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Regulatory Impact Analysis for the Federal Implementation Plans to Reduce Interstate Transport of Fine Particulate Matter and Ozone in 27 States; Correction of SIP Approvals for 22 States

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Chapter 5 Benefits Analysis and Results

Synopsis

This chapter contains a subset of the estimated health and welfare benefits of the Transport Rule remedy in 2014. This rule is expected to yield significant reductions in SO_2 and NO_x from EGUs, which in turn would lower overall ambient levels of $PM_{2.5}$ and ozone across much of the eastern U.S. In this chapter we quantify the health and welfare benefits resulting from these air quality improvements.

We estimate the monetized benefits of the selected remedy to be \$120 billion to \$280 billion at a 3% discount rate and \$110 billion to \$250 billion at a 7% discount rate in 2014. The benefits of the more and less stringent alternatives may be found in the benefit-cost comparison chapter. All estimates are in 2007\$. We estimate the benefits of the selected remedy using modeled changes in ambient pollution concentrations while the benefits of the more and less stringent remedies are based on a benefit per ton approach described below. This benefits analysis accounts for both decreases and increases in emissions across the country. These estimates omit the benefits from several important categories, including ecosystem benefits, mercury benefits, and the direct health benefits from reducing exposure to NO₂ and SO₂ due to time constraints.

The estimated benefits of this rule are substantial, particularly when viewed within the context of the total public health burden of $PM_{2.5}$ and ozone air pollution. A recent EPA analysis estimated that 2005 levels of $PM_{2.5}$ and ozone were responsible for between 130,000 and 320,000 $PM_{2.5}$ -related and 4,700 ozone-related premature deaths, or about 6.1% of total deaths from all causes in the continental U.S. (Fann et al. 2011). This same analysis attributed almost 200,000 non-fatal heart attacks, 90,000 hospital admissions due to respiratory or cardiovascular illness and 2.5 million cases of aggravated asthma among children--among many other impacts. We estimate the Transport Rule to reduce the number of $PM_{2.5}$ -related premature deaths in 2014 by between 13,000 and 34,000, 15,000 non-fatal heart attacks, 8,700 fewer hospital admissions and 400,000 fewer cases of aggravated asthma. By 2014, in combination with other federal and state air quality actions, the Transport Rule will address a substantial fraction of the total public health burden of $PM_{2.5}$ and ozone air pollution.

EPA expects greater emission reductions due to this rule in 2012 than in 2014, due to substantial emission reductions expected to occur in the baseline (i.e., unrelated to the Transport Rule) between those years. As a result, we anticipate that the avoided health impacts and monetized benefits would also be greater in 2012, though we have not calculated these estimates for this analysis.

Appendix A to this RIA contains an assessment of the distribution of health benefits among different populations. In this analysis, we considered the level of $PM_{2.5}$ mortality risk according to the race, income and educational attainment of the population before and after the implementation of the Transport Rule. We found those populations whose $PM_{2.5}$ mortality risk was before the implementation of the rule received the greatest risk reduction from the Transport Rule—irrespective of the race of the population. We also found that populations with lower levels of educational attainment, an attribute that may be associated with increased vulnerability to PM2.5 mortality risk, also received a significant reduction in risk.

Finally, Appendix E provides an alternate presentation of the benefits as an attempt to incorporate the recommendations from EPA's recently published Guidelines for Preparing Economic Analyses (U.S. EPA, 2010).

5.1 Overview

This chapter contains a subset of the estimated health and welfare benefits of the selected

and alternate rule remedies for the Transport Rule in 2014. The Transport Rule is expected to yield significant aggregate reductions in SO_2 and NO_x from EGUs, which in turn would lower overall ambient levels of $PM_{2.5}$ and ozone across much of the eastern U.S. To perform this analysis, EPA followed an approach that is generally consistent with the proposal Transport Rule analysis, with the exception of the baseline incidence rates that are an input into the health impact calculation for $PM_{2.5}$ and ozone health outcomes. These updated rates are both more current and provide better spatial resolution in many areas of the U.S. As we describe in section 5.4 below, these updated data are likely to yield a better overall estimate of PM and ozone-related health impacts.

The analysis in this chapter aims to characterize the benefits of the selected remedy by answering two key questions:

- 1. What are the health and welfare effects of changes in ambient particulate matter (PM_{2.5}) and ozone air quality resulting from reductions in precursors including NO_x and SO₂?
- 2. What is the economic value of these effects?

In this analysis we consider an array of health and welfare impacts attributable to changes in $PM_{2.5}$ and ozone air quality. The 2009 $PM_{2.5}$ Integrated Science Assessment (U.S. EPA, 2009d) and the 2006 ozone criteria document (U.S. EPA, 2006a) identify the human health effects associated with these ambient pollutants, which include premature mortality and a variety of morbidity effects associated with acute and chronic exposures. PM welfare effects include visibility impairment and materials damage. Ozone welfare effects include damages to agricultural and forestry sectors. NO_x welfare effects include aquatic and terrestrial acidification and nutrient enrichment (U.S. EPA, 2008f). SO₂ welfare effects include aquatic and terrestrial acidification and increased mercury methylation (U.S. EPA, 2008f). Though models exist for quantifying these ecosystem impacts, time and resource constraints precluded us from quantifying most of those effects in this analysis.

Table 5-1 summarizes the total monetized benefits of the final Transport Rule remedy in 2014. This table reflects the economic value of the change in $PM_{2.5}$ and ozone-related human health impacts occurring as a result of the Transport Rule.

Table 5-2 summarizes the human health and welfare benefits categories contained

within the primary benefits estimate, those categories that were unquantified due to limited data or time.

 Table 5-1: Estimated monetized benefits of the final Transport Rule remedy (billions of 2007\$)^A

		Outside		
	Within Transport	Transport		
Benefits Estimate	<i>Region^B</i>	Region	Total	
Pope et al. (2002) PM _{2.5} mortality and Bell et al. (2004) ozone mortality estimates				
Using a 3% discount rate	\$110 +B (\$8.8—\$340)	\$0.28 +B (\$0.01—\$0.9)	\$120 +B (\$14—\$350)	
Using a 7% discount rate	\$100 +B (\$8—\$310)	\$0.25 +B (\$0.01—\$0.85)	\$110 +B (\$13—\$320)	

Laden et al. (2006) PM_{2.5} mortality and Levy et al. (2005) ozone mortality estimates

Using a 3% discount rate	\$270 +B	\$0.7 +B	\$280 +B
	(\$24—\$800)	(\$0.05—\$0.21)	(\$29—\$810)
Using a 7% discount rate	\$250 +B	\$0.6 +B	\$250 +B
	(\$22—\$720)	(\$0.04—\$1.9)	(\$26—\$730)

^A For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. Data limitations prevented us from quantifying these endpoints, and as such, these benefits are inherently more uncertain than those benefits that we were able to quantify. A detailed listing of unquantified health and welfare effects is provided in Table 5-2. Estimates here are subject to uncertainties discussed further in the body of the document. Estimates include the value of CO_2 -related benefits and the monetized benefits of visibility improvements in Class I areas.

^BRounded to two significant figures.

The benefits analysis in this chapter relies on an array of data inputs—including air quality modeling, health impact functions and valuation estimates among others—which are themselves subject to uncertainty and may also in turn contribute to the overall uncertainty in this analysis. As a means of characterizing this uncertainty we employ two primary techniques. First, we use Monte Carlo methods for characterizing random sampling error associated with the concentration response functions from epidemiological studies and economic valuation functions. Second, because this characterization of random statistical error may omit important sources of uncertainty we also employ the results of an expert elicitation on the relationship between premature mortality and ambient $PM_{2.5}$ concentration (Roman et al., 2008); this provides additional insight into the likelihood of different outcomes and about the state of knowledge regarding the benefits estimates. Both approaches have different strengths and weaknesses, which are fully described in Chapter 5 of the PM NAAQS RIA (U.S. EPA, 2006).

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

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

Below we include a new analysis (Figures 5-19 and 5-20) in which we bin the estimated number of avoided $PM_{2.5}$ -related premature mortalities resulting from the implementation of the Transport Rule according to the projected 2014 baseline $PM_{2.5}$ air quality levels. This presentation is consistent with our approach to applying $PM_{2.5}$ mortality risk coefficients that have not been adjusted to incorporate an assumed threshold. The very large proportion of the avoided PM-related impacts we estimate in this analysis occur among populations exposed at or above the LML of each study, increasing our confidence in the PM mortality analysis. Approximately 69% of the avoided impacts occur at or above an annual

mean $PM_{2.5}$ level of 10 µg/m³ (the LML of the Laden et al. 2006 study); about 96% occur at or above an annual mean $PM_{2.5}$ level of 7.5 µg/m³ (the LML of the Pope et al. 2002 study). As we model mortality impacts among populations exposed to levels of $PM_{2.5}$ that are successively lower than the LML of each study our confidence in the results diminishes. However, the analysis below confirms that the great majority of the impacts occur at or above each study's LML.

 Table 5-2: Human Health and Welfare Effects of Pollutants Affected by the Transport Rule

Pollutant/	Quantified and monetized in base estimate	Unquantified
Effect	Premature mortality based on cohort study estimates ^b and expert elicitation estimates	Low birth weight, pre-term birth and other reproductive
	Hospital admissions: respiratory and cardiovascular	Pulmonary function
	Emergency room visits for asthma	Chronic respiratory diseases other than chronic bronchitis
	Nonfatal heart attacks (myocardial	Non-asthma respiratory emergency room visits
PM: health ^a	Lower and upper respiratory illness Minor restricted activity days Work loss days Asthma exacerbations (among asthmatic populations Respiratory symptoms (among asthmatic populations) Infant mortality	UVb exposure (+/-) ^c
PM: welfare	Visibility in Class I areas in SE, SW, and CA regions	Household soiling Visibility in residential areas Visibility in non-class I areas and class 1 areas in NW, NE, and Central regions UVb exposure (+/-) ^c Global climate impacts ^c
	Premature mortality based on short-term	Chronic respiratory damage
Ozone: health	Hospital admissions: respiratory Emergency room visits for asthma Minor restricted activity days School loss days	Premature aging of the lungs Non-asthma respiratory emergency room visits UVb exposure (+/-) ^c
Ozone: welfare	Decreased outdoor worker productivity	Yields for: Commercial forests Fruits and vegetables, and Other commercial and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions UVb exposure (+/-) ^c Climate impacts
NO ₂ : health		Respiratory hospital admissions Respiratory emergency department visits Asthma exacerbation Acute respiratory symptoms Premature mortality Pulmonary function
NO _X : welfare		Commercial fishing and forestry from acidic deposition effects Commercial fishing, agriculture and forestry from nutrient deposition effects Recreation in terrestrial and estuarine ecosystems from nutrient deposition effects Other ecosystem services and existence values for

	currently healthy ecosystems
	Coastal eutrophication from nitrogen deposition effects
	Respiratory hospital admissions
	Asthma emergency room visits
SO ₂ :	Asthma exacerbation
health	Acute respiratory symptoms
	Premature mortality
	Pulmonary function
	Commercial fishing and forestry from acidic deposition
SO .	effects
SUX:	Recreation in terrestrial and aquatic ecosystems from
wenare	acid deposition effects
	Increased mercury methylation
	Incidence of neurological disorders
Mercury:	Incidence of learning disabilities
health	Incidences in developmental delays
	T / 1'1 1 1 / 1 /
N	Impact on birds and mammals (e.g. reproductive
Mercury:	effects)
welfare	Impacts to commercial, subsistence and recreational fishing

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

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

^C May result in benefits or disbenefits.

5.2 Benefits Analysis Methods

We follow a "damage-function" approach in calculating total benefits of the modeled changes in environmental quality. This approach estimates changes in individual health and welfare endpoints (specific effects that can be associated with changes in air quality) and assigns values to those changes assuming independence of the individual values. Total benefits are calculated simply as the sum of the values for all non-overlapping health and welfare endpoints. The "damage-function" approach is the standard method for assessing costs and benefits of environmental quality programs and has been used in several recent published analyses (Levy et al., 2009; Hubbell et al., 2009; Tagaris et al., 2009).

To assess economic value in a damage-function framework, the changes in environmental quality must be translated into effects on people or on the things that people value. In some cases, the changes in environmental quality can be directly valued, as is the case for changes in

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

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

The welfare impacts analysis is limited to changes in the environment that have a direct impact on human welfare. For this analysis, we are limited by the available data to examine impacts of changes in visibility in Class 1 areas. We also provide qualitative discussions of the impact of changes in other environmental and ecological effects, for example, changes in deposition of nitrogen and sulfur to terrestrial and aquatic ecosystems, but we are unable to place an economic value on these changes due to time and resource limitations.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure directly either the health outcomes or their values for regulatory analyses. Thus, similar to Kunzli et al. (2000) and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the socio-demographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

5.2.1 Health Impact Assessment

The Health Impact Assessment (HIA) quantifies the changes in the incidence of adverse health impacts resulting from changes in human exposure to PM_{2.5} and ozone air quality. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants (Levy et al. 2009). PC-based tools such as the environmental <u>Benefits Mapping and Analysis Program</u> (BenMAP) can systematize health impact analyses by applying a database of key input parameters, including health impact functions and population projections. Analysts have applied the HIA approach to estimate human health impacts resulting from hypothetical changes in pollutant levels (Hubbell et al. 2005; Davidson et al. 2007, Tagaris et al. 2009). EPA and others

have relied upon this method to predict future changes in health impacts expected to result from the implementation of regulations affecting air quality (U.S. EPA, 2008a).

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

A typical health impact function might look as follows:

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

where y_0 is the baseline incidence rate for the health endpoint being quantified (for example, a health impact function quantifying changes in mortality would use the baseline, or background, mortality rate for the given population of interest); *Pop* is the population affected by the change in air quality; Δx is the change in air quality; and β is the effect coefficient drawn from the epidemiological study. Tools such as BenMAP can systematize the HIA calculation process, allowing users to draw upon a library of existing air quality monitoring data, population data and health impact functions.

Figure 5-1 provides a simplified overview of this approach.