in fish (although some studies have shown limited knowledge of and compliance with advisories by at risk populations (May and Burger, 1996; Burger, 2000)). To the extent that reductions in mercury emissions reduces the probability that a water body will have a FCA issued, there are a number of benefits that will result from fewer advisories, including increased fish consumption, increased fishing choices for recreational fishers, increased producer and consumer surplus for the commercial fish market, and increased welfare for subsistence fishing populations.
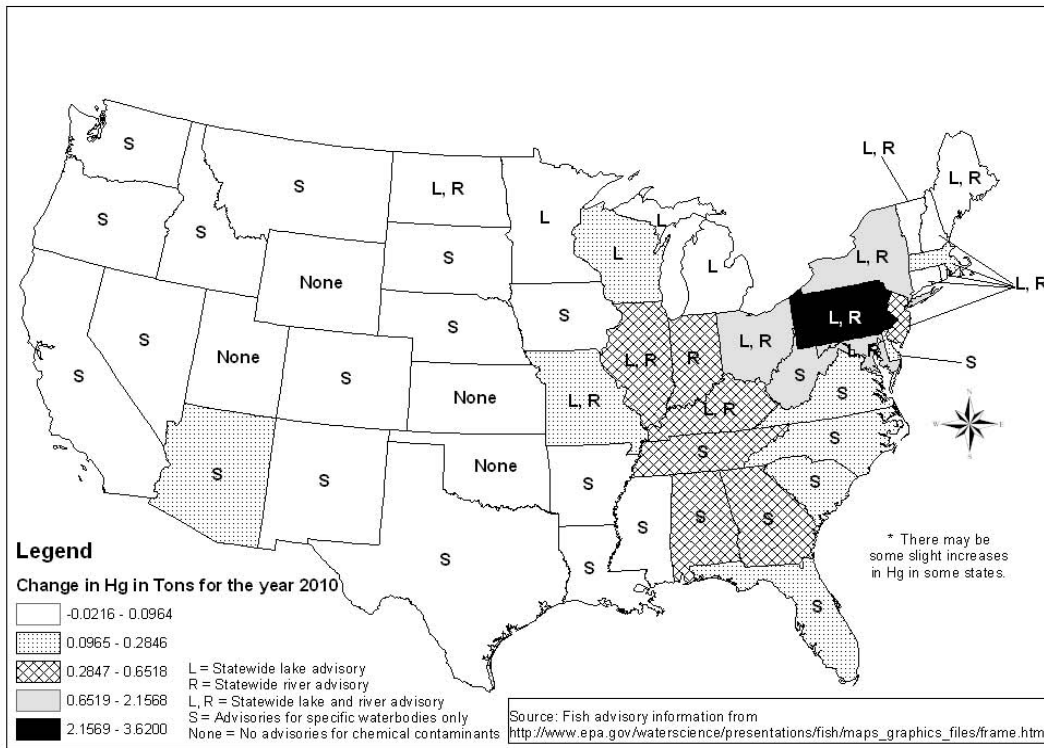
Figure 1-4: States with Fish Consumption Advisories and Estimated Change in Mercury Emissions Due to Regulation

There is a great deal of variability among individuals in fish consumption rates, however, critical elements in estimating methylmercury exposure and risk from fish consumption include the species of fish consumed, the concentrations of methylmercury in the fish, the quantity of fish consumed, and how frequently the fish is consumed. The typical U.S. consumer eating a wide variety of fish from restaurants and grocery stores is not in danger of consuming harmful levels of methylmercury from fish and is not advised to limit fish consumption. Those who regularly and frequently consume large amounts of fish, either marine or freshwater, are more exposed. Because the developing fetus may be the most sensitive to the effects from methylmercury, women of child-bearing age are regarded as the population of greatest interest. The EPA, Food and Drug Administration, and many States have issued fish consumption advisories to inform this population of protective consumption levels.

The EPA's 1997 Mercury Study RTC supports a plausible link between anthropogenic releases of Hg from industrial and combustion sources in the U.S. and methylmercury in fish.

However, these fish methylmercury concentrations also result from existing background concentrations of Hg (which may consist of Hg from natural sources, as well as Hg which has been re-emitted from the oceans or soils) and deposition from the global reservoir (which includes Hg emitted by other countries). Given the current scientific understanding of the environmental fate and transport of this element, it is not possible to quantify how much of the methylmercury in locally-caught fish consumed by the U.S. population is contributed by U.S. emissions relative to other sources of Hg (such as natural sources and re-emissions from the global pool). As a result, the relationship between Hg emission reductions from Utility Units and methylmercury concentrations in fish cannot be calculated in a quantitative manner with confidence. In addition, there is uncertainty regarding over what time period these changes would occur. This is an area of ongoing study.

Given the present understanding of the Hg cycle, the flux of Hg from the atmosphere to land or water at one location is comprised of contributions from: the natural global cycle; the cycle perturbed by human activities; regional sources; and local sources. Recent advances allow for a general understanding of the global Hg cycle and the impact of the anthropogenic sources. It is more difficult to make accurate generalizations of the fluxes on a regional or local scale due to the site-specific nature of emission and deposition processes. Similarly, it is difficult to quantify how the water deposition of Hg leads to an increase in fish tissue levels. This will vary based on the specific characteristics of the individual lake, stream, or ocean.

1.2   Benefits of Reducing Emissions of Nickel

According to baseline emission estimates, the sources affected by this proposal currently emit approximately 579 tons of nickel per year. The proposed regulation will reduce approximately 219 tons of nickel (or 38%) at electric utility facilities that generate steam using fossil fuels (i.e., coal or oil fuels). The HAP emission reductions achieved by this rule are expected to reduce exposure to ambient concentrations of nickel. Detailed information on the effects of nickel can be obtained from the *Integrated Risk Information System (IRIS),* an EPA system for disseminating information about the effects of several chemicals emitted to the air and/or water, and classifying these chemicals by cancer risk (EPA, 2000). According to IRIS, nickel is an essential element in some animal species, and it has been suggested it may be essential for human nutrition. Nickel dermatitis, consisting of itching of the fingers, hand and forearms, is the most common effect in humans from chronic (long-term) skin contact with nickel. Respiratory effects have also been reported in humans from inhalation exposure to nickel. No information is available regarding the reproductive or developmental effects of nickel 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 nickel refinery dusts and nickel subsulfide. Animal studies of soluble nickel compounds (i.e., nickel carbonyl) have reported lung tumors. EPA has classified nickel refinery subsulfide as Group A, human carcinogens and nickel carbonyl as a Group B2, probable human carcinogen.

1.3 Welfare Benefits of Nickel and Mercury Reductions

The welfare effects of exposure to nickel and mercury have received less attention from analysts than the health effects. However, this situation is changing, especially with respect to the effects of toxic substances on ecosystems. Over the past ten years, ecotoxicologists have started to build models of ecological systems which focus on interrelationships in function, the dynamics of stress, and the adaptive potential for recovery. Chronic sub-lethal exposures may affect the normal functioning of individual species in ways that make it less than competitive and therefore more susceptible to a variety of factors including disease, insect attack, and decreases in habitat quality (EPA, 1991). All of these factors may contribute to an overall change in the structure (i.e., composition) and function of the ecosystem. Therefore, the nickel and mercury emission reductions achieved through the proposed actions should reduce the associated adverse welfare (environmental) impacts.

The adverse, non-human biological effects of nickel emissions include ecosystem and recreational and commercial fishery impacts. Atmospheric deposition of nickel directly to land may affect terrestrial ecosystems. Atmospheric deposition of nickel also contributes to adverse aquatic ecosystem effects. This not only has adverse implications for individual wildlife species and ecosystems as a whole, but also the humans who may ingest contaminated fish and waterfowl.

A number of wildlife species are at risk from consuming mercury-contaminated fish (Duvall and Baron, 2000). Mercury can affect reproductive success in birds and mammals which may affect population levels (Peakall, 1996). This can affect human welfare in several ways. If changes in populations reduces biological diversity in an area this may impact the total ecological system. To the extent that people value biological diversity (existence value), there may be benefits to preventing this loss. Also, hunters may experience direct losses if populations of game birds or animals are reduced. Hunters may also experience welfare losses if game birds or animals are not fit for consumption. Hunters may also be affected if predator populations are reduced from reduced availability of prey species. In addition to hunting, other non-consumptive uses of wildlife including bird or wildlife viewing may be impacted by reductions in bird and animal populations.

1.4   Benefits of Reducing Other Pollutants Due to Utility MACT Controls

As is mentioned above, controls that will be required on fossil-fuel fired utilities to reduce HAPs will also reduce emissions of other pollutants, namely NO_x, and SO_2. According to baseline emission estimates, the sources affected by this proposal currently emit approximately 3.95 million tons per year of NO_x, and 9.76 million tons per year of SO_2. The proposed action will reduce approximately 902,000 tons of NO_x emissions, and 591,000 tons of SO_2 emissions. For more information on these HAP emissions and emission reductions, please refer to the memo titled "Economic and Energy Impact Analysis for Utility MACT Proposed Rulemaking" available in the docket for this proposal. A qualitative discussion of the adverse effects from

NO_x, SO_2, and secondarily formed $PM_{2.5}$ are presented below.

1.4.1 *Benefits of Nitrous Oxide Reductions.* Emissions of NO_x produce a wide variety of health and welfare effects. Nitrogen dioxide can irritate the lungs at high occupational levels and may lower resistance to respiratory infection, although the research has been equivocal. NO_x emissions are an important precursor to acid rain and may affect both terrestrial and aquatic ecosystems. Atmospheric deposition of nitrogen leads to excess nutrient enrichment problems ("eutrophication") in the Chesapeake Bay and several nationally important estuaries along the East and Gulf Coasts. Eutrophication can produce multiple adverse effects on water quality and the aquatic environment, including increased algal blooms, excessive phytoplankton growth, and low or no dissolved oxygen in bottom waters. Eutrophication also reduces sunlight, causing losses in submerged aquatic vegetation critical for healthy estuarine ecosystems. Deposition of nitrogen-containing compounds also affects terrestrial ecosystems. Nitrogen fertilization can alter growth patterns and change the balance of species in an ecosystem.

Nitrogen dioxide and airborne nitrate also contribute to pollutant haze (often brown in color), which impairs visibility and can reduce residential property values and the value placed on scenic views.

NO_x in combination with volatile organic compounds (VOC) also serves as a precursor to ozone. Based on a large number of recent studies, EPA has identified several key health effects that may be associated with exposure to elevated levels of ozone. Exposures to ambient ozone concentrations have been linked to increased hospital admissions and emergency room visits for respiratory problems. Repeated exposure to ozone may increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory disease, such as asthma. Repeated prolonged exposures (i.e., 6 to 8 hours) to ozone at levels between 0.08 and 0.12 ppb, over months to years may lead to repeated inflammation of the lung, impairment of lung defense mechanisms, and irreversible changes in lung structure, which could in turn lead to premature aging of the lungs and/or chronic respiratory illnesses such as emphysema, chronic bronchitis, and asthma.

Children have the highest exposures to ozone because they typically are active outside playing and exercising, during the summer when ozone levels are highest. Further, children are more at risk than adults from the effects of ozone exposure because their respiratory systems are still developing. Adults who are outdoors and moderately active during the summer months, such as construction workers and other outdoor workers, also are among those with the highest exposures. These individuals, as well as people with respiratory illnesses such as asthma, especially children with asthma, may experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during periods of moderate exertion. In addition to human health effects, ozone adversely affects crop yield, vegetation and forest growth, and the durability of materials. Ozone causes noticeable foliar damage in many crops, trees, and ornamental plants (i.e., grass, flowers, shrubs, and trees) and causes reduced growth in plants.

Particulate matter (PM) can also be formed from NO_x emissions. Secondary PM is formed in the atmosphere through a number of physical and chemical processes that transform gases such as NO_x, SO_2, and VOC into particles. A discussion of the effects of PM on human health and the environment are discussed further below. Overall, reducing the emissions of NO_x from fossil-fuel fired utilities can help to improve some of the effects discussed in this section - either those directly related to NO_x emissions, or the effects of ozone and PM resulting from the combination of NO_x with other pollutants.

1.4.2 Benefits of Sulfur Dioxide Reductions. Very high concentrations of sulfur dioxide (SO_2) affect breathing and ambient levels have been hypothesized to aggravate existing respiratory and cardiovascular disease. Potentially sensitive populations include asthmatics, individuals with bronchitis or emphysema, children and the elderly. SO_2 is also a primary contributor to acid deposition, or acid rain, which causes acidification of lakes and streams and can damage trees, crops, historic buildings and statues. In addition, sulfur compounds in the air contribute to visibility impairment in large parts of the country. This is especially noticeable in national parks.

PM can also be formed from SO_2 emissions. Secondary PM is formed in the atmosphere through a number of physical and chemical processes that transform gases, such as SO_2, into particles. A discussion of the effects of PM on human health and the environment are discussed further below. Overall, reducing the emissions of SO_2 from fossil-fuel fired utilities can help to improve some of the effects discussed in this section - either those directly related to SO_2 emissions, or the effects of ozone and PM resulting from the combination of SO_2 with other pollutants.

1.4.3 Benefits of Particulate Matter Reductions. Scientific studies have linked PM (alone or in combination with other air pollutants) with a series of health effects (EPA, 1996). Fine particles ($PM_{2.5}$) can penetrate deep into the lungs to contribute to a number of the health effects. These health effects include decreased lung function and alterations in lung tissue and structure and in respiratory tract defense mechanisms which may be manifest in increased respiratory symptoms and disease or in more severe cases, increased hospital admissions and emergency room visits or premature death. Children, the elderly, and people with cardiopulmonary disease, such as asthma, are most at risk from these health effects.

PM also causes a number of adverse effects on the environment. Fine PM is the major cause of reduced visibility in parts of the U.S., including many of our national parks and wilderness areas. Other environmental impacts occur when particles deposit onto soil, plants, water, or materials. For example, particles containing nitrogen and sulfur that deposit onto land or water bodies may change the nutrient balance and acidity of those environments, leading to changes in species composition and buffering capacity. Particles that are deposited directly onto leaves of plants can, depending on their chemical composition, corrode leaf surfaces or interfere with plant metabolism. Finally, PM causes soiling and erosion damage to materials.

Thus, reducing the emissions of PM and PM precursors from fossil-fuel fired utilities can help to improve some of the effects mentioned above - either those related to primary PM emissions, or the effects of secondary PM generated by the combination of NO_x or SO_2 with other pollutants in the atmosphere.

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SECTION 2

ANALYSIS OF NOX AND SO2 RELATED CO-BENEFITS OF THE PROPOSED UTILITY MACT RULE

Results of this analysis are based on the costs and emissions reductions associated with a particular mercury control scenario that is consistent with the reduction in nationwide mercury emissions expected by implementation of the section 112 utility MACT standard in this proposal. The specific emissions control scenario is derived from application of the Integrated Planning Model (IPM), which EPA has used to assess the costs and emissions reductions associated with a number of regulations of the power sector. While the mercury reduction estimates in the scenario are consistent with the Agency's assessment of control technologies, EPA is aware that estimates of associated reductions in other pollutants, notably SO2 and NOx (co-benefits) may vary significantly with alternative assumptions about the application of particular control technologies and incentives created by the existence of other major regulatory programs affecting the power sector. In particular, based on past EPA analyses of multi-pollutant strategies (e.g. Clear Skies Technical Support Document D, www.epa.gov/clearskies/ technical.html) and the analysis of the Interstate Air Quality Rule (IAQR; available in the docket) the control choices made pursuant to either a 112 or 111 based mercury program would likely be significantly affected by the requirements of the Interstate Air Quality Rule, which is intended to reduce the contribution of transported SO2 and NOx emissions to violations of the PM2.5 and ozone NAAQS. For these reasons, in addition to the findings of the analyses derived from the MACT only scenario, we also provide some rough estimates of the direction of costs and benefits under reasonably foreseeable alternative scenarios for implementing 112 and 111 standards that take such potential interactions into account.

Due to predicted adoption of activated carbon injection (ACI) with fabric filters by some sources, there is also likely to be reductions in directly emitted fine particles. However, we are not able to quantify the magnitude of these emission reductions, so we omit them from the quantified benefits analysis. Benefits from these reductions in direct fine particle emissions may be substantial and their omission will lead to a potentially significant underestimate of health impacts and dollar benefits. Due to technical limitations, we are currently unable to provide any quantified estimate of the human health benefits associated with reductions in mercury or nickel emissions. The EPA is working to better understand the environmental and health impacts associated with mercury emissions from power plants. We are developing methods for quantifying and valuing reductions in methylmercury concentrations in fish, and will be evaluating those methods for use in estimating mercury-related health benefits for the final rule. A qualitative discussion of mercury and nickel health impacts is provided in the previous section.

Changes in emissions of NO_x and SO_2 expected to occur as a result of applying controls to meet the MACT standard were estimated using the Integrated Planning Model (IPM). No

formal modeling of the impacts of these predicted changes in emissions on ambient concentrations of $PM_{2.5}$ was conducted. Instead, we use a benefits transfer method to scale the results of the benefits analysis conducted for the proposed Clear Skies legislation[2]. The Clear Skies program reflects a similar universe of affected sources and similar to the proposed Utility MACT, provided both NO_x and SO_2 reductions. The distribution of emission reductions across states differs between the two analyses, especially in the Western U.S. Given the very small reductions in NO_x and SO_2 expected to occur in the Western U.S. as a result of the rule and the potential for errors in transferring benefits, we limit the benefits analysis to the Eastern U.S., and derive the benefits transfer factors from the Eastern U.S. Clear Skies benefits results only. Recognizing the differences in emission reduction patterns in the Eastern U.S. between the Clear Skies analysis and the current proposed MACT standard, we believe that the benefits per ton of SO_2 and NO_x estimated for the Clear Skies analysis represents a reasonable approximation of the benefits per ton that might be realized from the reductions in NO_x and SO_2 expected under the current proposed rule. The benefits transfer method used to estimate benefits for the proposed standards is similar to that used to estimate benefits in the recent analyses of the proposed Nonroad Diesel rule and Large SI/Recreational Vehicles standards (see RIA, Docket A-2000-01). A similar method has also been used in recent benefits analyses for the proposed Industrial Boilers and Process Heaters MACT standards and the Reciprocating Internal Combustion Engines MACT standards. The analysis of the Utility MACT only includes health benefits related to NO_x and SO_2 reductions, omitting health benefits related to ozone reductions, visibility benefits, and other benefits including reduced nitrogen deposition and acidification. For the most part, quantifiable ozone benefits do not contribute significantly to the monetized benefits: thus, their omission will not materially affect the conclusions of the benefits analysis. Visibility benefits may be significant, however, they usually contributed only a few percent of total monetized benefits.

Table 2-1 lists the known quantified and unquantified effects considered for this analysis. It is important to note that there are significant categories of benefits which can not be monetized (or in many cases even quantified), resulting in a significant limitation to this analysis.

The benefit analysis that we performed for our proposed rule can be thought of as having four parts, each of which will be discussed separately in the Sections that follow. These four steps are:

1. Identification of proposed standard and calculation of the impact that the proposed standards will have on the nationwide inventories for NO_x and SO_2 emissions in 2010;
2. Calculation of scaling factors relating emissions changes resulting from the proposed standard to emissions changes from the IPM runs that were used to model air quality

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[2]For details on the analysis of the proposed Clear Skies Act 2003, see http://www.epa.gov/clearskies.

and benefits for the proposed Clear Skies legislation[3].

3. Apportionment of modeled benefits from the Clear Skies analysis to NO_x and SO_2 emissions.
4. Application of scaling factors to apportioned modeled benefits associated with NO_x and SO_2 in 2010 to estimate benefits for the Utility MACT emission reductions.

This primary analysis presents estimates of the potential benefits from the proposed Utility MACT rule occurring in 2010. The predicted emissions reductions that will result from the rule have yet to occur, and therefore the actual changes in human health outcomes to which economic values are ascribed are predictions. These predictions are based on the best available scientific evidence and judgment, but there is unavoidable uncertainty associated with each step in the complex process between regulation and specific health and welfare outcomes. Uncertainties associated with projecting input and parameter values into the future may contribute significantly to the overall uncertainty in the benefits estimates. However, we make these projections to more completely examine the impact of the program as the rule is implemented. The additional uncertainties added to the analysis through application of the Clear Skies based scaling factors instead of full scale air quality modeling are unknown. We discuss some potential sources of bias in the text, but a complete quantified characterization of uncertainty is not possible for this analysis.

In general, the chapter is organized around the steps laid out above. In section 2.1, we identify the potential standard to analyze and summarize emissions impacts. In section 2.2, we summarize the changes in emissions that were used in the Clear Skies benefits analysis and develop ratios of emissions that are used to scale Clear Skies benefits. In section 2.3, we summarize the modeled benefits associated with the emissions changes for the Clear Skies legislation and apportion those benefits to the individual emission species (NO_x and SO_2). Finally, in Section 2.4, we estimate the benefits in 2010 for the proposed standard, based on scaling of the modeled benefits of Clear Skies.

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[3]Detailed information on the emissions, air quality, and benefits analyses supporting the proposed Clear Skies legislation are available on the internet at http://www.epa.gov/clearskies.

Table 2-1.
Health and Welfare Effects of Pollutants Affected by the Proposed Utility MACT Standard

Pollutant/Effect	Quantified and Monetized	Potential Unquantified Effects
PM/Health	Premature mortality - adults Premature mortality - infants Bronchitis - chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Lower and upper respiratory illness Asthma exacerbations Minor restricted activity days Work loss days	Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Non-asthma respiratory emergency room visits Changes in cardiac function (e.g. heart rate variability) Allergic responses (to diesel exhaust)
PM/Welfare		Visibility in Class I areas Visibility in residential and non-Class I areas Household soiling
Ozone/Health		Increased airway responsiveness to stimuli Inflammation in the lung Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room visits Hospital admissions - respiratory Emergency room visits for asthma Minor restricted activity days School loss days Asthma attacks Cardiovascular emergency room visits Premature mortality – acute exposures Acute respiratory symptoms

Pollutant/Effect	Quantified and Monetized	Potential Unquantified Effects
Ozone/Welfare		Decreased commercial forest productivity Decreased yields for fruits and vegetables Decreased yields for commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions Decreased outdoor worker productivity
Nitrogen and Sulfate Deposition/ Welfare		Costs of nitrogen controls to reduce eutrophication in selected eastern estuaries Impacts of acidic sulfate and nitrate deposition on commercial forests Impacts of acidic deposition on commercial freshwater fishing Impacts of acidic deposition on recreation in terrestrial ecosystems Impacts of nitrogen deposition on commercial fishing, agriculture, and forests Impacts of nitrogen deposition on recreation in estuarine ecosystems Reduced existence values for currently healthy ecosystems
SO$_2$/Health		Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics
NO$_x$/Health		Lung irritation Lowered resistance to respiratory infection Hospital Admissions for respiratory and cardiac diseases
Mercury Health		Neurological disorders Learning disabilities Neonatal development delays Potential Cardiovascular effects* Altered blood pressure regulation* Increased heart rate variability* Myocardial infarctions* Potential Reproductive effects*
Mercury Deposition Welfare		Deposition Impacts on birds and mammals (e.g. reproductive effects) Impacts to commercial, subsistence, and recreational fishing Reduced existence values for currently healthy ecosystems

* These are potential effects as the literature is either contradictory or incomplete.

2.1 Emission Changes Expected to Result from Implementation of the Proposed Standard

The proposed standards have various cost and emission related components, as described in the "Economic and Energy Impact Analysis for the Utility MACT Proposed Rulemaking" memo available in the docket for this proposal. The controls and emission reductions are expected to be implemented by 2008. Our benefits analysis provide a snapshot of the expected human health impacts and dollar benefits in 2010. We chose 2010 due to the availability of air quality modeling for Clear Skies in 2010.

Table 2-2 summarizes the expected changes in emissions of SO_2 and NO_x, based on the IPM modeling for 2010. Over 99 percent of emission reductions for SO_2 and NO_x are predicted to occur in the Eastern U.S. As such, our omission of benefits occurring in the Western U.S. will not result in a large downward bias in our national benefits estimate.

Table 2-2.
Summary of 2010 Reductions in Emissions of SO_2 and NO_x Predicted from Utility MACT IPM Modeling

	Tons Reduced (% of baseline)	
	NO_x	SO_2
Eastern U.S.	899,179	590,846
	26.8%	6.3%
Western U.S.	2,742	592
	0.5%	0.2%
Total	901,921	591,438
	23.2%	6.1%

2.2 Development of Benefits Scaling Factors Based on Differences in Emission Impacts Between Proposed MACT and Clear Skies

The Clear Skies benefits analysis was based on the pattern of reductions in emissions of SO_2 and NO_x occurring as a result of a nationwide cap and trade program. Under the Clear Skies proposal, emissions of NO_x were expected to be reduced by about 1.7 million tons, while SO_2 emissions were expected to be reduced by 3.5 million tons. The pattern of projected emission reductions for the proposed MACT standard is somewhat different than that for Clear Skies. The main difference is that the MACT standard are expected to see over 85 percent of the emissions

reductions for NO_x and SO_2 in the Ohio Valley, the Southeast, and the Mid-Atlantic (48 percent of NO_x reductions and 75 percent of SO_2 reductions were in the Ohio Valley alone). Very little emissions reductions are predicted for the Midwest or Western states. In contrast, only 57 percent of NO_x emission reductions and 74 percent of SO_2 emission reductions occurred in these regions based on Clear Skies, and even within these regions, were much more spread out. We have attempted to minimize these differences somewhat by focusing only on the results for the Eastern U.S., however, it is likely that our benefits estimates will have some remaining biases due to the differences in emission reductions patterns in the Eastern U.S. Because the reductions under the Utility MACT are more concentrated in areas that are upwind of major population centers, we expect that benefits per ton of emissions reduced will be somewhat higher on average for the Utility MACT than for Clear Skies. As such, we are likely to underestimate the benefits of the Utility MACT by transferring benefits from the Clear Skies analysis. However, we are not able to account for this quantitatively in our estimates. Table 2-3 summarizes the reductions in emissions of NO_x and SO_2 from baseline for Clear Skies and the proposed standard, the difference between the two, and the ratio of emissions reductions from the proposed standard to Clear Skies. The ratios presented in the last column of Table 2-3 are the basis for the benefits scaling approach discussed below.

Table 2-3.
Comparison of Modeled Emission Reductions
in 2010 Between Clear Skies and the Proposed Utility MACT Standard (Eastern U.S. Only)

Emissions Species	Reduction from Baseline		Difference in Reductions (Proposed MACT-Clear Skies)	Ratio of Reductions (Proposed MACT/ Clear Skies)
	Clear Skies	Proposed MACT		
NO_x	1,764,882	901,918	-862,964	0.511
SO_2	3,526,491	591,459	-2,935,032	0.168

2.3   Summary of Modeled Benefits and Apportionment Method

Based on the air quality modeling conducted for the Clear Skies analysis, we conducted a benefits analysis to determine human health benefits resulting from the reductions in emissions of NO_x and SO_2. We used the air quality modeling results from the Clear Skies assessment. However, we have updated the health impact and valuation approaches to be consistent with those used in the upcoming proposed Interstate Air Quality rule analysis. The Clear Skies analysis is available on the internet at http://www.epa.gov/clearskies. The benefits analysis for the proposed Interstate Air Quality rule is documented in U.S. EPA, 2003b.

The reductions in emissions of NO_x and SO_2 from fossil-fuel fired utilities in the United States are expected to result in wide-spread overall reductions in ambient concentrations of

PM$_{2.5}$. These improvements in air quality are expected to result in substantial health benefits, based on the body of epidemiological evidence linking PM with health effects such as premature mortality, cardiovascular disease, chronic lung disease, hospital admissions, and acute respiratory symptoms. Based on modeled changes in ambient concentrations of PM$_{2.5}$, we estimate changes in the incidence of each health effect using health impact functions derived from the epidemiological literature with appropriate baseline populations and incidence rates. We then apply estimates of the dollar value of each health effect to obtain a monetary estimate of the total PM-related health benefits of the rule.

2.3.1   Overview of Analytical Approach

This section summarizes our analysis of the modeled air quality changes from the Clear Skies assessment to determine the changes in human health and welfare, both in terms of physical effects and monetary value that result from modeled changes in PM$_{2.5}$. The methodology closely follows that used in the analyses of the proposed Nonroad Diesel rule and proposed Interstate Air Quality rule. Details of the analytical approach can be found in the Regulatory Impact Analyses for these rules (U.S. EPA, 2003a, 2003b) and in the User's Manual for the environmental Benefits Mapping and Analysis Program (BenMAP) (Abt Associates, 2003).

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. This imposes no overall preference structure, and does not account for potential income or substitution effects, i.e. adding a new endpoint will not reduce the value of changes in other endpoints. The "damage-function" approach is the standard approach for most cost-benefit analyses of regulations affecting environmental quality, and it has been used in several recent published analyses (Banzhaf et al., 2002; Levy et al, 2001; Kunzli et al, 2000; Levy et al, 1999; Ostro and Chestnut, 1998). Time and resource constraints prevented us from performing extensive new research to measure either the health outcomes or their values for this analysis. Thus, similar to these studies, 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 available for the environmental quality change under analysis.

There are significant categories of PM-related benefits that cannot be monetized (or in many cases even quantified), and thus they are not included in our accounting of health and welfare benefits. These unquantified effects include low birth weight, changes in pulmonary function, chronic respiratory diseases other than chronic bronchitis, morphological changes, altered host defense mechanisms, non-fatal cancers, and non-asthma respiratory emergency room visits. A complete discussion of PM related health effects can be found in the PM Criteria Document (U.S. EPA, 1996). Since many health effects overlap, such as minor restricted activity

days and asthma symptoms, we made assumptions intended to reduce the chances of "double-counting" health benefits, which may result in an underestimate of the total health benefits of the pollution controls.

2.3.2 Health Impact Functions

Health impact functions are derived from the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics like the Centers for Disease Control), the affected population, and the estimated change in the relevant ozone summary measure.

A typical health impact function might look like:

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

where y_0 is the baseline incidence, equal to the baseline incidence rate times the potentially affected population, β is the effect estimate, and Δx is the estimated change in the summary $PM_{2.5}$ measure. There are other functional forms, but the basic elements remain the same.

Integral to the estimation of the impact functions are reasonable estimates of future population projections. The underlying data used to create county-level 2010 population projections is based on county level allocations of national population projections from the U.S. Census Bureau (Hollman, Mulder and Kallan, 2000). County-level allocations of populations by age, race, and sex are based on economic forecasting models developed by Woods and Poole, Inc (WP), which account for patterns of economic growth and migration.

The WP projections of county level population are based on historical population data from 1969-1999, and do not include the 2000 Census results. Given the availability of detailed 2000 Census data, we constructed adjusted county level population projections for each future year using a two stage process. First, we constructed ratios of the projected WP populations in a future year to the projected WP population in 2000 for each future year by age, sex, and race. Second, we multiplied the block level 2000 Census population data by the appropriate age, sex, and race specific WP ratio for the county containing the census block, for each future year. This results in a set of future population projections that is consistent with the most recent detailed census data.

Specific populations matching the study populations in each epidemiological study are constructed by accessing the appropriate age-specific projections from the overall population database. For some endpoints, such as asthma attacks, we further limit the population by applying disease prevalence rates to the overall population. We do not have sufficient information to quantitatively characterize uncertainty in the population estimates.

Fundamental to the estimation of health benefits was our utilization of the PM epidemiology literature. We rely upon effect estimates derived from published epidemiological studies that relate health effects to ambient concentrations of PM. The specific studies from which effect estimates are drawn are listed in Table 2-4. While a broad range of serious health effects have been associated with exposure to elevated PM levels, we include only a subset of health effects in this benefit analysis due to limitations in available effect estimates and concerns about double-counting of overlapping effects (U.S. EPA, 1996). For the most part, we use the same set of effect estimates as we used in the analysis of the proposed Nonroad Diesel Engines rule. However, based on recent advice from the Science Advisory Board, we use an updated effect estimate for premature mortality and include two additional health effects, infant mortality and asthma exacerbations. Because of their significance in the analysis, we provide a more detailed discussion of premature mortality and chronic illness endpoints below. Complete details on the effect estimates used in the analysis can be found in the benefits analysis for the proposed Interstate Air Quality rule (U.S. EPA, 2003) and the BenMAP User's Manual (Abt Associates, 2003).

To generate health outcomes, projected changes in ambient PM concentrations were entered into BenMAP, a customized geographic information system based program. BenMAP aggregates populations to air quality model grids and calculates changes in air pollution metrics (e.g., daily averages) for input into health impact functions. BenMAP uses grid cell level population data and changes in pollutant concentrations to estimate changes in health outcomes for each grid cell. Details on the BenMAP program can be found in the BenMAP User's Manual (Abt Associates, 2003).

The baseline incidences for health outcomes used in our analyses are selected and adapted to match the specific populations studied. For example, we use age- and county-specific baseline total mortality rates in the estimation of PM-related premature mortality. County-level incidence rates are not available for other endpoints. We used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, 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. Sources of baseline incidence rates are reported in Table 2-5.

In this assessment we made analytical judgements affecting both the selection of effect estimates and the application of those estimates in formulating health impact functions. In general, we selected effect estimates that 1) most closely match the pollutants of interest, i.e. $PM_{2.5}$) cover the broadest potentially exposed population (i.e. all ages functions would be preferred to adults 27 to 35), 3) have appropriate model specification (e.g. control for confounding pollutants), 4) have been peer-reviewed, and 5) are biologically plausible. Other factors may also affect our selection of effect estimates for specific endpoints, such as premature mortality. Some of the more important of these relating to premature mortality and chronic illness are discussed below. Alternative assumptions about these judgements may lead to substantially different results.

While there is a consistent body of evidence supporting a relationship between a number of adverse health effects and ambient PM levels, there is often only a single study of a specific endpoint covering a specific age group. There may be multiple estimates examining subgroups (i.e. asthmatic children). However, for the purposes of assessing national population level benefits, we chose the most broadly applicable effect estimate to more completely capture health benefits in the general population.

Based on a review of the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope et al, 2002; Rossi et al., 1999; Schwartz, 2000), we chose for the purposes of this analysis to assume that PM-related health effects occur down to natural background (i.e. there is no health effects threshold). We assume that all of the health impact functions are continuous and differentiable down to natural background levels. Our assumptions regarding thresholds are supported as being plausible by the National Research Council in its recent review of methods for estimating the public health benefits of air pollution regulations. In their review, the National Research Council did not find evidence for departing from linearity in the observed range of exposure to PM_{10} or $PM_{2.5}$, nor any indication of a threshold. They cite the weight of evidence available from both short and long term exposure models and the similar effects found in cities with low and high ambient concentrations of PM.

Premature Mortality

As recommended by the SAB-HES (2003), we focus on the prospective cohort long-term exposure studies in deriving the health impact function for our base estimate of premature mortality. We selected an effect estimate from the extended analysis of the American Cancer Society (ACS) cohort (Pope et al., 2002). This effect estimate quantifies the relationship between annual mean $PM_{2.5}$ levels and all-cause mortality in adults 30 and older. We selected the effect estimate based on the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979-1984 and 1999-2000 PM2.5 levels. EPA is investigating ways of characterizing the uncertainty in the concentration-response function estimates.

In previous analyses, infant mortality has not been evaluated as part of the primary analysis. Instead, benefits estimates related to reduced infant mortality have been included as part of the sensitivity analyses. However recently published studies have strengthened the case for an association between PM exposure and respiratory inflamation and infection leading to premature mortality in infants under five years of age. Specifically, the SAB's Health Effects Subcommittee (HES) noted the release of the World Health Organization Global Burden of Disease Study focusing on ambient air which cites several recently-published time-series studies relating daily PM exposure to mortality in children (EPA-SAB-COUNCIL-ADV-03-00x). The HES also cites the study by Belanger et al., (2003) as corroborating findings linking PM exposure to increased respiratory inflamation and infections in children. With regard to the cohort study conducted by Woodruff et al. (1997), the HES notes several strengths of the study including the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control for a variety of individual risk factors in children (e.g., maternal educational level, maternal

ethnicity, parental marital status and maternal smoking status). We follow the HES recommendation to include infant mortality in the primary benefits estimate using the effect estimate from the Woodruff et al. (1997) study.

Chronic Illness

Although there are several studies examining the relationship between PM of different size fractions and incidence of chronic bronchitis, we use a study by Abbey et al (1995) to obtain our estimate of avoided incidences of chronic bronchitis in adults aged 25 and older, because Abbey et al (1995) is the only available estimate of the relationship between $PM_{2.5}$ and chronic bronchitis. Based on the Abbey et al study, we estimate the number of new chronic bronchitis cases that will "reverse" over time and subtract these reversals from the estimate of avoided chronic bronchitis incidences. Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Given the relatively high value assigned to chronic bronchitis, this ensures that we do not overstate the economic value of this health effect.

Non-fatal heart attacks have been linked with short term exposures to $PM_{2.5}$ in the U.S. (Peters et al, 2001) and other countries (Poloniecki et al, 1997). We use a recent study by Peters et al. (2001) as the source for the effect estimate quantifying the relationship between $PM_{2.5}$ and non-fatal heart attacks in adults. 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 et al (2000) show a consistent relationship between all cardiovascular hospital admissions, including for non-fatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we choose to provide a separate estimate for non-fatal heart attacks 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 U.S. These studies provide a weight of evidence for this type of effect. 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 is consistent with an increased risk of heart attacks.

2.3.3 Economic Values for Health Outcomes

Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore willingness-to-pay (WTP) for changes in risk prior to the regulation (Freeman, 1993). 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. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect, reflecting the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect (Harrington and Portney, 1987; Berger, 1987). Unit values for health endpoints are provided in Table 2-6. All values are in constant year 1999 dollars.

The size of the delay between changes in chronic PM exposures and changes in mortality rates is unknown. The size of such a time lag is important for the valuation of premature mortality incidences as economic theory suggests benefits occurring in the future should be discounted relative to benefits occurring today. Although there is no specific scientific evidence of the size of PM effects lag, current scientific literature on adverse health effects associated with smoking and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure would not occur in the same year as the exposure reduction. This literature implies that lags of a few years or longer are plausible. For our analysis, we have assumed a five-year distributed lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years[4]. To account for the preferences of individuals for current risk reductions relative to future risk reductions, we discount the value of avoided premature mortalities occurring beyond the analytical year (2010) using three and seven percent discount rates.

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. The economics literature suggests that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP (Alberini, 1997; Miller, 2000; Evans and Viscusi, 1993). As such, we use different factors to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. Adjustment factors used to account for projected growth in real income from 1990 to 2010 are 1.03 for minor health effects, 1.11 for severe and chronic health effects, and 1.10 for premature mortality.

2.3.4   Treatment of Uncertainty

In any complex analysis, there are likely to be many sources of uncertainty. This analysis is no exception. Many inputs are used to derive the final estimate of economic benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological effect estimates, estimates of values, population estimates, income estimates,

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[4] The SAB-HES has recently recommended that EPA rethink the use of a 5-year lag. They recommend that a more complex lag structure be considered incorporation components dealing with short-term (0-6 months), intermediate (1-2 years) and long-term (15-25 years) exposures. EPA is evaluating techniques for characterizing lag structures and will incorporate new methods as they become available.

and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Some of the key uncertainties in the benefits analysis are presented in Table 2-7. For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. For other parameters or inputs, the necessary information is not available.

In addition to uncertainty, the annual benefit estimates presented in this analysis are also inherently variable due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as electricity demand and weather display constant variability regardless of our ability to accurately measure them. As such, the estimates of annual benefits should be viewed as representative of the magnitude of benefits expected, rather than the actual benefits that would occur every year.

A key source of uncertainty for this analysis is the scaling approach used to estimate the benefits associated with the emission reductions for the Utility MACT. As noted earlier, while we believe this to be a valid approach, we are unable to quantify any uncertainties related to the scaling approach. To the extent that the effectiveness in reducing ambient $PM_{2.5}$ of each ton of NO_x and SO_2 reduced by the Utility MACT over or understates the effectiveness of the tons reduced by Clear Skies, the benefits of the Utility MACT will be over or underestimated.

2.3.5   Results of Revised Clear Skies Analysis

In order to generate benefits estimates consistent with the analytical assumptions underlying the benefits estimates for the Interstate Air Quality Rule, we have revised the Clear Skies benefits analysis to use a consistent set of assumptions. Based on the application of the health impact functions to the modeled changes in ambient PM2.5, we estimated the change in incidence and economic value of health effects for the updated set of health endpoints listed in Table 2-4. The results of the estimation are provided in Table 2-8. Compared to the original Clear Skies benefits analysis, the updated estimates show an increase in avoided cases of premature mortality due to the change in the effect estimate and slight changes in other endpoints, due to minor changes in the set of air quality monitoring data used in defining the change in ambient $PM_{2.5}$.

Table 2-4. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

Endpoint	Study	Study Population
Premature Mortality		
Premature Mortality - Adult, all-cause	Pope et al. (2002)	>29 years
Premature Mortality - Infant	Woodruff et al. (1997)	<1
Chronic Illness		
Chronic Bronchitis	Abbey, et al. (1995)	> 26 years
Non-fatal Heart Attacks	Peters et al. (2001)	Adults
Hospital Admissions		
Respiratory	Pooled estimate: Moolgavkar (2003) - ICD 490-496 (COPD) Ito (2003) - ICD 490-496 (COPD)	> 64 years
	Moolgavkar (2000) - ICD 490-496 (COPD)	20-64 years
	Ito (2003) - ICD 480-486 (pneumonia)	> 64 years
	Sheppard, et al. (2003) - ICD 493 (asthma)	< 65 years
Cardiovascular	Pooled estimate: Moolgavkar (2003) - ICD 390-429 (all cardiovascular) Ito (2003) - ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	> 64 years
	Moolgavkar (2000) - ICD 390-429 (all cardiovascular)	20-64 years
Asthma-Related ER Visits	Norris et al. (1999)	0-18 years
Other Health Endpoints		
Acute Bronchitis	Dockery et al. (1996)	8-12 years
Upper Respiratory Symptoms	Pope et al. (1991)	Asthmatics, 9-11 years
Lower Respiratory Symptoms	Schwartz and Neas (2000)	7-14 years
Asthma Exacerbations	Pooled estimate: Ostro et al. (2001) Cough Ostro et al. (2001) Wheeze Ostro et al. (2201) Shortness of breath Vedal et al. (1998) Cough	6-18 years[A]
Work Loss Days	Ostro (1987)	18-65 years
Minor Restricted Activity Days	Ostro and Rothschild (1989)	18-65 years

[B] The original study populations were 8-13 for the Ostro et al (2001) study and 6-13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES, we have extended the applied population to 6-18, reflecting the common biological basis for the effect in children in the broader age group.

Table 2-5
Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions

Endpoint	Parameter	Rates	
		Value	Source[1]
Mortality	Daily or annual mortality rate	Age, cause, and county-specific rate	CDC Wonder (1996-1998)
Hospitalizations	Daily hospitalization rate	Age, region, cause-specific rate	1999 NHDS public use data files[2]
Asthma ER visits	Daily asthma ER visit rate	Age, Region specific visit rate	2000 NHAMCS public use data files[3]; 1999 NHDS public use data files[2]

Chronic Bronchitis	Annual prevalence rate per person
	     Age 18-44
	     Age 45-64
	     Age 65 and older
		
		0.0367
		0.0505
		0.0587
			1999 HIS (American Lung Association, 2002b, Table 4)

	Annual incidence rate per person	0.00378	Abbey et al. (1993, Table 3)

Nonfatal MI (heart attacks)	Daily nonfatal myocardial infarction incidence rate per person, 18+
	     Northeast
	     Midwest
	     South
	     West
		
		0.0000159
		0.0000135
		0.0000111
		0.0000100
			1999 NHDS public use data files[2]; adjusted by 0.93 for prob. of surviving after 28 days (Rosamond et al., 1999)

Asthma Exacerbations	Incidence (and prevalence) among asthmatic African American children - daily wheeze - daily cough - daily dyspnea	 0.076 (0.173) 0.067 (0.145) 0.037 (0.074)	Ostro et al. (2001)
	Prevalence among asthmatic children - daily wheeze - daily cough - daily dyspnea	 0.038 0.086 0.045	Vedal et al. (1998)
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002a, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[4]	0.0012	Schwartz (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)
	     Age 18-24
	     Age 25-44
	     Age 45-64
		
		0.00540
		0.00678
		0.00492
			1996 HIS (Adams et al., 1999, Table 41); U.S. Bureau of the Census (2000)

Endpoint	Parameter	Rates	
		Value	Source[1]
Minor Restricted Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

1. 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.

2. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/

3. See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

4. Lower Respiratory Symptoms are defined as ≥ 2 of the following: cough, chest pain, phlegm, wheeze

Table 2-6
Unit Values Used for Economic Valuation of Health Endpoints (2000$)

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Estimates
	1990 Income Level	2010 Income Level	
Premature Mortality	$5,500,000	$6,100,000	Point estimate is the mean of a normal distribution with a 95% confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature. $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2000) meta-analysis. $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003) meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population.
Chronic Bronchitis (CB)	$340,000	$370,000	Base value is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Estimates
	1990 Income Level	2010 Income Level	
Non-fatal Myocardial Infarction (heart attack) 3% discount rate Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over 7% discount rate Age 0-24 Age 25-44 Age 45-54 Age 55-65 Age 66 and over	 $66,902 $74,676 $78,834 $140,649 $66,902 $65,293 $73,149 $76,871 $132,214 $65,293	 $66,902 $74,676 $78,834 $140,649 $66,902 $65,293 $73,149 $76,871 $132,214 $65,293	Age specific cost-of-illness values reflecting lost earnings and direct medical costs over a 5 year period following a non-fatal MI. Lost earnings estimates based on Cropper and Krupnick (1990). Direct medical costs based on simple average of estimates from Russell et al. (1998) and Wittels et al. (1990). Lost earnings: Cropper and Krupnick (1990). Present discounted value of 5 yrs of lost earnings: age of onset: at 3% at 7% 25-44 $8,774 $7,855 45-54 $12,932 $11,578 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-yr period. ($22,331 at 3% discount rate; $21,113 at 7% discount rate)
Hospital Admissions			
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	$12,378	$12,378	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).
Pneumonia (ICD codes 480-487)	$14,693	$14,693	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 pneumonia category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).

Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Estimates
	1990 Income Level	2010 Income Level	
Asthma admissions	$6,634	$6,634	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 (ICD codes 390-429)	$18,387	$18,387	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).
Emergency room visits for asthma	$286	$286	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			
Asthma Exacerbations	$42	$43	Asthma exacerbations are valued at $42 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 estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study.
Upper Respiratory Symptoms (URS)	$25	$26	Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 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. The dollar value for URS is the average of the dollar values for the 7 different types of URS.

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Health Endpoint	Central Estimate of Value Per Statistical Incidence		Derivation of Estimates
	1990 Income Level	2010 Income Level	
Lower Respiratory Symptoms (LRS)	$16	$17	Combinations of the 4 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.
Acute Bronchitis	$360	$370	Assumes a 6 day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann, et al. 1994.
Restricted Activity and Work Loss Days			
Work Loss Days (WLDs)	Variable (national median = $115)	Variable (national median = $115)	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	$53	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .

Table 2-7
Primary Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Health Impact Functions
– The value of the PM effect estimate in each impact function. – Application of a single effect estimate to pollutant changes and populations in all locations. – Similarity of future year effect estimates to current effect estimates. – Correct functional form of each impact function. – Application of effect estimates to changes in PM outside the range of PM concentrations observed in the study. – Application of effect estimates only to those subpopulations matching the original study population.
2. Uncertainties Associated With PM Concentrations
– Responsiveness of the models to changes in precursor emissions. – Projections of future levels of precursor emissions, especially ammonia and crustal materials. – Model chemistry for the formation of ambient nitrate concentrations. – Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions. – Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
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 limited ambient $PM_{2.5}$ 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 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 may therefore not accurately represent the actual location-specific rates. – Current baseline incidence rates may not approximate well baseline incidence rates in 2010. – 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 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 due to differences in income or other factors.
7. Uncertainties Associated With Aggregation of Monetized Benefits
– Health benefits estimates are limited to the available effect estimates. Thus, unquantified or unmonetized benefits are not included.

Table 2-8.
Results of Revised Clear Skies Benefits Analysis

Endpoint	Cases Avoided[A]	Economic Value (Millions of 2000$)
Premature mortality -		
Long-term exposure (adults, 30 and over)[B]	9,800	$60,000
Long-term exposure (infant, <1 yr)	23	$140
Chronic bronchitis (adults, 26 and over)	5,300	$2,000
Non-fatal myocardial infarctions (adults, 18 and older)[C]	13,000	$1,100
Hospital admissions – Respiratory (all ages)[D]	4,300	$76
Hospital admissions – Cardiovascular (adults, 20 and older)[E]	3,700	$82
Emergency Room Visits for Asthma (18 and younger)	7,400	$2.1
Acute bronchitis (children, 8-12)	12,000	$4.5
Lower respiratory symptoms (children, 7-14)	150,000	$2.4
Upper respiratory symptoms (asthmatic children, 9-11)	110,000	$3.0
Asthma exacerbations	190,000	$8.5
Work loss days (adults, 18-65)	1,000,000	$130
Minor restricted activity days (adults, age 18-65)	6,200,000	$320
Total Economic Value of Health Benefits[F]		$64,000

[A] Incidences and values are rounded to two significant digits.
[B] Economic value calculated using a 3 percent discount rate. Economic value using a 7 percent discount rate is $57,000 million.
[C] Economic value calculated using a 3 percent discount rate. Economic value using a 7 percent discount rate is also $1,100 million (difference is within the margin of rounding).
[D] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.
[E] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.
[F] Total economic value calculated using 3 percent discount rate results. Total economic value using a 7 percent discount rate is $61,000 million.

2.3.6 Apportionment of Benefits to NO_x and SO_2 Emissions Reductions

    In order to develop benefits estimates for the set of emission reductions expected to result from the proposed MACT standard, it is necessary for us to scale the Clear Skies based benefits to reflect the difference in emissions reductions between the proposed MACT standards and the Clear Skies analysis. In order to do so, however, we must first apportion total benefits to the NO_x and SO_2 reductions for the modeled Clear Skies scenario. This apportionment is necessary due to the differential contribution of each emission species to the total change in ambient PM

and total benefits.

PM is a complex mixture of particles of varying species, including nitrates, sulfates, and primary particles, including organic and elemental carbon. These particles are formed in complex chemical reactions from emissions of precursor pollutants, including NO_x, SO_2, ammonia, hydrocarbons, and directly emitted particles. Different emissions species contribute to the formation of PM in different amounts, so that a ton of emissions of NO_x contributes to total ambient PM mass differently than a ton of SO_2. As such, it is inappropriate to scale benefits by simply scaling the sum of all precursor emissions. A more appropriate scaling method is to first apportion total PM benefits to the changes in underlying emission species and then scale the apportioned benefits.

PM formation relative to any particular reduction in an emission species is a highly nonlinear process, depending on meteorological conditions and baseline conditions, including the amount of available ammonia to form ammonium nitrate and ammonium sulfate. Given the limited air quality modeling conducted for this analysis, we make several simplifying assumptions about the contributions of emissions reductions for specific species to changes in particle species. For this exercise, we assume that changes in sulfate particles are attributable to changes in SO_2 emissions, and changes in nitrate and secondary organic particles are attributable to changes in NO_x emissions. These assumptions essentially assume independence between SO_2 and NO_x in the formation of ambient PM. This is a potentially significant source of uncertainty, as SO_2 and NO_x emissions interact with other compounds in the atmosphere to form $PM_{2.5}$. For example, ammonia reacts with SO_2 first to form ammonium sulfate. If there is remaining ammonia, it reacts with NO_x to form ammonium nitrate. When SO_2 alone is reduced, ammonia is freed to react with any NO_x that has not been used in forming ammonium nitrate. If NO_x is also reduced, then there will be less available NO_x to form ammonium nitrate from the newly available ammonia. Thus, reducing SO_2 can potentially lead to decreased ammonium sulfate and increased nitrate, so that overall ambient PM benefits are less than the reduction in sulfate particles. If NO_x alone is reduced, there will be a direct reduction in ammonium nitrate, although the amount of reduction depends on whether an area is ammonia limited. If there is not enough ammonia in an area to use up all of the available NO_x, then NO_x reductions will only have an impact if they reduce emissions to the point where ammonium nitrate formation will be affected. NO_x reductions will not result in any offsetting increases in ambient PM under most conditions. The implications of this for apportioning benefits between NO_x and SO_2, is that some of the sulfate related benefits will be offset by reductions in nitrate benefits, so benefits from SO_2 reductions will be overstated, while NO_x benefits will be understated. It is not immediately apparent the size of this bias.

The measure of change in ambient particle mass that is most related to health benefits is the population-weighted change in $PM_{2.5}$ $\mu g/m^3$, because health benefits are driven both by the size of the change in $PM_{2.5}$ and the populations exposed to that change. We calculate the proportional share of total change in mass accounted for by sulfate particles and the sum of nitrate and secondary organic particles. Results of these calculations for the 2010 Clear Skies REMSAD modeling analysis are presented in Table 2-9. The sulfate percentage of total change

is used to represent the SO_2 contribution to health benefits and the nitrate plus secondary organics percentage is used to represent the NO_x contribution to health benefits. These percentages are then applied to the PM-related health benefits estimates from the analysis of the Clear Skies $PM_{2.5}$ air quality modeling and combined with the emission scaling factors developed in section 2.2 to estimate benefits for the proposed Utility MACT standard.

Table 2-9. Apportionment of Population Weighted Change in Ambient $PM_{2.5}$ to Nitrate, Sulfate, and Secondary Organic Particles

	Population-weighted Change (μg/m3)	Percent of Total Change
Total PM2.5	0.610	
Sulfate	0.520	85.2%
Nitrate	0.081	13.4%
Secondary Organic	0.008	1.4%

2.4   Estimated Benefits of Proposed MACT Standard in 2010

To estimate the benefits of the NO_x and SO_2 emission reductions from the proposed standard in 2010, we apply the emissions scaling factors derived in section 2.2 and the apportionment factors described in section 2.3.6 to the benefits estimates for 2010 estimated using the Clear Skies $PM_{2.5}$ air quality modeling. The scaled avoided incidence estimate for any particular health endpoint is calculated using the following equation:

$$Scaled\ Incidence = Modeled\ Incidence * \sum_i R_i A_i \ ,$$

where Modeled Incidence is the estimated change in incidence of the health effect from the updated Clear Skies analysis from Table 2-8, R_i is the emissions ratio for emission species i from Table 2-3, and A_i is the health benefits apportionment factor for emission species i, from Table 2-9. Essentially, benefits are scaled using a weighted average of the species specific emissions ratios. For example, the calculation of the avoided incidence of premature mortality in 2010 is:

Scaled Premature Mortality Incidence = 9,800 * (0.852*0.168 + 0.147*0.574) = 2,200

The economic value for each endpoint is obtained by scaling the estimated Clear Skies economic value from Table 2-8 using the same function. The estimated changes in incidence and economic value of PM-related health effects in 2010 for the proposed Utility MACT standard based on application of the weighted scaling factors are presented in Table 2-10.

The benefits estimates generated for the proposed rule are subject to a number of assumptions and uncertainties, which are discussed throughout the document. As the table indicates, total benefits are driven primarily by the reduction in premature fatalities each year, which account for over 90 percent of total benefits. Key assumptions underlying the primary estimate for the mortality category include the following:

(1)   Inhalation of fine particles is causally associated with premature death at concentrations near those experienced by most Americans on a daily basis. Although biological mechanisms for this effect have not yet been definitively established, the weight of the available epidemiological evidence supports an assumption of causality.

(2)   All fine particles, regardless of their chemical composition, are equally potent in causing premature mortality. This is an important assumption, because PM produced via transported precursors emitted from EGUs may differ significantly from direct PM released from automotive engines and other industrial sources, but no clear scientific grounds exist for supporting differential effects estimates by particle type.

(3)   The C-R function for fine particles is approximately 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, including both regions that are in attainment with fine particle standard and those that do not meet the standard.

Although recognizing the difficulties, assumptions, and inherent uncertainties in the overall enterprise, these analyses are based on peer-reviewed scientific literature and up-to-date assessment tools, and we believe the results are highly useful in assessing this proposal.

Table 2-10.
Estimated PM-related Health Benefits of the Proposed Utility MACT Standards in 2010

Endpoint	Cases Avoided[A]	Economic Value (Millions of 1999$)
Premature mortality -		
Long-term exposure (adults, 30 and over)[B]	2,200	$14,000
Long-term exposure (infant, <1 yr)	5	$32
Chronic bronchitis (adults, 26 and over)	1,200	$460
Non-fatal myocardial infarctions (adults, 18 and older)[C]	2,900	$250
Hospital admissions – Respiratory (all ages)[D]	980	$17
Hospital admissions – Cardiovascular (adults, 20 and older)[E]	850	$19
Emergency Room Visits for Asthma (18 and younger)	1,700	$0.48
Acute bronchitis (children, 8-12)	2,800	$1.0
Lower respiratory symptoms (children, 7-14)	33,000	$0.54
Upper respiratory symptoms (asthmatic children, 9-11)	25,000	$0.69
Asthma exacerbations	43,000	$1.9
Work loss days (adults, 18-65)	240,000	$31
Minor restricted activity days (adults, age 18-65)	1,400,000	$74
Total Economic Value of Health Benefits[F]		$15,000

[A] Incidences and values are rounded to two significant digits.

[B] Economic value calculated using a 3 percent discount rate. Economic value using a 7 percent discount rate is $13,000 million.

[C] Economic value calculated using a 3 percent discount rate. Economic value using a 7 percent discount rate is $250 million.

[D] Respiratory hospital admissions for PM includes admissions for COPD, pneumonia, and asthma.

[E] Cardiovascular hospital admissions for PM includes total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

[F] Total economic value calculated using 3 percent discount rate results. Total economic value using a 7 percent discount rate is $14,000 million.

2.5 Welfare Effects

There are a number of environmental resources which may be adversely affected by emissions of NO_x and SO_2 or ambient $PM_{2.5}$ Changes in these environmental resources may affect human welfare, but due to a lack of appropriate physical effects or valuation methods, we are unable to quantify or monetize these effects for our analysis of the proposed MACT standard. Qualitative discussions of these benefits are provided in Section 1. A brief discussion of some of the benefits which are known to have significant economic value is provided below.

Changes in the level of ambient particulate matter caused by the reduction in emissions from fossil-fuel fired utility sources will change the level of visibility in much of the U.S. Visibility directly affects people's enjoyment of a variety of daily activities. 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 Grand Canyon.

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_x caused by the proposed rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999). Direct impact functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these impact functions and on estimates of the value of environmental responses. Because neither appropriate impact functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible.

Reductions in NO_x emissions will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects.

The proposed Utility MACT standard are also expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefit analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970's) are too out of date to provide a reliable enough estimate of current household soiling damages (EPA-SAB-Council-ADV-003, 1998) to include in our primary estimate. We are also unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions.   For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

2.6 Comparison of Costs and Benefits

Table 2-11 summarizes the results of the benefit-cost analysis of the proposed section 112 MACT scenario and compares them with estimates of the range of potential costs and benefits associated with an alternative scenario that addresses combined implementation of section 111 Hg requirements in coordination with proposed SO_2 and NO_x requirements in the proposed IAQR. The potential influence of such a combined scenario is illustrated in the second column of Table 2-11, which assumes the proposed section 111 requirements are implemented in combination with the IAQR. The IAQR analysis projects that the Hg reductions associated with implementing the SO_2/NO_x requirements in the Eastern U.S. in 2010 would be approximately 10.6 tons per year, which is almost identical to those estimated from the proposed section 112 MACT-only scenario.

If the goal for the proposed section 111 program in 2010 is limited to these co-control reductions, there might be no additional costs or benefits to the program, over those achieved by the IAQR – this is indicated in the lower portion of the ranges in Table 2-11. By contrast, if the proposed section 111 regulation adopts a 2010 goal similar to the Phase I Clear Skies Hg cap, additional Hg reductions would be required over those forecast for the IAQR. Based on a multipollutant analyses conducted for Clear Skies (p D-9, Technical appendix D, at www.epa.gov/airmarkets/epa-ipm), power generators would likely opt for some additional SO_2 and NO_x controls beyond those needed for the IAQR, as well as considering additional direct Hg controls. Although the actual results are uncertain, the Clear Skies results suggest that the costs and benefits associated with a section 112 MACT-only approach may reflect a reasonable lower bound for the additional costs and benefits. These potential additional costs and benefits related to additional Hg controls are reflected in the upper end of the ranges in Table 2-11. In the decade beyond 2010, the proposed section 111 program would establish a 15 ton cap for Hg in 2018, similar to Clear Skies. Based on Clear Skies analyses, this would result in further Hg controls, which would likely include at least some additional SO_2/NO_x controls as well as direct Hg controls. The IAQR program alone produces only small additional reductions in Hg emissions in 2020. The Hg reductions estimated for the proposed section 112 MACT and the proposed section 111 and proposed IAQR programs are summarized in Table 2-12. These forecasts are based on IPM analyses of the proposed section 112 MACT scenario outlined above, the proposed IAQR analysis, and estimates derived from earlier analyses of the Clear Skies program.

Every benefit-cost analysis examining the potential effects of a change in environmental protection requirements is limited, to some extent, by data gaps, limitations in model capabilities

(such as geographic coverage), and uncertainties in the underlying scientific and economic studies used to configure the benefit and cost models. Deficiencies in the scientific literature often result in the inability to estimate changes in health and environmental effects. Deficiencies in the economics literature often result in the inability to assign economic values even to those health and environmental outcomes that can be quantified. While these general uncertainties in the underlying scientific and economics literatures are discussed in detail in the RIA and its supporting documents and references, the key uncertainties which have a bearing on the results of the benefit-cost analysis of today's action are the following:

      1. The exclusion of potentially significant benefit categories (e.g., health and ecological benefits of reduction in hazardous air pollutants emissions);

      2. Errors in measurement and projection for variables such as population growth;

      3. Uncertainties in the estimation of future year emissions inventories and air quality;

      4. Uncertainties associated with the extrapolation of air quality monitoring data to some unmonitored areas required to better capture the effects of the standards on the affected population;

      5. Variability in the estimated relationships of health and welfare effects to changes in pollutant concentrations; and

      6. Uncertainties associated with the benefit transfer approach.

      Despite these uncertainties, we believe the benefit-cost analysis provides a reasonable indication of the expected economic benefits of the proposed actions under a given set of assumptions.

      Based on estimated compliance costs (control + administrative costs associated with Paperwork Reduction Act requirements associated with the proposed rule and predicted changes in the price and output of electricity), the estimated social costs of the proposed section 112 MACT-only scenario are $1.6 billion (1999$). Social costs are different from compliance costs in that social costs take into account the interactions between affected producers and the consumers of affected products in response to the imposition of the compliance costs. In this action, coal-fired utilities are the affected producers and users of electricity are the consumers of the affected product.

      As explained above, we estimate $15 billion in benefits from the proposed section 112 MACT, compared to less than $2 billion in costs. It is important to put the results of this analysis in the proper context. The large benefit estimate is not attributable to reducing human and environmental exposure to Hg. It arises from ancillary reductions in SO_2 and NO_x that result from controls aimed at complying with the proposed MACT. Although consideration of ancillary benefits is reasonable, we note that these benefits are not uniquely attributable to Hg

regulation. Under the IAQR, coal-fired units would achieve much larger reductions in SO_2 and NO_x emissions than they would under the proposed section 112 MACT. In the years ahead, as the Agency and the States develop rules, guidance and policies to implement the new air quality standards for ozone and PM, coal-fired power plants will be required to implement additional controls to reduce SO_2 and NO_x (e.g., scrubbers, SCR units, year-round NO_x controls in place of summertime only controls, conversion to low-sulfur coals, and so forth). Thus, most or all of the ancillary benefits of Hg control would be achieved anyway, regardless of whether a section 112 MACT is promulgated. Based on analysis of the Clear Skies legislation, EPA believes that the proposed 2018 Hg cap in the proposed section 111 rule would result in additional SO_2 and NO_x reductions beyond those that would be required under the proposed IAQR. Thus, the section 111 approach, unlike the section 112 approach, may achieve SO_2 and NO_x reduction benefits beyond those that would be achieved under the IAQR. We believe, however, that even if no Hg controls were imposed, most major coal-fired units would still have to reduce their SO_2 and NO_x emissions as part of the efforts to bring the nation into attainment with the new air quality standards. In light of these considerations, the Agency believes that the key rationale for controlling Hg is to reduce public and environmental exposure to Hg, thereby reducing risk to public health and wildlife. Although the available science does not support quantification of these benefits at this time, the Agency believes the qualitative benefits are large enough to justify substantial investment in Hg emission reductions.

It should be recognized, however, that this analysis does not account for many of the potential benefits that may result from these actions. The net benefits would be greater if all the benefits of the Hg, Ni, and other pollutant reductions could be quantified. Notable omissions to the net benefits include all benefits of HAP reductions, including reduced cancer incidences, toxic morbidity effects, and cardiovascular and CNS effects, and all health and welfare effects from reduction of ambient NO_x and SO_2.

Table 2-11. Summary of Monetized Benefits, Costs, and Net Benefits under the Proposed Section 112 MACT Standard, and the Proposed Section 111 Rule and the Proposed IAQR[A] ($billions/yr)

	MACT-only Scenario	Sec. 111 plus IAQR Combined[D]
Social Costs[B]	$1.6	$2.9 to 4.5+
Social Benefits[C]:		
PM-related Health benefits	$15+B	$58 to 73+B
Net Benefits (Benefits- Costs)[C]	$13+B	$55 to $68+B

[A] All costs and benefits are rounded to two significant digits.

[B] Note that costs are the total costs of reducing all pollutants, including Hg and other metallic air toxics, as well as NO_x and SO_2. Benefits in this table are associated only with NO_x and SO_2 reductions.

^C Not all possible benefits or disbenefits are quantified and monetized in this analysis. In particular, ozone health and welfare and PM welfare benefits are omitted. Other potential benefit categories that have not been quantified and monetized are listed in Table 2-1. B is the sum of all unquantified benefits and disbenefits.

^D Estimated combined benefits of Sec. 111 plus IAQR costs and benefits in 2010. Ranges do not reflect actual analyses of combined programs. Rough estimates based on consideration of available IAQR, MACT, and Clear Skies analyses. See text.

Table 2-12. Forecast Mercury Emissions under the Proposed Section 112 MACT, and the Proposed Section 111 Rule and the Proposed IAQR[A]

Program/Year	2010	2020
MACT only	34	31
IAQR only	34	30
IAQR and section 111 caps	[B]	18 - 22

[A] Annual reductions from base case forecast under current programs to reduce Utility Unit emissions. MACT only value for 2015 based on interpolation of 2010 and 2015. Lower bound of IAQR and section 111 caps in 2010 assumes Hg cap is set at co-control level achieved by IAQR. Upper bound in 2010 and ranges thereafter estimates derived from Clear Skies analyses.

[B] Mercury emissions will reflect the level of emissions resulting from the co-benefits of controlling SO_2 and NO_x. See section IV.B.1 for a detailed discussion.

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16. ABSTRACT

This document is a benefits analysis of the Section 112 (MACT) proposal to reduce mercury emissions from power plants. The analysis provided qualitative and quantitative estimates of the benefits from the emission reductions, which include reductions of nitrogen oxides and sulfur dioxide as well as mercury. Extensive background on the health effects from mercury exposure is also included.

17. KEY WORDS AND DOCUMENT ANALYSIS

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