Proposal to Designate an Emission Control Area for Nitrogen Oxides, Sulfur Oxides and Particulate Matter

Technical Support Document

Chapter 4
Quantified Health Impacts Analysis

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4   Quantified Health Impacts Analysis

Ship emissions are responsible for a large number of adverse human health and environmental impacts, especially in densely populated coastal areas. As demonstrated in Chapters 2 and 3, ships that would operate in the proposed ECA generate emissions of NO_X (a precursor to ozone formation and secondarily-formed $PM_{2.5}$), SO_X (a precursor to secondarily-formed $PM_{2.5}$) and directly-emitted $PM_{2.5}$. These pollutants contribute to ambient concentrations of $PM_{2.5}$ and ozone that cause harm to human health and the environment. This chapter presents the U.S.-related health impacts associated with emissions from ships, both in terms of the expected contribution of overall ship emissions to adverse health impacts on land and the reductions in adverse health impacts that can be expected to occur from the adoption of the proposed ECA. Reductions in ambient $PM_{2.5}$ and ozone that will result from the proposed ECA are expected to benefit human health in the form of avoided premature deaths and other serious human health effects, as well as other important public health and environmental effects.

The most conservative premature mortality estimates (Pope et al., 2002 for $PM_{2.5}$ and Bell et al., 2004 for ozone)[1,2] suggest that implementation of the proposed ECA would reduce approximately 3,500 premature mortalities in 2020. The upper end of the premature mortality estimates (Laden et al., 2006 for $PM_{2.5}$ and Levy et al., 2005 for ozone)[3,4] suggest that implementation of the proposed ECA would increase the estimate of avoided premature mortalities to approximately 8,100 in 2020. Thus, even taking the most conservative premature mortality assumptions, the health impacts of the proposed ECA are clearly substantial.

The health impacts modeling presented in this Chapter is based on peer-reviewed studies of air quality and health and welfare effects associated with improvements in air quality. The health impact estimates for the proposed ECA are based on an analytical structure and sequence consistent with health impacts analyses performed by the United States Environmental Protection Agency (US EPA) for its recent analyses in support of the final Ozone National Ambient Air Quality Standard (NAAQS) and the final PM NAAQS as well as all of its recent mobile source emission control programs.[5,6] For a more detailed discussion of the principles of health impacts analysis used here, we refer the reader to those NAAQS documents.

Benefits estimated for this analysis were generated using the Environmental Benefits Mapping and Analysis Program (BenMAP). BenMAP is a computer program developed by the US EPA that integrates a number of modeling elements (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effect incidence estimates. Interested parties may wish to consult the webpage http://www.epa.gov/ttn/ecas/benmodels.html for more information.

The general health impacts analysis framework is as follows:

- Using baseline and control emissions inventories for the emission species expected to affect ambient air quality (NO_X, SO_2, and $PM_{2.5}$; see Chapter 2), we carried out sophisticated photochemical air quality models to estimate baseline and control ambient concentrations of PM and ozone for 2020 (see Chapter 3).

- The estimated changes in ambient concentrations are then combined with monitoring data to estimate population-level potential exposures to changes in ambient concentrations for use in estimating health effects (see Chapter 3). Modeled changes in ambient data are also used to estimate changes in visibility.

- Changes in population exposure to ambient air pollution are used along with impact functions[A] to generate estimated reductions in the incidence of health effects. Because these estimates contain uncertainty, we characterize the health impact estimates probabilistically when appropriate information is available.

Table 4-1 presents the human health impacts we are able to quantify using this methodology. However, the full complement of human health and welfare effects associated with PM and ozone remains unquantified because of current limitations in methods or available data. We have not quantified a number of known or suspected health effects linked with ozone and PM for which appropriate health impact functions are not available or which do not provide easily interpretable outcomes (i.e., changes in heart rate variability). Additionally, we are unable to quantify a number of known environmental (welfare) effects, including reduced acid and particulate deposition damage to cultural monuments and other materials, and environmental benefits due to reductions of impacts of eutrophication in coastal areas. These unquantified welfare effects are also listed in Table 4-1. Both the unquantified and quantified environmental benefits of the proposed ECA are described further in Chapter 5. In sum, the health benefits quantified in this Chapter are likely underestimates of the total benefits attributable to the implementation of the proposed ECA.

[A] The term "impact function" as used here refers to the combination of a) an effect estimate obtained from the epidemiological literature, b) the baseline incidence estimate for the health effect of interest in the modeled population, c) the size of that modeled population, and d) the change in the ambient air pollution metric of interest. These elements are combined in the impact function to generate estimates of changes in incidence of the health effect. The impact function is distinct from the concentration-response (C-R) function, which strictly refers to the estimated equation from the epidemiological study relating incidence of the health effect and ambient pollution. We refer to the specific value of the relative risk or estimated coefficients in the epidemiological study as the "effect estimate." In referencing the functions used to generate changes in incidence of health effects for this analysis, we use the term "impact function" rather than C-R function because "impact function" includes all key input parameters used in the incidence calculation.

Table 4-1 Human Health and Welfare Effects of Pollutants Affected by the Proposed ECA

POLLUTANT/ EFFECT	QUANTIFIED ESTIMATES[A]	UNQUANTIFIED EFFECTS - CHANGES IN:
PM/Health[b]	Premature mortality based on both cohort study estimates [c,d] Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Nonfatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population) Infant mortality	Subchronic bronchitis cases Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Nonasthma respiratory emergency room visits
PM/Welfare		Value of recreational and residential visibility Household soiling
Ozone/Health[e]	Premature mortality: short-term exposures Hospital admissions: respiratory Emergency room visits for asthma Minor restricted-activity days School loss days Asthma attacks Acute respiratory symptoms	Cardiovascular emergency room visits Chronic respiratory damage[f] Premature aging of the lungs[f] Nonasthma respiratory emergency room visits
Ozone/Welfare	Decreased outdoor worker productivity Forest biomass	Yields for commercial crops Yields for commercial forests and noncommercial crops Damage to urban ornamental plants Recreational demand from damaged forest aesthetics Ecosystem functions
Nitrogen Deposition/ Welfare		Commercial forests due to acidic sulfate and nitrate deposition Commercial freshwater fishing due to acidic deposition Recreation in terrestrial ecosystems due to acidic deposition Commercial fishing, agriculture, and forests due to nitrogen deposition Recreation in estuarine ecosystems due to nitrogen deposition Ecosystem functions Passive fertilization
NO$_X$/Health		Lung irritation Lowered resistance to respiratory infection Hospital admissions for respiratory and cardiac diseases

[a] Primary quantified effects are those included in this analysis.

[b] In addition to primary endpoints, there are a number of biological responses that have been associated with PM and ozone 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.

^c 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, 2001 for a discussion of this issue).

^d 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.

^e The public health impact of biological responses such as increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection are likely partially represented by our quantified endpoints.

^f The public health impact of effects such as chronic respiratory damage and premature aging of the lungs may be partially represented by quantified endpoints such as hospital admissions or premature mortality, but a number of other related health impacts, such as doctor visits and decreased athletic performance, remain unquantified.

4.1 Health Impacts Analysis Results for the Proposed ECA

Tables 4.1-1 and 4.1-2 present the annual $PM_{2.5}$ and ozone health impacts for two scenarios. The first scenario assesses the annual health impact of ship emissions if current levels of per-unit emissions are assumed to occur in 2020. The second scenario assesses the annual reduction of ship-related health impacts if the ECA standards are in place in 2020.

Table 4.1-1. Estimated $PM_{2.5}$-Related Health Impacts Associated with Ship Emissions[a]

Health Effect	2020 Annual Ship-Related Incidence ($5^{th}\% - 95^{th}\%$ile)	2020 Annual Reduction in Ship-Related Incidence w/ 200nm ECA ($5^{th}\% - 95^{th}\%$ile)
Premature Mortality[b]		
Adult, age 30+, ACS Cohort Study (Pope et al., 2002)	4,300 (1,700-7,000)	3,400 (1,300 – 5,500)
Adult, age 25+, Six-Cities Study (Laden et al., 2006)	9,800 (5,400-14,000)	7,800 (4,300 – 11,000)
Infant, age <1 year (Woodruff et al., 1997)	16 (0-42)	12 (0 – 33)
Chronic bronchitis (adult, age 26 and over)	4,300 (810-7,800)	3,300 (620 – 6,000)
Non-fatal myocardial infarction (adult, age 18 and over)	8,900 (4,900-13,000)	7,200 (3,900 – 10,000)
Hospital admissions - respiratory (all ages)[c]	990 (490-1,500)	780 (380 – 1,200)
Hospital admissions - cardiovascular (adults, age >18)[d]	2,100 (1,500-2,400)	1,600 (1,200 – 1,900)
Emergency room visits for asthma (age 18 years and younger)	2,500 (1,500-3,500)	1,900 (1,100 – 2,700)
Acute bronchitis, (children, age 8-12)	11,000 (0-22,000)	8,500 (0 – 17,000)
Lower respiratory symptoms (children, age 7-14)	84,000 (40,000-130,000)	66,000 (32,000 – 99,000)
Upper respiratory symptoms (asthmatic children, age 9-18)	62,000 (19,000-100,000)	48,000 (15,000 – 82,000)
Asthma exacerbation (asthmatic children, age 6-18)	79,000 (8,600-220,000)	62,000 (6,700 – 180,000)

Work loss days	580,000 (510,000-650,00)	460,000 (400,000 – 520,000)
Minor restricted activity days (adults age 18-65)	3,400,000 (2,900,000-4,000,000)	2,700,000 (2,300,000 – 3,100,000)

Notes:

[a] Incidence is rounded to two significant digits. Estimates represent incidence within the 48 contiguous United States.

[b] PM-related adult mortality based upon the American Cancer Society (ACS) Cohort Study (Pope et al., 2002) and the Six-Cities Study (Laden et al., 2006). Note that these are two alternative estimates of adult mortality and should not be summed. PM-related infant mortality based upon a study by Woodruff, Grillo, and Schoendorf, (1997).

[c] Respiratory hospital admissions for PM include admissions for chronic obstructive pulmonary disease (COPD), pneumonia and asthma.

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

Table 4.1-2. Estimated Ozone-Related Health Impacts Associated with Ship Emissions[a]

Health Effect	2020 Annual Ship-Related Incidence (5th% - 95th%ile)	2020 Annual Reduction in Ship-Related Incidence w/ 200nm ECA (5th% - 95th%ile)
Premature Mortality, All ages[b] Multi-City Analyses Bell et al (2004) – Non-accidental	370 (160-570)	61 (23 – 98)
Huang et al (2005) – Cardiopulmonary	620 (290-940)	100 (43 – 160)
Schwartz, (2005) – Non-accidental	560 (240-890)	93 (34 – 150)
Meta-analyses: Bell et al (2005) – All cause	1,200 (660-1,700)	200 (100 – 290)
Ito et al (2005) – Non-accidental	1,600 (1,100-2,200)	270 (170 – 370)
Levy et al (2005) – All cause	1,700 (1,200-2,100)	280 (200 – 360)
Hospital admissions- respiratory causes (adult, 65 and older)[c]	2,900 (400-4,800)	470 (46 – 830)
Hospital admissions -respiratory causes (children, under 2)	2,400 (1,200-3,500)	380 (180 – 590)
Emergency room visit for asthma (all ages)	1,300 (0-3,500)	210 (0 – 550)
Minor restricted activity days (adults, age 18-65)	2,300,000 (1,100,000-3,400,000)	360,000 (160,000 – 570,000)
School absence days	810,000 (360,000-1,100,000)	130,000 (51,000 – 190,000)

[a] Incidence is rounded to two significant digits. Estimates represent incidence within the 48 contiguous United States.

[b] Estimates of ozone-related premature mortality are based upon incidence estimates derived from several alternative studies: Bell et al. (2004); Huang et al. (2005); Schwartz (2005) ; Bell et al. (2005); Ito et al. (2005); Levy et al. (2005). The estimates of ozone-related premature mortality should therefore not be summed.

[c] Respiratory hospital admissions for ozone include admissions for all respiratory causes and subcategories for COPD and pneumonia.

As can be seen in Tables 4.1-1 and 4.1-2, ship emissions contribute to large numbers of adverse health impacts within the U.S. By designating an ECA, we estimate that by 2020,

emission reductions will result in major reductions in health impacts, especially those associated with PM exposure. For example, we estimate that in 2020, ships emitting at their current performance would be responsible for approximately 4,300 – 9,800 cases of premature mortality in adults (range based on the health impact function used – Pope et al., 2002 and Laden et al., 2006, respectively). Improving ship emissions to ECA standards will avoid between 3,400 – 7,800 premature deaths in 2020, a reduction of approximately 79%.

We also estimate that ships are responsible for a large number of $PM_{2.5}$-related morbidity impacts. For example, we estimate that in 2020, ships emitting at their current performance would be responsible for approximately 4,300 cases of chronic bronchitis, 8,900 non-fatal heart attacks, 5,600 hospital admissions and emergency room visits, 580,000 days of work lost, and 3,400,000 days of restricted physical activity. Improving ship emissions to ECA standards will result in the avoidance of 3,300 cases of chronic bronchitis, 7,200 non-fatal heart attacks, 4,400 hospital admissions and emergency room visits, 460,000 days of work lost, and 2,700,000 days of restricted physical activity. Again, improving to ECA standards will reduce the incidence of $PM_{2.5}$-related non-fatal health impacts associated with ships by approximately 78%.

Similarly, ship emissions contribute to adverse health impacts associated with ozone exposure. For example, we estimate that in 2020, ships emitting at their current performance would be responsible for approximately 370 – 1,700 cases of premature mortality, depending on the health impact function, 6,600 hospital admissions and emergency room visits, 810,000 days of school absence, and 2,300,000 day of restricted physical activity. Improving to ECA standards will avoid between 61 – 280 premature deaths in 2020. Furthermore, it will result in the avoidance of 1,100 hospital admissions and emergency room visits, 130,000 days of school absence, and 360,000 days of restricted physical activity.

It is clear that the avoided health impacts associated with the proposed ECA are substantial. Implementation of a North American ECA would significantly improve human health, both in terms of reduced premature mortality and avoided morbidity effects.

4.2 Methodology

4.2.1 Human Health Impact Functions

Health impact functions measure the change in a health endpoint of interest, such as hospital admissions, for a given change in ambient ozone or PM concentration. Health impact functions are derived from primary epidemiology studies, meta-analyses of multiple epidemiology studies, or expert elicitations. A standard health impact function has four components: 1) an effect estimate from a particular study; 2) a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics such as the Centers for Disease Control); 3) the size of the potentially affected population; and 4) the estimated change in the relevant ozone or PM summary measures.

A typical health impact function might look like:

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

where y_0 is the baseline incidence (the product of the baseline incidence rate times the potentially affected population), β is the effect estimate, and Δx is the estimated change in the summary pollutant measure. There are other functional forms, but the basic elements remain the same. The following subsections describe the sources for each of the first three elements: size of the potentially affected populations; $PM_{2.5}$ and ozone effect estimates; and baseline incidence rates. Section 4.2.2 describes the ozone and PM air quality inputs to the health impact functions.

4.2.1.1 Potentially Affected Populations

The starting point for estimating the size of potentially affected populations is the 2000 U.S. Census block level dataset.[7] Benefits Modeling and Analysis Program (BenMAP) incorporates 250 age/gender/race categories to match specific populations potentially affected by ozone and other air pollutants. The software constructs specific populations matching the populations in each epidemiological study by accessing the appropriate age-specific populations from the overall population database. BenMAP projects populations to 2020 using growth factors based on economic projections.[8]

4.2.1.2 Effect Estimate Sources

The most significant quantifiable benefits of reducing ambient concentrations of ozone and PM are attributable to reductions in human health risks. EPA's Ozone and PM Criteria Documents[9,10] and the World Health Organization's 2003 and 2004[11,12] reports outline numerous human health effects known or suspected to be linked to exposure to ambient ozone and PM. US EPA recently evaluated the ozone and PM literature for use in the benefits analysis for the final 2008 Ozone NAAQS and final 2006 PM NAAQS analyses. We use the same literature in this analysis.

It is important to note that we are unable to separately quantify all of the possible PM and ozone health effects that have been reported in the literature for three reasons: (1) the possibility of double counting (such as hospital admissions for specific respiratory diseases versus hospital admissions for all or a sub-set of respiratory diseases); (2) uncertainties in applying effect relationships that are based on clinical studies to the potentially affected population; or (3) the lack of an established concentration-response (CR) relationship. Table 4-1 lists the possible human health and welfare effects of pollutants affected by the proposed ECA. Table 4.2-1 lists the health endpoints included in this analysis.

Table 4.2-1 Ozone- and PM-Related Health Endpoints

ENDPOINT	*POLLUTANT*	*STUDY*	*STUDY POPULATION*
Premature Mortality			
Premature mortality – daily time series	O3	Bell et al (2004) (NMMAPS study)[13] – Non-accidental Huang et al (2005)[14] - Cardiopulmonary Schwartz (2005)[15] – Non-accidental <u>Meta-analyses:</u> Bell et al (2005)[16] – All cause Ito et al (2005)[17] – Non-accidental	All ages

ENDPOINT	*POLLUTANT*	*STUDY*	*STUDY POPULATION*
		Levy et al (2005)[18] – All cause	
Premature mortality —cohort study, all-cause	PM$_{2.5}$	Pope et al. (2002)[19] Laden et al. (2006)[20]	>29 years >25 years
Premature mortality — all-cause	PM$_{2.5}$	Woodruff et al. (1997)[21]	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	PM$_{2.5}$	Abbey et al. (1995)[22]	>26 years
Nonfatal heart attacks	PM$_{2.5}$	Peters et al. (2001)[23]	Adults (>18 years)
Hospital Admissions			
Respiratory	O3	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp)[24] Schwartz (1994a; 1994b) - ICD 480-486 (pneumonia)[25,26] Moolgavkar et al. (1997) - ICD 480-487 (pneumonia)[27] Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al. (1997) – ICD 490-496 (COPD)	>64 years
		Burnett et al. (2001)[28]	<2 years
	PM$_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 490-496 (COPD)[29] Ito (2003)—ICD 490-496 (COPD)[30]	>64 years
	PM$_{2.5}$	Moolgavkar (2000)—ICD 490-496 (COPD)[31]	20–64 years
	PM$_{2.5}$	Ito (2003)—ICD 480-486 (pneumonia)	>64 years
	PM$_{2.5}$	Sheppard (2003)—ICD 493 (asthma)[32]	<65 years

ENDPOINT	POLLUTANT	STUDY	STUDY POPULATION
Cardiovascular	PM$_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 390-429 (all cardiovascular) Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	PM$_{2.5}$	Moolgavkar (2000)—ICD 390-429 (all cardiovascular)	20–64 years
Asthma-related ER visits	O3	Pooled estimate: Jaffe et al (2003)[33] Peel et al (2005)[34] Wilson et al (2005)[35]	5–34 years All ages All ages
Asthma-related ER visits (con't)	PM$_{2.5}$	Norris et al. (1999)[36]	0–18 years
Other Health Endpoints			
Acute bronchitis	PM$_{2.5}$	Dockery et al. (1996)[37]	8–12 years
Upper respiratory symptoms	PM$_{2.5}$	Pope et al. (1991)[38]	Asthmatics, 9–11 years
Lower respiratory symptoms	PM$_{2.5}$	Schwartz and Neas (2000)[39]	7–14 years
Asthma exacerbations	PM$_{2.5}$	Pooled estimate: Ostro et al. (2001)[40] (cough, wheeze and shortness of breath) Vedal et al. (1998)[41] (cough)	6–18 years[a]
Work loss days	PM$_{2.5}$	Ostro (1987)[42]	18–65 years
School absence days	O3	Pooled estimate: Gilliland et al. (2001)[43] Chen et al. (2000)[44]	5–17 years[b]
Minor Restricted Activity Days (MRADs)	O3	Ostro and Rothschild (1989)[45]	18–65 years
	PM$_{2.5}$	Ostro and Rothschild (1989)	18–65 years

[a] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the Science Advisory Board Health Effects Subcommittee (SAB-HES), we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. Science Advisory Board. 2004. Advisory Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis –Benefits and Costs of the Clean Air Act, 1990—2020. EPA-SAB-COUNCIL-ADV-04-004. See also National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.

[b] Gilliland et al. (2001) studied children aged 9 and 10. Chen et al. (2000) studied children 6 to 11. Based on recent advice from the National Research Council and the EPA SAB-HES, we have calculated reductions in school absences for all school-aged children based on the biological similarity between children aged 5 to 17.

In selecting epidemiological studies as sources of effect estimates, we applied several criteria to develop a set of studies that is likely to provide the best estimates of impacts in the U.S. To account for the potential impacts of different health care systems or underlying health status of populations, we give preference to U.S. studies over non-U.S. studies. In addition, due to the potential for confounding by co-pollutants, we give preference to effect estimates from models including both ozone and PM over effect estimates from single-

pollutant models.[46,47]

4.2.1.2.1 PM$_{2.5}$-Related Health Impact Functions

PM$_{2.5}$-Related Adult Premature Mortality

Both long- and short-term exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. The size of the mortality risk estimates from epidemiological studies, the serious nature of the effect itself, and the high monetary value ascribed to prolonging life make mortality risk reduction the most significant health endpoint quantified in this analysis.

Although a number of uncertainties remain to be addressed by continued research (NRC, 1998),[48] a substantial body of published scientific literature documents the correlation between elevated PM concentrations and increased mortality rates (US EPA, 2004).[49] Time-series methods have been used to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Cohort methods have been used to examine the potential relationship between community-level PM exposures over multiple years (i.e., long-term exposures) and community-level annual mortality rates. Researchers have found statistically significant associations between PM and premature mortality using both types of studies. In general, the risk estimates based on the cohort studies are larger than those derived from time-series studies. Cohort analyses are thought to better capture the full public health impact of exposure to air pollution over time, because they capture the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002).[50,51] This section discusses some of the issues surrounding the estimation of premature mortality.

Over a dozen studies have found significant associations between various measures of long-term exposure to PM and elevated rates of annual mortality, beginning with Lave and Seskin (1977).[52] Most of the published studies found positive (but not always statistically significant) associations with available PM indices such as total suspended particles (TSP). However, exploration of alternative model specifications sometimes raised questions about causal relationships (e.g., Lipfert, Morris, and Wyzga [1989]).[53] These early "ecological cross-sectional" studies (e.g., Lave and Seskin [1977]; Ozkaynak and Thurston [1987][54]) were criticized for a number of methodological limitations, particularly for inadequate control at the individual level for variables that are potentially important in causing mortality, such as wealth, smoking, and diet. Over the last 10 years, several studies using "prospective cohort" designs have been published that appear to be consistent with the earlier body of literature. These new "prospective cohort" studies reflect a significant improvement over the earlier work because they include individual-level information with respect to health status and residence. The most extensive analyses have been based on data from two prospective cohort groups, often referred to as the Harvard "Six-Cities Study" (Dockery et al., 1993;[55] Laden et al, 2006) and the "American Cancer Society or ACS study" (Pope et al., 1995;[56] Pope et al, 2002; Pope et al, 2004[57]); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the United States. A third major data set comes from the California-based 7th Day Adventist Study (e.g., Abbey et al., 1999),[58] which reported associations between long-term PM exposure and mortality in

men. Results from this cohort, however, have been inconsistent, and the air quality results are not geographically representative of most of the United States, and the lifestyle of the population is not reflective of much of the U.S. population. Analysis is also available for a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000; Lipfert et al, 2003, 2006).[59,60,61] The characteristics of this group differ from the cohorts in the Six-Cities, ACS, and 7th Day Adventist studies with respect to income, race, health status, and smoking status. Unlike previous long-term analyses, this study found some associations between mortality and ozone but found inconsistent results for PM indicators. Because of the selective nature of the population in the veteran's cohort, we have chosen not to include any effect estimates from the Lipfert et al. (2000) study in our benefits assessment.[B]

Given their consistent results and broad geographic coverage, and importance in informing the NAAQS development process, the Six-Cities and ACS data have been particularly important in benefits analyses. The credibility of these two studies is further enhanced by the fact that the initial published studies (Pope et al, 1995 and Dockery et al 1993) were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by the Health Effects Institute (HEI) (Krewski et al., 2000).[62] The final results of the reanalysis were then independently peer reviewed by a Special Panel of the HEI Health Review Committee. The results of these reanalyses confirmed and expanded those of the original investigators. While the HEI reexamination lends credibility to the original studies, it also highlights sensitivities concerning the relative impact of various pollutants, such as SO_2, the potential role of education in mediating the association between pollution and mortality, and the influence of spatial correlation modeling.

Further confirmation and extension of the findings of the 1993 Six City Study and the 1995 ACS study were recently completed using more recent air quality and a longer follow-up period for the ACS cohort was recently published (Pope et al, 2002, 2004; Laden et al, 2006). The follow up to the Harvard Six City Study both confirmed the effect size from the first analysis and provided additional confirmation that reductions in $PM_{2.5}$ are likely to result in reductions in the risk of premature death. This additional evidence stems from the observed reductions in $PM_{2.5}$ in each city during the extended follow-up period. Laden et al. (2006) found that mortality rates consistently went down at a rate proportionate to the observed reductions in $PM_{2.5}$.

[B] US EPA recognizes that the ACS cohort also is not representative of the demographic mix in the general population. The ACS cohort is almost entirely white and has higher income and education levels relative to the general population. US EPA's approach to this problem is to match populations based on the potential for demographic characteristics to modify the effect of air pollution on mortality risk. Thus, for the various ACS-based models, we are careful to apply the effect estimate only to ages matching those in the original studies, because age has a potentially large modifying impact on the effect estimate, especially when younger individuals are excluded from the study population. For the Lipfert analysis, the applied population should be limited to that matching the sample used in the analysis. This sample was all male, veterans, and diagnosed hypertensive. There are also a number of differences between the composition of the sample and the general population, including a higher percentage of African Americans (35%) and a much higher percentage of smokers (81% former smokers, 57% current smokers) than the general population (12% African American, 24% current smokers).

The extended analyses of the ACS cohort data (Pope et al., 2002, 2004) provides additional refinements to the analysis of PM-related mortality by a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; b) substantially increasing exposure data, including additional measurement of cohort exposure to $PM_{2.5}$ following implementation of the $PM_{2.5}$ standard in 1999; c) controlling for a variety of personal risk factors including occupational exposure and diet; and d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates including the possibility of spatial autocorrelation of survival times in communities located near each other.

For this analysis, we use the ACS study because it includes a large sample size and longer exposure interval and covers more locations (e.g., 50 cities compared to the Six-Cities Study) than other studies of its kind. The relative risks derived from the ACS study are based on the average exposure to $PM_{2.5}$, measured by the average of two $PM_{2.5}$ measurements, over the periods 1979–1983 and 1999–2000. In addition to relative risks for all-cause mortality, the ACS study provides relative risks for cardiopulmonary, lung cancer, and all-other cause mortality. Because of concerns regarding the statistical reliability of the "all-other" cause mortality relative risk estimates, we calculate mortality impacts for this analysis using the all-cause relative risk.

We also include a separate estimate based on the Six-cities study to complement the estimate based on the ACS study. We use this specific estimate because it reflects the most up-to-date science and reflects the weight that experts have placed on both the ACS and Harvard Six-city studies (see the results of the PM mortality expert elicitation).[63]

Because of the differences in the study designs and populations considered in the ACS and Harvard Six-cities studies, we do not pool the results of the studies and instead present a range of estimates reflecting the two sources of impact estimates.

A number of additional analyses have been conducted on the ACS cohort data (Jerrett et al., 2005;[64] Krewski et al., 2005;[65] Pope et al., 2004). These studies have continued to find a strong significant relationship between $PM_{2.5}$ and mortality outcomes. Specifically, much of the recent research has suggested a stronger relationship between cardiovascular mortality and lung cancer mortality with $PM_{2.5}$, and a less significant relationship between respiratory-related mortality and $PM_{2.5}$.

$PM_{2.5}$-Related Infant Mortality

Recently published studies have strengthened the case for an association between PM exposure and respiratory inflammation and infection leading to premature mortality in children under 5 years of age. Specifically, the release of the WHO Global Burden of Disease Study focusing on ambient air cites several recently published time-series studies relating daily PM exposure to mortality in children   The study by Belanger et al. (2003)[66] also corroborates findings linking PM exposure to increased respiratory inflammation and infections in children. A study by Chay and Greenstone (2003)[67] found that reductions in TSP caused by the recession of 1981–1982 were related to reductions in infant mortality at the county level. With regard to the cohort study conducted by Woodruff et al. (1997),[68] we note

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 infants (e.g., maternal educational level, maternal ethnicity, parental marital status, and maternal smoking status). Based on these findings, the US EPA estimates infant mortality using an impact function developed from the Woodruff et al. (1997) study.

Chronic Bronchitis

Chronic bronchitis (CB) is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5% of the U.S. population (American Lung Association, 1999).[69] A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993)[70] and Abbey et al. (1995)[71] provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Because the proposed ECA is expected to reduce primarily $PM_{2.5}$, this analysis uses only the Abbey et al. (1995) study, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB.

Nonfatal Myocardial Infarctions (heart attacks)

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001)[72] and other countries (Poloniecki et al., 1997).[73] We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. A more recent study by Zanobetti and Schwartz (2005)[74] used a similar method to Peters et al. (2001), but focused on adults 65 and older, and used PM_{10} as the PM indicator. They found a significant relationship between nonfatal heart attacks and PM_{10}, although the magnitude of the effect was much lower than Peters et al. This may reflect the use of PM_{10}, the more limited age range, or the less precise diagnosis of heart attack used in defining the outcome measure. Other studies, such as Domenici et al. (2006),[75] Samet et al. (2000),[76] and Moolgavkar (2000),[77] show a consistent relationship between all cardiovascular hospital admissions, including those for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on long-term health costs and earnings, we provide a separate estimate for nonfatal heart attacks. The estimate used in the analysis of the proposed ECA is based on the single available U.S. $PM_{2.5}$ effect estimate from Peters et al. (2001). The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001)[78,79,80] 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).[81,82,83,84] As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

Hospital and Emergency Room Admissions

Because of the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and

air pollution. Because of this, many of the hospital admission endpoints use pooled impact functions based on the results of a number of studies. In addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Since most emergency room visits do not result in an admission to the hospital (the majority of people going to the emergency room is treated and return home), we treat hospital admissions and emergency room visits separately, taking account of the fraction of emergency room visits that are admitted to the hospital.

The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking PM with other types of hospital admissions. The only type of emergency room visits that have been consistently linked to PM in the United States are asthma-related visits.

To estimate avoided incidences of $PM_{2.5}$ related cardiovascular hospital admissions in populations aged 65 and older, we use effect estimates from studies by Moolgavkar (2003)[85] and Ito (2003).[86] However, only Moolgavkar (2000)[87] provided a separate effect estimate for populations 20 to 64.[C] Total cardiovascular hospital admissions are thus the sum of the pooled estimates from Moolgavkar (2003) and Ito (2003) for populations over 65 and the Moolgavkar (2000) based impacts for populations aged 20 to 64. Cardiovascular hospital admissions include admissions for myocardial infarctions. To avoid double-counting benefits from reductions in myocardial infarctions when applying the impact function for cardiovascular hospital admissions, we first adjusted the baseline cardiovascular hospital admissions to remove admissions for myocardial infarctions.

To estimate total avoided incidences of respiratory hospital admissions, we used impact functions for several respiratory causes, including chronic obstructive pulmonary disease (COPD), pneumonia, and asthma. As with cardiovascular admissions, additional published studies show a statistically significant relationship between PM_{10} and respiratory hospital admissions. We used only those focusing on $PM_{2.5}$. Both Moolgavkar (2000) and Ito (2003) provide effect estimates for COPD in populations over 65, allowing us to pool the impact functions for this group. Only Moolgavkar (2000) provides a separate effect estimate for populations 20 to 64. Total COPD hospital admissions are thus the sum of the pooled estimate for populations over 65 and the single study estimate for populations 20 to 64. Only Ito (2003) estimated pneumonia and only for the population 65 and older. In addition, Sheppard (2003) provided an effect estimate for asthma hospital admissions for populations under age 65. Total avoided incidence of PM-related respiratory-related hospital admissions is the sum of COPD, pneumonia, and asthma admissions.

[C] Note that the Moolgavkar (2000) study has not been updated to reflect the more stringent GAM convergence criteria. However, given that no other estimates are available for this age group, we chose to use the existing study. Updates have been provided for the 65 and older population, and showed little difference. Given the very small (<5%) difference in the effect estimates for people 65 and older with cardiovascular hospital admissions between the original and reanalyzed results, we do not expect the difference in the effect estimates for the 20 to 64 population to differ significantly. As such, the choice to use the earlier, uncorrected analysis will likely not introduce much bias.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the effect estimate from a study of children 18 and under by Norris et al. (1999).[88] As noted earlier, there is another study by Schwartz examining a broader age group (less than 65), but the Schwartz study focused on PM_{10} rather than $PM_{2.5}$. We selected the Norris et al. (1999) effect estimate because it better matched the pollutant of interest. Because children tend to have higher rates of hospitalization for asthma relative to adults under 65, we will likely capture the majority of the impact of $PM_{2.5}$ on asthma emergency room visits in populations under 65, although there may still be significant impacts in the adult population under 65.

Acute Health Events and Work Loss Days

As indicated in Table 4.2-1, in addition to mortality, chronic illness, and hospital admissions, a number of acute health effects not requiring hospitalization are associated with exposure to ambient levels of PM. The sources for the effect estimates used to quantify these effects are described below.

Around four percent of U.S. children between the ages of 5 and 17 experience episodes of acute bronchitis annually (American Lung Association, 2002).[89] Acute bronchitis is characterized by coughing, chest discomfort, slight fever, and extreme tiredness, lasting for a number of days. According to the MedlinePlus medical encyclopedia,[D] with the exception of cough, most acute bronchitis symptoms abate within 7 to 10 days. Incidence of episodes of acute bronchitis in children between the ages of 5 and 17 were estimated using an effect estimate developed from Dockery et al. (1996).[90]

Incidences of lower respiratory symptoms (e.g., wheezing, deep cough) in children aged 7 to 14 were estimated using an effect estimate from Schwartz and Neas (2000).[91]

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (e.g., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms has thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged 9 to 11 are estimated using an effect estimate developed from Pope et al. (1991).[92]

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Days of work lost due to $PM_{2.5}$ were estimated using an effect estimate developed from Ostro (1987).[93]

Minor restricted activity days (MRADs) result when individuals reduce most usual daily activities and replace them with less strenuous activities or rest, yet not to the point of missing work or school. For example, a mechanic who would usually be doing physical work

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[D] See http://www.nlm.nih.gov/medlineplus/ency/article/000124.htm, accessed January 2002.

most of the day will instead spend the day at a desk doing paper and phone work because of difficulty breathing or chest pain. The effect of $PM_{2.5}$ and ozone on MRAD was estimated using an effect estimate derived from Ostro and Rothschild (1989).[94]

In analyzing the proposed ECA, we focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation to avoid double counting.[E] Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, we would likely double-count incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

To characterize asthma exacerbations in children, we selected two studies (Ostro et al., 2001; Vedal et al., 1998)[95,96] that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between $PM_{2.5}$, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates (see below). Vedal et al. (1998) followed a group of elementary school children, including 74 asthmatics, located on the west coast of Vancouver Island for 18 months including measurements of daily peak expiratory flow (PEF) and the tracking of respiratory symptoms (e.g., cough, phlegm, wheeze, chest tightness) through the use of daily diaries. Association between PM_{10} and respiratory symptoms for the asthmatic population was only reported for two endpoints: cough and PEF. Because it is difficult to translate PEF measures into clearly defined health endpoints that can be monetized, we only included the cough-related effect estimate from this study in quantifying asthma exacerbations. We employed the following pooling approach in combining estimates generated using effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled the separate incidence estimates for shortness of breath, wheeze, and cough generated using effect estimates from the Ostro et al. study, because each of these endpoints is aimed at capturing the same overall endpoint (asthma exacerbations) and there could be overlap in their predictions. The pooled estimate from the Ostro et al. study is then pooled

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[E] Estimating asthma exacerbations associated with air pollution exposures is difficult, due to concerns about double-counting of benefits. Concerns over double-counting stem from the fact that studies of the general population also include asthmatics, so estimates based solely on the asthmatic population cannot be directly added to the general population numbers without double-counting. In one specific case (upper respiratory symptoms in children), the only study available is limited to asthmatic children, so this endpoint can be readily included in the calculation of total benefits. However, other endpoints, such as lower respiratory symptoms and MRADs, are estimated for the total population that includes asthmatics. Therefore, to simply add predictions of asthma-related symptoms generated for the population of asthmatics to these total population-based estimates could result in double-counting, especially if they evaluate similar endpoints.

with the cough-related estimate generated using the Vedal study. The rationale for this second pooling step is similar to the first; both studies are attempting to quantify the same overall endpoint (asthma exacerbations).

Additional epidemiological studies are available for characterizing asthma-related health endpoints (the full list of epidemiological studies considered for modeling asthma-related incidence is presented in Table 4.2-2). However, we do not use these additional studies in this analysis. In particular, the Yu et al. (2000)[97] estimates show a much higher baseline incidence rate than other studies, which may lead to an overstatement of the expected impacts in the overall asthmatic population. The Whittemore and Korn (1980)[98] study did not use a well-defined endpoint, instead focusing on a respondent-defined "asthma attack." Other studies looked at respiratory symptoms in asthmatics but did not focus on specific exacerbations of asthma.

Treatment of Potential Thresholds in PM$_{2.5}$-Related Health Impact Functions

Unless specifically noted, our premature mortality benefits estimates are based on an assumed cutpoint in the premature mortality concentration-response function at 10 µg/m^3, and an assumed cutpoint of 10 µg/m^3 for the concentration-response functions for morbidity associated with short term exposure to PM$_{2.5}$. The 10 µg/m^3 threshold reflects comments from the U.S. EPA's Science Advisory Board Clean Air Science Advisory Committee (CASAC) (U.S. EPA Science Advisory Board, 2005).[99]

Table 4.2-2. Studies Examining Health Impacts in the Asthmatic Population Evaluated for Use in the Health Impacts Analysis

ENDPOINT	DEFINITION	POLLUTANT	STUDY	STUDY POPULATION
Asthma Attack Indicators				
Shortness of breath	Prevalence of shortness of breath; incidence of shortness of breath	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Cough	Prevalence of cough; incidence of cough	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Wheeze	Prevalence of wheeze; incidence of wheeze	$PM_{2.5}$	Ostro et al. (2001)	African-American asthmatics, 8–13
Asthma exacerbation	>= 1 mild asthma symptom: wheeze, cough, chest tightness, shortness of breath	PM_{10}, $PM_{1.0}$	Yu et al. (2000)	Asthmatics, 5–13
Cough	Prevalence of cough	PM_{10}	Vedal et al. (1998)	Asthmatics, 6–13
Other Symptoms/Illness Endpoints				
Upper respiratory symptoms	>= 1 of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes	PM_{10}	Pope et al. (1991)	Asthmatics, 9–11
Moderate or worse asthma	Probability of moderate (or worse) rating of overall asthma status	$PM_{2.5}$	Ostro et al. (1991)	Asthmatics, all ages
Acute bronchitis	>= 1 episodes of bronchitis in the past 12 months	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9–15
Phlegm	"Other than with colds, does this child usually seem congested in the chest or bring up phlegm?"	$PM_{2.5}$	McConnell et al. (1999)	Asthmatics, 9–15
Asthma attacks	Respondent-defined asthma attack	$PM_{2.5}$	Whittemore and Korn (1980)	Asthmatics, all ages

4.2.1.2.2 Ozone-Related Health Impact Functions

Ozone-Related Premature Mortality

While particulate matter is the criteria pollutant most clearly associated with premature mortality, research suggests that short-term repeated ozone exposure likely contributes to premature death. In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC),[100] a panel of experts and reviewers concluded that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure. The report also recommended that little or no weight be given to the assumption that there is no causal association between ozone exposure and premature mortality.

We estimate the change in mortality incidence and estimated credible interval[F] resulting from application of the effect estimate from the following studies: the Bell et al. (2004) NMMAPS analysis, Huang et al. (2004), Schwartz (2004), and effect estimates from the three meta-analyses - Bell et al. (2005), Ito et al. (2005), and Levy et al. (2005). The results from each study are presented separately to reflect differences in the study designs and assumptions about causality. However, it is important to note that this procedure only captures the uncertainty in the underlying epidemiological work, and does not capture other sources of uncertainty, such as uncertainty in the estimation of changes in air pollution exposure.

Respiratory Hospital Admissions Effect Estimates

Detailed hospital admission and discharge records provide data for an extensive body of literature examining the relationship between hospital admissions and air pollution. This is especially true for the portion of the population aged 65 and older, because of the availability of detailed Medicare records. In addition, there is one study (Burnett et al., 2001)[101] providing an effect estimate for respiratory hospital admissions in children under two.

Because the number of hospital admission studies we considered is so large, we used results from a number of studies to pool some hospital admission endpoints. Pooling is the process by which multiple study results may be combined in order to produce better estimates of the effect estimate, or β. For a complete discussion of the pooling process, see the BenMAP manual for technical details.[G] To estimate total respiratory hospital admissions associated with changes in ambient ozone concentrations for adults over 65, we first estimated the change in hospital admissions for each of the different effects categories that each study provided for each city. These cities included Minneapolis, Detroit, Tacoma and New Haven. To estimate total respiratory hospital admissions for Detroit, we added the pneumonia and COPD estimates, based on the effect estimates in the Schwartz study (1994).[102] Similarly, we summed the estimated hospital admissions based on the effect estimates the Moolgavkar study reported for Minneapolis (Moolgavkar et al., 1997).[103] To estimate total respiratory hospital admissions for Minneapolis using the Schwartz study (1994),[104] we simply estimated pneumonia hospital admissions based on the effect estimate. Making this assumption that pneumonia admissions represent the total impact of ozone on hospital admissions in this city will give some weight to the possibility that there is no relationship between ozone and COPD, reflecting the equivocal evidence represented by the different studies. We then used a fixed-effects pooling procedure to combine the two total respiratory hospital admission estimates for Minneapolis. Finally, we used random effects pooling to combine the results for Minneapolis and Detroit with results from studies in Tacoma and New Haven from Schwartz (1995).[105] As noted above, this pooling approach incorporates both the precision of the individual effect estimates and between-study variability characterizing differences across study locations.

[F] A credible interval is a posterior probability interval used in Bayesian statistics, which is similar to a confidence interval used in frequentist statistics.

[G] BenMAP and its supporting manual are available for download at http://www.epa.gov/air/benmap. Accessed January 9, 2009.

Asthma-Related Emergency Room Visits Effect Estimates

We used three studies as the source of the concentration-response functions we used to estimate the effects of ozone exposure on asthma-related emergency room (ER) visits: Peel et al. (2005);[106] Wilson et al. (2005);[107] and Jaffe et al. (2003).[108] We estimated the change in ER visits using the effect estimate(s) from each study and then pooled the results using the random effects pooling technique (see the BenMAP manual for technical details). The study by Jaffe et al. (2003) examined the relationship between ER visits and air pollution for populations aged five to 34 in the Ohio cities of Cleveland, Columbus and Cincinnati from 1991 through 1996. In single-pollutant Poisson regression models, ozone was linked to asthma visits. We use the pooled estimate across all three cities as reported in the study. The Peel et al. study (2005) estimated asthma-related ER visits for all ages in Atlanta, using air quality data from 1993 to 2000. Using Poisson generalized estimating equations, the authors found a marginal association between the maximum daily 8-hour average ozone level and ER visits for asthma over a 3-day moving average (lags of 0, 1, and 2 days) in a single pollutant model. Wilson et al. (2005) examined the relationship between ER visits for respiratory illnesses and asthma and air pollution for all people residing in Portland, Maine from 1998-2000 and Manchester, New Hampshire from 1996-2000. For all models used in the analysis, the authors restricted the ozone data incorporated into the model to the months ozone levels are usually measured, the spring-summer months (April through September). Using the generalized additive model, Wilson et al. (2005) found a significant association between the maximum daily 8-hour average ozone level and ER visits for asthma in Portland, but found no significant association for Manchester. Similar to the approach used to generate effect estimates for hospital admissions, we used random effects pooling to combine the results across the individual study estimates for ER visits for asthma. The Peel et al. (2005) and Wilson et al. (2005) Manchester estimates were not significant at the 95 percent level, and thus, the confidence interval for the pooled incidence estimate based on these studies includes negative values. This is an artifact of the statistical power of the studies, and the negative values in the tails of the estimated effect distributions do not represent improvements in health as ozone concentrations are increased. Instead these should be viewed as a measure of uncertainty due to limitations in the statistical power of the study. Note that we included both hospital admissions and ER visits as separate endpoints associated with ozone exposure, because our estimates of hospital admission costs do not include the costs of ER visits, and because most asthma ER visits do not result in a hospital admission.

Minor Restricted Activity Days Effects Estimate

Minor restricted activity days (MRADs) occur when individuals reduce most usual daily activities and replace them with less-strenuous activities or rest, but do not miss work or school. We estimated the effect of ozone exposure on MRADs using a concentration-response function derived from Ostro and Rothschild (1989).[109] These researchers estimated the impact of ozone and $PM_{2.5}$ on MRAD incidence in a national sample of the adult working population (ages 18 to 65) living in metropolitan areas. We developed separate coefficients for each year of the Ostro and Rothschild analysis (1976-1981), which we then combined for use in EPA's analysis. The effect estimate used in the impact function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4), using the inverse of the variance as the weight.

School Absences Effect Estimate

Children may be absent from school due to respiratory or other acute diseases caused, or aggravated by, exposure to air pollution. Several studies have found a significant association between ozone levels and school absence rates. We use two studies (Gilliland et al., 2001; Chen et al., 2000)[110,111] to estimate changes in school absences resulting from changes in ozone levels. The Gilliland et al. study estimated the incidence of new periods of absence, while the Chen et al. study examined daily absence rates. We converted the Gilliland et al. estimate to days of absence by multiplying the absence periods by the average duration of an absence. We estimated 1.6 days as the average duration of a school absence, the result of dividing the average daily school absence rate from Chen et al. (2000) and Ransom and Pope (1992) by the episodic absence duration from Gilliland et al. (2001). Thus, each Gilliland et al. period of absence is converted into 1.6 absence days.

Following recent advice from the National Research Council (2002),[112] we calculated reductions in school absences for the full population of school age children, ages five to 17. This is consistent with recent peer-reviewed literature on estimating the impact of ozone exposure on school absences (Hall et al. 2003).[113] We estimated the change in school absences using both Chen et al. (2000) and Gilliland et al. (2001) and then, similar to hospital admissions and ER visits, pooled the results using the random effects pooling procedure.

4.2.1.3 Baseline PM Health Effect Incidence Rates

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical result might be that a 10 µg/m^3 decrease in daily PM$_{2.5}$ levels might decrease hospital admissions by 3 percent. To then convert this relative change into a number of cases, the baseline incidence of the health effect is necessary. The baseline incidence rate provides an estimate of the incidence rate (number of cases of the health effect per year, usually per 10,000 or 100,000 general population) in the assessment location corresponding to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number (e.g., if the baseline incidence rate is number of cases per year per 100,000 population, it must be multiplied by the number of 100,000s in the population).

Some epidemiological studies examine the association between pollution levels and adverse health effects in a specific subpopulation, such as asthmatics or diabetics. In these cases, it is necessary to develop not only baseline incidence rates, but also prevalence rates for the defining condition (e.g., asthma). For both baseline incidence and prevalence data, we use age-specific rates where available. Impact functions are applied to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits.

In most cases, because of a lack of data or methods, we have not attempted to project incidence rates to future years, instead assuming that the most recent data on incidence rates is the best prediction of future incidence rates. In recent years, better data on trends in incidence and prevalence rates for some endpoints, such as asthma, have become available. We are

working to develop methods to use these data to project future incidence rates. However, for our primary benefits analysis, we continue to use current incidence rates. The one exception is in the case of premature mortality. In this case, we have projected mortality rates such that future mortality rates are consistent with our projections of population growth. Compared with previous analyses, this will result in a reduction in the mortality related impacts of air pollution in future years.

Table 4.2-3 summarizes the baseline incidence data and sources used in the benefits analysis. We use the most geographically disaggregated data available. For premature mortality, county-level data are available. For hospital admissions, regional rates are available. However, for all other endpoints, a single national incidence rate is used, due to a lack of more spatially disaggregated data. In these cases, 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.

Table 4.2-3: Baseline Incidence Rates and Population Prevalence Rates for Use in Impact Functions, General Population

ENDPOINT	PARAMETER	RATES	
		Value	Source[a]
Mortality	Daily or annual mortality rate	Age-, cause-, and county-specific rate	CDC Wonder (1996–1998)
Hospitalizations	Daily hospitalization rate	Age-, region-, and cause-specific rate	1999 NHDS public use data files[b]
Asthma ER Visits	Daily asthma ER visit rate	Age- and region-specific visit rate	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]
Chronic Bronchitis	Annual prevalence rate per person - Aged 18–44 - Aged 45–64 - Aged 65 and older	 0.0367 0.0505 0.0587	1999 NHIS (American Lung Association, 2002, Table 4)
	Annual incidence rate per person	0.00378	Abbey et al. (1993, Table 3)
Nonfatal Myocardial Infarction (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[b]; adjusted by 0.93 for probability 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		Vedal et al. (1998)

	- daily wheeze - daily cough - daily dyspnea	0.038 0.086 0.045	
Acute Bronchitis	Annual bronchitis incidence rate, children	0.043	American Lung Association (2002, Table 11)
Lower Respiratory Symptoms	Daily lower respiratory symptom incidence among children[d]	0.0012	Schwartz et al. (1994, Table 2)
Upper Respiratory Symptoms	Daily upper respiratory symptom incidence among asthmatic children	0.3419	Pope et al. (1991, Table 2)
Work Loss Days	Daily WLD incidence rate per person (18–65) - Aged 18–24 - Aged 25–44 - Aged 45–64	 0.00540 0.00678 0.00492	1996 HIS (Adams, Hendershot, and Marano, 1999, Table 41); U.S. Bureau of the Census (2000)
Minor Restricted-Activity Days	Daily MRAD incidence rate per person	0.02137	Ostro and Rothschild (1989, p. 243)

[a]   The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.

[b]   See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/.

[c]   See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/.

[d]   Lower respiratory symptoms are defined as two or more of the following: cough, chest pain, phlegm, and wheeze.

Baseline age, cause, and county-specific mortality rates were obtained from the U.S. Centers for Disease Control and Prevention (CDC) for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year-old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

To estimate age- and county-specific mortality rates in years 2000 through 2020, we calculated adjustment factors, based on a series of Census Bureau projected national mortality rates, to adjust the CDC Wonder age- and county-specific mortality rates in 1996-1998 to corresponding rates for each future year. For the analysis year 2020, these adjustment factors ranged across age categories from 0.76 to 0.86

For the set of endpoints affecting the asthmatic population, in addition to baseline incidence rates, prevalence rates of asthma in the population are needed to define the applicable population. Table 4.2-3 lists the baseline incidence rates and their sources for asthma symptom endpoints. Table 4.2-4 lists the prevalence rates used to determine the

applicable population for asthma symptom endpoints. Note that these reflect current asthma prevalence and assume no change in prevalence rates in future years.

Table 4.2-4. Asthma Prevalence Rates Used to Estimate Asthmatic Populations in Impact Functions

POPULATION GROUP	ASTHMA PREVALENCE RATES	
	Value	Source
All Ages	0.0386	American Lung Association (2002, Table 7)—based on 1999 HIS
< 18	0.0527	American Lung Association (2002, Table 7)—based on 1999 HIS
5–17	0.0567	American Lung Association (2002, Table 7)—based on 1999 HIS
18–44	0.0371	American Lung Association (2002, Table 7)—based on 1999 HIS
45–64	0.0333	American Lung Association (2002, Table 7)—based on 1999 HIS
65+	0.0221	American Lung Association (2002, Table 7)—based on 1999 HIS
Male, 27+	0.021	2000 HIS public use data files[a]
African American, 5 to 17	0.0726	American Lung Association (2002, Table 9)—based on 1999 HIS
African American, <18	0.0735	American Lung Association (2002, Table 9)—based on 1999 HIS

[a]   See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000/.

4.2.1.4 Baseline Incidence Rates for Ozone-related Health Impacts

Epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the *relative risk* of a health effect, rather than estimating the absolute number of avoided cases. For example, a typical result might be that a 100 ppb decrease in daily ozone levels might, in turn, decrease hospital admissions by 3 percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases. A baseline incidence rate is the estimate of the number of cases of the health effect per year in the assessment location, as it corresponds to baseline pollutant levels in that location. To derive the total baseline incidence per year, this rate must be multiplied by the corresponding population number. For example, if the baseline incidence rate is the number of cases per year per 100,000 people, that number must be multiplied by the number of 100,000s in the population.

Table 4.2-5 summarizes the sources of baseline incidence rates and provides average incidence rates for the endpoints included in the analysis. For both baseline incidence and prevalence data, we used age-specific rates where available. We applied concentration-response functions to individual age groups and then summed over the relevant age range to provide an estimate of total population benefits. In most cases, we used a single national incidence rate, due to a lack of more spatially disaggregated data. Whenever possible, the national rates used are national averages, because these data are most applicable to a national assessment of benefits. For some studies, however, the only available incidence information comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level. Regional incidence rates are available for hospital admissions, and county-level data are available for premature mortality. We have projected mortality rates such that future mortality rates are consistent with our projections of population growth.

Table 4.2-5. National Average Baseline Incidence Rates[a]

ENDPOINT	SOURCE	NOTES	RATE PER 100 PEOPLE PER YEAR[D] BY AGE GROUP						
			<18	*18-24*	*25-34*	*35-44*	*45-54*	*55-64*	*65+*
Mortality	CDC Compressed Mortality File, accessed through CDC Wonder (1996-1998)	non-accidental	0.03	0.02	0.06	0.15	0.38	1.01	4.94
Respiratory Hospital Admissions.	1999 NHDS public use data files[b]	incidence	0.04	0.08	0.21	0.68	1.93	4.40	11.63
Asthma ER visits	2000 NHAMCS public use data files[c]; 1999 NHDS public use data files[b]	incidence	1.01	1.09	0.75	0.44	0.35	0.43	0.23
Minor Restricted Activity Days (MRADs)	Ostro and Rothschild (1989, p. 243)	incidence	–	780	780	780	780	780	–
School Loss Days	National Center for Education Statistics (1996) and 1996 HIS (Adams et al., 1999, Table 47); estimate of 180 school days per year	all-cause	990	–	–	–	–	–	–

[a] The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS - National Hospital Discharge Survey; NHAMCS - National Hospital Ambulatory Medical Care Survey.
[b] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/
[c] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/
[d] All of the rates reported here are population-weighted incidence rates per 100 people per year. Additional details on the incidence and prevalence rates, as well as the sources for these rates are available upon request.

Table 4.2-5. National Average Baseline Incidence Rates (continued)

ENDPOINT	SOURCE	NOTES		RATE PER 100 PEOPLE PER YEAR
Asthma Exacerbations	Ostro et al. (2001)	Incidence (and prevalence) among asthmatic African-American children	Daily wheeze Daily cough Daily dyspnea	0.08 (0.17) 0.07 (0.15) 0.04 (0.07)
	Vedal et al. (1998)	Incidence (and prevalence) among asthmatic children	Daily wheeze Daily cough Daily dyspnea	0.04 0.09 0.05

4.2.2 Manipulating Air Quality Modeling Data for Health Impacts Analysis

In Chapter 3, we summarized the methods for and results of estimating air quality for the 2020 base case and proposed ECA scenario. These air quality results are in turn associated with human populations to estimate changes in health effects. For the purposes of this analysis, we focus on the health effects that have been linked to ambient changes in ozone and $PM_{2.5}$ related to emission reductions estimated to occur due to the proposed ECA. We

estimate ambient $PM_{2.5}$ and ozone concentrations using the Community Multiscale Air Quality model (CMAQ). This section describes how we converted the CMAQ modeling output into full-season profiles suitable for the health impacts analysis.

4.2.2.1 General Methodology

First, we extracted hourly, surface-layer PM and ozone concentrations for each grid cell from the standard CMAQ output files. For ozone, these model predictions are used in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.[H,I] The predicted changes in ozone concentrations from the future-year base case to future-year control scenario serve as inputs to the health and welfare impact functions of the benefits analysis (i.e., BenMAP).

To estimate ozone-related health effects for the contiguous United States, full-season ozone data are required for every BenMAP grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in two steps: (1) we combined monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 12-km by 12-km population grid cells for the contiguous 48 states, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily 8-hour maximum.[J,K]

For $PM_{2.5}$, we also use the model predictions in conjunction with observed monitor data. CMAQ generates predictions of hourly PM species concentrations for every grid. The species include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). $PM_{2.5}$ is calculated as the sum of the primary fine fraction and all of the secondarily formed particles. Future-year estimates of $PM_{2.5}$ were calculated using relative reduction factors (RRFs) applied to 2002 ambient $PM_{2.5}$ and $PM_{2.5}$ species concentrations. A gridded field of $PM_{2.5}$ concentrations was created by interpolating Federal Reference Monitor ambient data and IMPROVE ambient data. Gridded fields of $PM_{2.5}$ species concentrations were created by interpolating US EPA speciation network (ESPN) ambient data and IMPROVE data. The ambient data were interpolated to the CMAQ 12 km grid.

The procedures for determining the RRFs are similar to those in US EPA's draft guidance for modeling the $PM_{2.5}$ standard (EPA, 1999). The guidance recommends that model predictions be used in a relative sense to estimate changes expected to occur in each major $PM_{2.5}$ species. The procedure for calculating future-year $PM_{2.5}$ design values is called

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[H] The ozone season for this analysis is defined as the 5-month period from May to September.
[I] Based on AIRS, there were 961 ozone monitors with sufficient data (i.e., 50 percent or more days reporting at least nine hourly observations per day [8 am to 8 pm] during the ozone season).
[J] The 12-km grid squares contain the population data used in the health benefits analysis model, BenMAP.
[K] This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation. See the BenMAP manual for technical details, available for download at http://www.epa.gov/air/benmap.

the "Speciated Modeled Attainment Test (SMAT)." EPA used this procedure to estimate the ambient impacts of the proposed ECA controls.

4.2.2.2 Emissions Inventory Boundary Distance Error

As noted in Appendix 2F to Chapter 2, the air quality modeling used for this analysis is based on inventory estimates that were modeled using incorrect boundary information. The impact of this difference, while modest, leads to an underestimate of the benefits that are presented in this Chapter. Please refer to Appendix 2F for more information on the emissions excluded from the health impacts analysis of the proposed ECA.

4.3 Methods for Describing Uncertainty

For this analysis, consistent with the approach used in the analyses for the recent PM and Ozone NAAQS, we addressed key sources of uncertainty through Monte Carlo propagation of uncertainty in the concentration-response (CR) functions. It should be noted that the Monte Carlo-generated distributions of health impacts reflect only some of the uncertainties in the input parameters. Uncertainties associated with emissions, air quality modeling, populations, and baseline health effect incidence rates are not represented in the distributions of avoided health impacts associated with the implementation of the proposed ECA. A complete description of uncertainty related to health impacts analyses can be found in the regulatory impact analysis drafted in support of the final Ozone NAAQS analysis.[114]

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