APPENDIX A

OVERVIEW OF EMISSIONS AND COST MODELING TOOLS FOR ESTIMATING THE PROSPECTIVE BENEFITS AND COSTS OF THE 1990 CLEAN AIR ACT AMENDMENTS

In its analysis of the costs and benefits of the Clean Air Act Amendments of 1990 (CAAA), EPA will employ several modeling tools to estimate CAAA compliance costs and project emissions under different regulatory scenarios. This appendix describes the modeling systems EPA proposes to use for these aspects of the second prospective analysis, as well as several other models that EPA considered in developing this analytic blueprint. Several of the organizations that developed the models described here have produced model documentation, which is cited in the reference list located at the end of this appendix.

The first section of this appendix describes ControlNet, EPA's proposed model for estimating costs and projecting emissions for non-EGU point sources in the second prospective. In its analysis of electric utility emissions and costs, EPA plans to use the Integrated Planning Model (IPM), which is presented in the second section of this appendix. EPA also considered Resources for the Future's Haiku model for this component of the second prospective. An overview of Haiku follows the presentation of IPM. After describing the main characteristics of Haiku, the focus of this appendix then shifts to computable general equilibrium (CGE) modeling. Although EPA examined several economic models to assess the social costs of the CAAA, the Agency eventually narrowed its options to two modeling systems: the Jorgenson/Ho/Wilcoxen (J/H/W) model and the All-Modular Industry Growth Assessment model (AMIGA).

MODELING EMISSIONS AND DIRECT COSTS

Since the emissions reductions and compliance costs attributable to the CAAA are so closely related, some of the tools that EPA proposes to use in the second prospective solve for the two simultaneously. This type of tool is particularly useful for analyzing rules that allow sources to choose from an array of control strategies, each of which has different implications for emissions and costs. Summaries of these models are presented below. In addition, the reference list at the end of this appendix contains a citation for the ControlNet User's Guide.

ControlNet

To support the development and implementation of the National Ambient Air Quality Standards (NAAQS) for criteria pollutants established by the U.S. Environmental Protection Agency (EPA), Pechan developed ControlNET. ControlNET is a relational database system in which control

technologies are linked to sources within point, area, and mobile sources emissions inventories. The database of control measures contains comprehensive information on each measure, including control efficiency and costing data. Currently, ControlNET contains 453 source category and pollutant-specific control measures, applied within a 759,733 record data file. Controls are supplied for all criteria pollutants and NH_3. The control measure data file in ControlNET includes each technology's control efficiency, calculated emission reductions by source, and estimates of the costs (annual and capital) for application of each control.

ControlNET includes data gathered for more than 450 different control measures for NO_x, VOC, SO_2, PM_{10}, $PM_{2.5}$, and NH_3 for utility, non-utility point, area, mobile, and non-road sources. Each control measure has an associated control efficiency, annual cost, capital cost, and operation and maintenance costs. Every control measure is applied to relevant sources in the 1999 National Emission Inventory (NEI) to create a large database of possible controls with their associated emission reductions and costs. Pechan's recently developed interface for ControlNET allows users to view and filter the database of all possible controls (by state, county, regional area, SIC, SCC, sector, pollutant, and cost per ton value) and specify specific controls to create control scenarios.

Because ControlNET is designed for evaluating the cost and effectiveness of adding additional controls to point, area and mobile sources, the model's control cost equations were developed so that information typically reported in emissions databases are the primary drivers of the equations included in the model. Key variables included in these databases include stack gas flow rate, design capacity, and emissions. Stack gas flow rate is the primary variable used for estimating the costs of PM controls such as electrostatic precipitators and baghouses, whose sizing and cost are a function of flow rate. Flow rates typically reported in point source databases are of central importance in estimating stack gas plume rise. Estimates of control costs for many other point source controls, such as SCR, NSCR, and low NO_x burners, are based largely on design capacity. For sources such as electric utility boilers, design capacity is usually reported in megawatts, while for non-EGUs, design capacity is reported in SCC units per year or per hour, where SCC units are normally fuel consumption or the production rate. Finally, all cost equations are designed to use emissions as an important variable in case other primary variables (e.g., flow rate or design capacity) are missing, or for area source categories for which no other information related to the size of individual sources is available.

IPM

This appendix provides a brief overview of IPM. Additional information is available in IPM's supporting documentation as cited in the reference list at the end of this appendix.

IPM is a dynamic, linear programming model of the electric power sector that represents a number of key components of energy markets--fuel markets, emission markets, and electricity markets--as well as the linkages between them. The model determines the least-cost method of meeting energy and peak demand requirements over a specified period of time, considering a

number of (non)regulatory constraints (e.g. emissions limits, transmission capabilities, fuel market constraints, etc.).

IPM models electricity markets in different regions of the country by modeling electricity demand, generation, and intra-regional transmission and distribution. All existing power generators are captured in the analysis, including those that use renewable resources and independent and cogeneration facilities that sell back to the grid. In addition, IPM accounts for demand-side resource options and the hourly load impacts they have.

IPM endogenously forecasts fuel prices for coal, natural gas, and biomass by balancing fuel demand and supply for electric generation. The model also includes information on fuel quality parameters. Other items IPM estimates endogenously include emissions changes, regional wholesale energy and capacity prices, incremental electric power system costs, changes in fuel use, and capacity and dispatch projections.

To simplify the model, IPM analyzes model plants over a series of model years. Model plants represent aggregations of existing units; retrofit, repowering, and retirement options available to existing units; and new units the model can build over the time horizon of a model run. Model years group a cluster of years together, which significantly lowers model run time.

As a linear programming model, IPM minimizes an objective function representing the summation of all costs incurred by the electricity sector over the entire planning horizon of the model, expressed as the net present value of all component costs. Since IPM minimizes the total cost function for the entire utility sector, the choices that a model plant makes in the model may not represent the least-cost solution for that particular plant. Choices that minimize costs for the entire sector might not always coincide with choices that minimize costs for individual units.

To minimize the value of the objective function, IPM systematically changes the value of several decision variables that directly affect component costs. The decision variables in IPM are as follows:

1. *Generation Dispatch Decision Variables* represent generation from each model plant. IPM uses these variables to calculate plant fuel costs and plant VOM costs.

2. *Capacity Decision Variables* represent the capacity of each existing model plant and possible new plants in each model run year. These variables are necessary for calculation of total fixed operating and maintenance (FOM) costs for each model plant as well as the capital costs associated with capacity addition.

3. *Transmission Decision Variables* represent electricity transmission along each transmission link between model regions in each run year. IPM multiplies these

variables by variable transmission cost rates to obtain the total cost of transmission across each link.

4. *Emission Allowance Decision Variables* represent the total number of emission allowances for a given model year that are bought and sold in that or subsequent run years. IPM uses the emission allowance decision variables to capture the inter-temporal trading and banking of allowances.

5. *Fuel Decision Variables* represent the quantity of fuel delivered from each fuel supply region to model plants in each demand region for each fuel type and each model run year. These variables are compared to constraints (see below) that define the types of fuel that each model plant is eligible to use and the supply regions eligible to provide fuel to each specific model plant.

Manipulation of these decision variables is subject to a number of constraints:

6. *Reserve Margin Constraints*–Each generating unit must maintain a minimum margin of reserve capacity.

7. *Demand Constraints*–Model plants must meet demand. The model divides regional annual demand into seasonal load segments as specified by a load duration curve, represented as a step function. Each segment of the function defines the minimum amount of generation required to meet the region's demand in the specified season.

8. *Capacity Constraints*–Generation at each model plant may not exceed maximum plant generating capacity.

9. *Turn Down/Area Protection Constraints*–Some generating units can shut down at night, but others must operate at all times.

10. *Emissions Constraints*–Model plants must comply with emission constraints. IPM can consider any of a number of emissions constraints for SO2, NOx, mercury, and CO2, including tonnage caps and maximum emission rates.

11. *Transmission Constraints*–Transmission is constrained by the maximum capacity of each transmission link or the maximum capacity of two or more links (joint limits) to different regions.

12. *Fuel Supply Constraints*–Each generating unit can consume only those fuels compatible with its particular generating technology. In addition, a plant can only purchase fuels from supply regions eligible to provide fuel to that plant.

Haiku

We present a brief description of Haiku in this appendix. The interested reader can find additional information in the Haiku reference manual, which is cited in the reference list at the end of this appendix.

Developed by Resources for the Future, Haiku is a simulation model of regional electricity markets and interregional electricity trade in the United States. Using an iterative convergence algorithm, Haiku simulates utilities' responses to public policy choices and estimates multiple equilibria in multiple linked markets. In the past Haiku has been used to model responses to potential NO_x, SO_2, and CO_2 emissions regulations.

Haiku simulates several aspects of utility behavior. Using separate electricity demand functions for residential, commercial, and industrial customers, Haiku estimates electricity prices, the composition of electricity supply, inter-regional electricity trading among National Electricity Reliability Council (NERC) regions, and emissions of NO_x, SO_2, CO_2, and mercury. Estimates of NO_x and SO_2 emissions are based in part on the endogenous selection of NO_x and SO_2 control technologies. Haiku estimates generator dispatch based on the minimization of the short-run variable costs of generation. Estimation of all these items occurs for 4-6 model run years over a 20-year time horizon.

In estimating market equilibrium, Haiku first finds an equilibrium for each region of the country before solving for the level of inter-regional electricity trade necessary for prices to equilibrate. At the regional level, Haiku estimates market equilibrium for each of four time periods (super peak, peak, shoulder, and baseload), three seasons (summer, winter, and spring/fall), and each of 13 NERC subregions. Regional supply functions are constructed using information on capacity net of outages, operating and maintenance costs (including pollution control costs), and fuel costs for 46 model plants (31 existing, 15 possible in the future), each representing a group of generators aggregated by region, fuel type, technology and vintage classifications. Haiku adjusts model plant supply functions to reflect endogenously selected NO_x and SO_2 emissions control technologies.

Haiku also includes modules for coal and natural gas markets that calculate prices based on changes in factor demand. All other fuel prices are specified exogenously. Haiku holds the cost of capital and the cost of labor constant.

COMPUTABLE GENERAL EQUILIBRIUM MODELS

As part of its update of the 1999 *Benefits and Costs of the Clean Air Act: 1990 to 2010* (*Prospective Analysis)*, EPA proposes the use of a computable general equilibrium (CGE) modeling approach to estimate the impacts of the 1990 Clean Air Act Amendments (CAAA) on the U.S. economy. EPA's *Benefits and Costs of the Clean Air Act: 1970 to 1990* included a CGE analysis of the social costs associated with the implementation of the Clean Air Act's provisions, using the Jorgenson/Wilcoxen dynamic CGE model of the U.S. economy. However, the Agency's 1999 *Prospective Analysis* did not include a general equilibrium modeling approach due in part to the level of effort required to calibrate and run a CGE model, as well as limits on the resolution of available cost data.

CGE modeling efforts estimate the comprehensive macroeconomic effects of broad policies (such as the Clean Air Act) that affect multiple industries and products within the economy.[1] These models provide a relatively complete estimate of the social costs of regulation because they capture both the positive and negative impacts of price changes throughout the economy. At a minimum, CGE models estimate changes in production by sector for the geographic scope of the model. In addition, most identify employment effects by sector, relative price changes among both inputs and products, and the impacts of policies on trade (i.e., changes in levels of import and export). Finally, several recent efforts estimate net impacts by incorporating productivity-linked benefits (e.g., avoided health effects) into modeling scenarios.

Given these recent advances in CGE design, we have reviewed recent CGE modeling efforts that address environmental policy. We have identified two potential CGE modeling options:[2]

< **Jorgenson/Ho/Wilcoxen Model of the U.S. Economy:** An update of the dynamic national CGE model used to assess the social costs of Clean Air Act in EPA's Retrospective Analysis. The model was recently updated to address benefits and to perform prospective assessments of impacts.

< **All-Modular Industry Growth Assessment Modeling System (AMIGA):**

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[1] For a brief overview of the use of different types of general equilibrium models (i.e., input/output models, linear programming models, and CGE models) as well as partial equilibrium and multi-market models, in the assessment of costs related to environmental regulation, see EPA's *Guidelines for Performing Economic Analyses*, September 2000, EPA 240-R-00-003.

[2] In addition, a number of available "world models" (e.g., Wilcoxen's G-Cubed Model and MIT's EPPA recursive-dynamic CGE model, and CRA's Multi-sector, Multi-regional Trade (MS-MRT) model) address general equilibrium effects of international environmental policy issues, such as efforts aimed at reducing greenhouse gas emissions. While many world models have regional (i.e., national or multi-national) capabilities, the level of aggregation in these models is generally too high to address specific sectors within a single national economy. If EPA wishes to address potential international trade or environmental effects associated with the Clean Air Act, a limited application of one of the available world models may be useful.

A dynamic CGE model recently used for the Jeffords-Lieberman request for an analysis of a multi-pollutant emission reduction strategy. The model possesses a rich representation of technology and disaggregates the economy to a finer degree than most CGE models.

Our review concludes that both the Jorgenson/Ho/Wilcoxen (J/H/W) model and AMIGA could be used to assess the prospective impacts of the 1990 CAAA. Below we provide a brief overview of each of the models; Exhibit A-1 provides a summary of key model features.

Exhibit A-1

Comparison of J/H/W and AMIGA General Equilibrium Models

Traits	**Jorgenson/Ho/Wilcoxen**	**AMIGA**
Calibration/ Estimation	Econometrically estimated from 25 years of data.	Calibrated to 1992 BEA data.
Number of Sectors	35 sectors included in model	200 sectors included in model
Reporting	Economy wide and by industry	Economy wide and by industry
Employment Impacts	Reported in model	Reported in model
Treatment of Technology	Exogenous and endogenous components of technological progress.	Extremely rich representation of technology. Technology assumptions based on EIA projections of technology cost and efficiency. Updated periodically.
Treatment of taxation	Captures effects resulting from the interaction of taxes and environmental policy.	Captures effects resulting from the interaction of taxes and environmental policy.
Intertemporal Optimization	The model calculates a dynamic equilibrium where consumers and capital owners optimize with consideration for the future.	The model calculates a dynamic equilibrium where consumers and capital owners optimize with consideration for the future.
Treatment of Productivity Increases from Health Improvements	Can introduce exogenously. Improves the quality component of labor.	Can introduce exogenously by entering estimated change in worker productivity.
Peer Reviewed/ Published works	Theoretical basis of the model peer reviewed in several journal articles. The model itself is not available for review.	Peer reviewed paper forthcoming in *Energy Economics*. Unpublished reviews from Cornell, MIT, and EMF. The model code is available for review.

Exhibit A-1

Comparison of J/H/W and AMIGA General Equilibrium Models

Traits	**Jorgenson/Ho/Wilcoxen**	**AMIGA**
Past Uses	CAA Retrospective, NCEE applications	Jeffords-Lieberman request on a multi-pollutant emissions strategy, Possible use for Lieberman-McCain greenhouse gas proposal
Cost	Unclear	Less than $100,000 for this application.
Availability	Current Production Changes: Summer 2003. Consumption changes: Summer or Fall 2004	Summer 2003

Jorgenson/Ho/Wilcoxen Model

The Jorgenson/Ho/Wilcoxen (J/H/W) model is a dynamic computable general equilibrium (CGE) model that was used to estimate the social costs associated with regulations under the 1970 Clean Air Act.[3] The model estimates several macroeconomic effects resulting from the compliance with environmental regulations, including changes in gross national product (GNP), aggregate consumption, and energy flows between sectors. The model estimates long-run changes in the supply of production factors (i.e., capital, labor, imports, and intermediate inputs to production) and rates of technical change, degrees of substitutability among inputs and commodities in production and final demand (i.e., levels of consumption, investment, government activity, and foreign trade). It includes the following basic features:[4]

- **Dynamic model:** The J/H/W model is a dynamic model. In other words, it contains functions that update time-dependent variables (e.g., labor supply or technology development) endogenously, based on projections of the trends for these variables in the economy and the activity that is predicted in the model. An advantage of dynamic models (in addition to a potentially more realistic reflection of changes in activity over time) is that they can be used

[3] The current version of this model reflects efforts by Mun F. Ho and is referred to as the Jorgenson/Ho/Wilcoxen Intertemporal General Equilibrium Model.

[4] For a more detailed description of the Jorgenson/Wilcoxen model and its application to the Clean Air Act, see Appendix B of EPA's *Benefits and Costs of the Clean Air Act: 1970 to 1990.*

to develop and compare analyses with different time horizons.[5]

- **Detailed production and consumption functions:** The J/W/H model contains a unified accounting framework consistent with national product accounts for 35 distinct industry sectors, as well as household and government functions. This allows for a relatively detailed treatment of impacts in industries specifically affected by the CAA and amendments, including the incorporation of industry-specific compliance costs.

- **Parameters estimated econometrically from historical data:** The J/H/W model incorporates information on economic activity (including production factor pricing and technological change) dating back to 1977. These data are used to predict household and firm behavior in a manner consistent with the historical record, as opposed to relying on theoretical values and behavioral predictions.

In addition, the J/H/W model incorporates a detailed representation of saving and investment, reflecting changes in behavior as prices change as a result of policy (e.g., energy prices). Consistent with long-run assumptions, the model reflects free mobility of labor and capital between industries that is appropriate for the 30-year time horizon considered in the second prospective.

[5] In contrast, *static* models provide a single projection of a market's adjustment to a new equilibrium after a shock (e.g., a policy) has been introduced; the time horizon is determined by the point at which the market achieves its new balance.

AMIGA

We present a brief summary of AMIGA in this appendix.[6] More detailed information is available in AMIGA's supporting documentation as cited in the reference list located at the end of this appendix.

AMIGA is a dynamic general equilibrium modeling system of the U.S. economy that covers the period from 1992 through 2030. It was originally developed by the Policy and Economic Analysis Group at the Argonne National Laboratory to evaluate the effects of policy combinations dealing with climate change. AMIGA includes information on more than 200 sectors of the economy, which allows it to present extremely disaggregated information on the effects of policy changes. Some of AMIGA's most important characteristics include the following:

- The model computes a full-employment general equilibrium solution for demands, prices, costs, and outputs of interrelated products, including induced activities such as transportation and wholesale/retail trade.

- AMIGA calculates national income, Gross Domestic Product (GDP), employment, a comprehensive list of consumption goods and services, the trade balance, and net foreign assets and examines inflationary pressures.

- The model projects economic growth paths and long-term, dynamic effects of alternative investments including accumulation of residential, vehicle, and producer capital stocks.

- AMIGA reads in files with detailed lists of technologies (currently with a focus on the electric power generating industry) containing performance characteristics, availability status, costs, anticipated learning effects, and emission rates where appropriate.

- AMIGA benchmarks to the 1992 Bureau of Economic Analysis (BEA) interindustry data for more than 200 sectors of the economy.

The AMIGA modeling system is programmed in the C language. Like other large, integrated modeling systems, AMIGA includes modules for a number of key sectors of the economy. The output of each module may be used as input for other modules. AMIGA includes the following modules:

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[6] This section is based largely on the abstract of *A Framework for Economic Impact Analysis and Industry Growth Assessment: Description of the AMIGA System*, Donald A. Hanson, Argonne National Laboratory, Argonne, IL, April 1999. Several excerpts included in this section are drawn directly from this document.

- **Household demand**: AMIGA includes a module for household demand which uses consumer preferences, relative prices of delivered goods, and permanent income to determine consumer spending.

- **Government purchases and programs**: Most government expenditures are taken to be exogenous. Energy purchases, however, are based on the energy efficiency of the stock of equipment used by government agencies.

- **Residential buildings and appliances**: This module represents existing housing and appliance stocks, available new technologies, and near commercialized technologies soon to be available. It allows the average efficiency of household equipment and residential structures to change with time. It also allows the penetration of more efficient technologies to lower the cost of supplying energy-intensive building services.

- **Commercial buildings and appliances**: This component of the model includes floor space and capital equipment services to the commercial business and government sectors of the economy including personal and business services, administrative offices, wholesale/retail trade, warehousing, financial services, schools and hospitals.

- **Motor vehicles**: This module provides personal transportation services to households, businesses, and federal, state and local governments. It allows transportation demand and fuel efficiencies to change over time.

- **Utilities**: This module represents the operation of the existing stock of generating equipment and power plants to determine their capacity factors, dispatching units against the load curve in order of variable costs. It also can incorporate the costs of SO_2 emission allowances and any future carbon charges.

- **Industrial production activities**: Industrial production activities are organized into separate modules to more easily handle the representation of different production technologies and their characteristics. Each module contains representations of labor, capital, and energy substitutions using a hierarchy of production functions. AMIGA currently uses five distinct lists/modules. Within these modules is information on more than 200 individual industry sectors.

- **Industrial Capital**: AMIGA contains disaggregated data on the capital stocks of a number of industries, allowing the model to capture effects such as the depreciation and retirement of capital, as well as substitution between different types of capital equipment.

Model Structure

AMIGA goes through the following series of steps to arrive at equilibrium:

1. Computation of all prices.

2. Calculation of flow quantities, such as sector output, demands and labor effort, taking prices, market shares, and input intensities as given.

3. Verification that all variables have converged with sufficient precision. If they have not converged enough, the model readjusts wages and/or the opportunity cost of capital so that excess demand for labor and capital is closer to zero.

4. The model returns to step 2, given the revised values for input intensities, market shares, and flow quantities.

The model repeats this routine until it reaches equilibrium. Since AMIGA calculates equilibria within and between modules simultaneously, the model's operating shell first calls pricing programs from the individual modules, then the input intensity programs, followed by the quantity programs.

Social Costs

AMIGA can capture the social costs associated with environmental regulation in several ways. Since the model allows prices to change throughout the economy in both intermediate and final output markets, equilibrium quantities under different regulatory scenarios can change from their pre-regulatory equilibrium, which allows the model to capture deadweight losses associated with regulation. In addition, AMIGA incorporates taxes into its modeling framework, so it therefore measures any tax interaction effects that result from regulation.

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APPENDIX B

MODEL PERFORMANCE DOCUMENTATION FOR REMSAD, CAMX, AND OTHER COMPETING AIR QUALITY MODELS

This appendix provides references to a collection of technical documentation used in support of the air quality model selections made in Chapter 5. This documentation includes model evaluations, user's guides, model performance statistics, and comparative analyses and peer reviews of a number of competing air quality modeling systems. EPA's decision to use REMSAD for PM modeling and CAMx for ozone modeling relied upon careful consideration of the results presented in these documents. Also provided are references to documentation supporting the incorporation of the BEIS-3 emissions inventory model to treat biogenic emissions. The references listed here are grouped by air quality model, and the order of references proceeds from general information to model evaluation and finally to comparative analyses:

Regulatory Modeling System for Aerosols and Deposition (REMSAD)

- ICF Consulting. 2002. *User's Guide to the Regional Modeling System for Aerosols and Deposition (REMSAD) Version 7*. July. http://remsad.saintl.com/documents/remsad_users_guide_07-22-02.pdf

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- *An Assessment of Models-3 Performance During the 1999 SOS Nashville Study*. **http://www.cmascenter.org/workshop/session3/bailey_abstract.pdf**

- *2002 Models-3 Users' Workshop References List*. **http://www.cmascenter.org/workshop/2002wspresent.html**

Comprehensive Air Quality Model with Extensions (CAMx)

- *User's Guide to the Comprehensive Air Quality Model with Extensions (CAMx) Version 3.10.* 2002. **http://www.camx.com/pdf/CAMx3.UsersGuide.020410.pdf**

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Biogenic Emissions Inventory System (BEIS)

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  http://www.epa.gov/asmdnerl/biogen.html

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  http://www.epa.gov/ttn/chief/conference/ei11/modeling/arunachalampres.pdf

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APPENDIX C

POPULATION FORECASTING FOR BENEFITS ANALYSIS

This appendix summarizes the steps used to estimate 2020 and 2030 population. In addition, we include a table with age-specific population estimates by state for the years 2000, 2020, and 2030.

Population Grid Cells

BenMAP calculates health impacts at the level of U.S. counties as well as for a variety of grid structures used in air quality modeling (e.g.,, REMSAD, and CAMx). In this description, we use the term "population grid-cells" to refer to counties or the cells within an air quality modeling grid. The foundation for calculating the population level in the population grid-cells is the 2000 Census block data.[1] A separate application developed by Abt Associates, called "PopGrid," combines the Census block data with any user-specified set of population grid-cells, so long as they are defined by a GIS shape file.

If the center of a Census block falls within a population grid-cell, PopGrid assigns the block population to this particular population grid-cell. Note that the grid-cells in air quality model, such as REMSAD and CAMx, may cross multiple county boundaries. To account for this, PopGrid keeps track of the total number of people by county within a particular populatin grid-cell. Keeping track of the total number of people in a county is useful in the estimation of adverse health effects, where the calculation of premature mortality depends on county-level mortality rates. It is also useful in the presentation of health benefits, when users may want estimates at the state- and county-level, as opposed to estimates by, say, the area covered by an air quality model.

Within any given population grid-cell, BenMAP has 256 demographic variables, including 180 unique racial-gender-age groups: 19 age groups by gender by 5 racial groups (19*2*5=180). In addition there is an Hispanic ethnicity variable, which includes a number of different racial groups, as well as a number of variables that aggregate the population by race and gender. Exhibit C-1 presents the 256 population variables available in BenMAP. As discussed below, these variables are available for use in developing age estimates in whatever grouping desired by the user.

[1] Geolytics (2001; 2002a) provided the 1990 and 2000 census data.

Exhibit C-1. Demographic Groups and Variables Available in BenMAP

Racial/Ethnic Group	Gender	Age	# Variables
White, African American, Asian, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	228
All	–	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	19
All	Female, Male	–	2
White, African American, Asian, American Indian, Other, Hispanic	–	–	6
All	–	–	1

Census Data 2000

In addition to forecasting post-2000 population levels based on the 2000 Census, BenMAP also allows the user to estimate the impacts for 1991-1999 by interpolating between the results of the 1990 and 2000 Census. As a result, we have developed a consistent set of demographic variables, based on the 1990 Census, which provides somewhat less detail than the 2000 Census.

The 2000 Census allows respondents to choose more than one racial category, unlike the 1990 Census, which allowed only one choice. As a result there are seven racial categories in the 2000 Census versus five in the 1990 Census (Exhibit C-2). To make the 2000 Census data consistent with the 1990 Census, we reduced the seven racial groups to the five used in the 1990 Census.

The initial data set at the block level includes 368 demographic groups: seven racial groups and Hispanic ethnicity, by 23 pre-defined age groups by gender (Exhibit C-2). Because the 2000 Census includes somewhat different age groupings than that for the final set generated for the 1990 Census. Age variables 15-17 and 18-19 are combined, 20, 21, and 22-24 are combined, 60-61 and 62-24 are combined, and 65-66 and 67-69 are combined at the block level. One variable, under 5 years, must be split into two variables (Under 1 and 1-4 years). Assuming that the population is uniformly distributed within age groups, we apply a factor of 1/5 to create the 0-1 age group and 4/5 to create the 1-4 age group.

Exhibit C-2. Race, Ethnicity and Age Variables in 2000 Census Block Data

	Race / Ethnicity	Gender	Age
Initial Variables	White Alone, Black Alone, Native American Alone, Asian Alone, Pacific Islander / Hawaiian Alone, Other Alone, Two or More Alone, Hispanic (Non-Exclusive)	Male, Female	0-5, 5-10, 10-14, 15-17, 18-19, 20, 21, 22-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-61, 62-64, 65-66, 67-69, 70-74, 75-79, 80-84 85+
Final Variables (identical to 1990 variables)	White, African American, Asian & Pacific Islander, American Indian, Other, Hispanic	Female, Male	0-1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+

Source: Geolytics (2002a). Note: Some population values were errors in the original Census data (e.g., values of a billion or more). Following personal communication with Geolytics, these were set to zero.

Matching Racial Categories in the 1990 and 2000 Censuses

Unlike the 1990 Census, respondents in the 2000 Census respondents could check more than one box for race, so the reported results included a grouping of individuals that had checked two or more racial categories. In addition, the 2000 Census separately reported the categories "Pacific Islander / Hawaiian Along" and "Asian Alone." To make the racial groupings comparable with the 1990 Census, we first combined Pacific Islander / Hawaiian Alone with the Asian Alone category to form the category Asian and Pacific Islander category. Then we divided the category Two-or-More between the remaining five racial categories.

Exhibit C-3 presents the estimated percentage of the national population by five racial groups: (1) American Indian or Alaska Native, (2) Asian or Pacific Islander, (3) Black, (4) White, and (5) Other, as well as for four combinations: (1) American Indian or Alaska Native (AIAN)/White, (2) Asian or Pacific Islander (API)/White, (3) Black/White, and (4) Other combinations. Slightly over 98 percent of individuals chose a single racial category, with 1.45 percent choosing three AIAN/White, API/White, and Black/White, and 0.30 choosing other combinations (e.g., Black/Asian). Exhibit C-3 also presents the estimated primary racial affiliation of individuals in these subcategories if they were to choose a single racial affiliation.

Exhibit C-3. Distribution of Racial Groups

Racial Category	% of Total U.S. Population [a]	% of Population in Sub-Groups by Primary Racial Affiliation [b]					
		AIAN	API	Black	White	Other	All
American Indian or Alaska Native (AIAN)	0.85	100	–	–	–	–	100
Asian or Pacific Islander (API)	3.35	–	100	–	–	–	100
Black	12.07	–	–	100	–	–	100
White	79.72	–	–	–	100	–	100
Other race	2.25	–	–	–	–	100	100
AIAN/White	0.89	12.4	–	–	80.9	6.7	100
API/White	0.30	–	34.6	–	46.9	18.4	100
Black/White	0.26	–	–	48.2	25.2	26.6	100
Other combinations [c]	0.30	–	–	–	–	100.0	100
Two-or-More Sub-Total [d]	1.75	6.3	5.9	7.2	52.9	27.7	100

[a] All percentages weighted to be nationally representative. Percentages taken from Parker and Makuc (2001, Table 2), who cited the National Health Interview Survey 1993-1995, APPENDIX: Percent Distribution (Standard Error) of Primary Racial Identification for Selected Detailed Race Groups.

[b] Primary racial affiliation based on survey results from Parker and Makuc (2001, Appendix).

[c] Parker and Makuc (2001) did not provide an estimate of the primary racial affiliation for "Other combinations, so we assume that it belongs to the "Other" category. Note that they did provide the primary racial affiliation for a fourth group "Black/AIAN:" 85.4% Black, 7.0% AIAN, and 7.6% Other. However, we do not have an estimate of the relative abundance of Black/AIAN in the general population, so we have dropped it from further consideration.

[d] As described in the text below, we calculated the percentages in this row from the percentages in the previous four rows for AIAN/White, API/White, Black/White, and Other combinations.

To estimate how to assign a single racial group for individuals that chose two or more racial groups, we used the results of Exhibit C-3 for the three main categories for which we an estimate of the primary racial affiliation: AIAN/White, API/White, and Black/White. To account for the 0.30 percent of the population in other combinations, we For each Census block, we assume that .89 / (.89+.30+.26+.30) = 50.8% of respondents in the Two or More category will fall into the AIAN / White category, and of these, 80.9% would primarily identify themselves as White if they were to choose a single racial category, 12.4% would primarily identify themselves as American Indian or Alaska Native, and 6.7% would primarily identify themselves as Other. Thus 0.508 * .809 = 41% of Two or More we will call White, 10% we identify as Native American, and 5% as Other.

We did not attempt to predict what respondents in the 'Other Combinations' category would have selected if they were to choose a single racial category, so we assume they are part

of the "Other" category. To estimate the number of individuals in each of the five races, we performed the following calculations:

$$NativeAmerican = NativeAmericanAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot AIAN\%_{AIAN/White} \right)$$

$$Asian = AsianAlone_{Pop} + PacificIslander/Hawaiian_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot API\%_{API/White} \right)$$

$$Black = BlackAlone_{Pop} + TwoorMore_{Pop} \cdot \left(\frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Black\%_{Black/White} \right)$$

$$White = White\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot White\%_{Black/White} \end{array} \right)$$

$$Other = Other\ Alone_{Pop} + Two\ \ or\ More_{Pop} \cdot \left(\begin{array}{l} \frac{AIAN/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{AIAN/White} + \\ \frac{API/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{API/White} + \\ \frac{Black/White_{Nat\%}}{MultipleRace_{Nat\%}} \cdot Other\%_{Black/White} + \\ \frac{Other\ \ Combinations_{Nat\%}}{MultipleRace_{Nat\%}} \end{array} \right)$$

This then reduces to:

$$Native\ American_{Pop} = Native\ American\ Alone_{Pop} + (0.063)Two\ or\ More_{Pop}$$

$$Asian_{Pop} = Asian\ Alone_{Pop} + Pacific\ Islander / Hawaiian_{Pop} + (0.059)Two\ or\ More_{Pop}$$

$$Black_{Pop} = Black\ Alone_{Pop} + (0.072)Two\ or\ More_{Pop}$$

$$White_{Pop} = White\ Alone_{Pop} + (0.530)Two\ or\ More_{Pop}$$

$$Other_{Pop} = White\ Alone_{Pop} + (0.276)Two\ or\ More_{Pop} \quad .$$

Estimating Population Levels in Alternative Age Groups

In calculating the population in age groups that may include a portion of one of the pre-specified demographic groups in Exhibit C-1, BenMAP assumes the population is uniformly distributed in the age group. For example, to calculate the number of children ages 3 through 12, BenMAP calculates:

$$age_{3-12} = \frac{1}{4} \cdot age_{1-4} + age_{5-9} + \frac{3}{5} \cdot age_{10-14} \quad .$$

<u>Estimating Population Levels in Non-Census Years</u>

To forecast population levels beyond 2000, BenMAP scales the 2000 Census-based estimate with the ratio of the county-level forecast for the future year of interest over the 2000 county-level population level. Woods & Poole (2001) provides the county-level population forecasts used to calculate the scaling ratios.

In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2010, CAMPS calculates:

$$age_{4-9,\,g,\,2010} = age_{4-9,\,g,\,2000} \cdot \frac{age_{4-9,\,county,\,2010}}{age_{4-9,\,county,\,2000}}$$

where the g^{th} population grid-cell is wholly located within a given county.

In the case, where the g[th] grid-cell includes "n" counties in its boundary, the situation is somewhat more complicated. BenMAP first estimates the fraction of individuals in a given age group (e.g., ages 4 to 9) that reside in the part of each county within the g[th] grid-cell. BenMAP calculates this fraction by simply dividing the population all ages of a given county within the g[th] grid-cell by the total population in the g[th] grid-cell:

$$fraction\ of\ \ age_{4-9,\ g\ in\ county_c} = \frac{age_{all,\ g\ in\ county_c}}{age_{all,\ g}}$$

Multiplying this fraction with the number of individuals ages 4 to 9 in the year 2000 gives an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the g[th] grid-cell in the year 2000:

$$age_{4-9,\ g\ in\ county_c,\ 2000} = age_{4-9,\ g,\ 2000} \cdot fraction\ age_{4-9,\ g\ in\ county_c}$$

To then forecast the population in 2010, we scale the 2000 estimate with the ratio of the county projection for 2010 to the county projection for 2000:

$$age_{4-9,\ g\ in\ county_c,\ 2010} = age_{4-9,\ g\ in\ county_c,\ 2000} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}} \quad .$$

Combining all these steps for "c" counties within the g[th] grid-cell, we forecast the population of persons ages 4 to 9 in the year 2010 as follows:

$$age_{4-9,\ g,\ 2010} = \sum_{c=1}^{n} age_{4-9,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}}$$

In the case where there are multiple age groups and multiple counties, BenMAP first calculates the forecasted population level for individual age groups, and then combines the forecasted age groups. In calculating the number of children ages 4 to 12, BenMAP calculates:

$$age_{4-9,\ g,\ 2010} = \sum_{c=1}^{n} age_{4-9,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{4-9,\ county_c,\ 2010}}{age_{4-9,\ county_c,\ 2000}}$$

$$age_{10-14,\ g,\ 2010} = \sum_{c=1}^{n} age_{10-14,\ g,\ 2000} \cdot \frac{total\ \ pop_{g\ in\ county_c}}{total\ \ pop_g} \cdot \frac{age_{10-14,\ county_c,\ 2010}}{age_{10-14,\ county_c,\ 2000}}$$

$$age_{4-12,\,g,\,2010} = age_{4-9,\,g,\,2010} + \frac{3}{5} \cdot age_{10-14,\,g,\,2010} \quad .$$

Since the Woods and Poole (2001) projections only extend through 2025, we used the existing projections and constant growth factors to provide additional projections. To estimate population levels beyond 2025, BenMAP linearly extrapolates from the final two years of data. For example, to forecast population in 2030, BenMAP calculates:

$$age_{4-9,\,2030} = age_{4-9,\,2025} + 5 \cdot \left(age_{4-9,\,2025} - age_{4-9,\,2024} \right) \quad .$$

Exhibit C-4 summarizes the forecasted age-stratified, state-level populations for 2020 and 2030. In addition, to provide a point of comparison, it includes population levels for year 2000.

Exhibit C-4. State-Level Population Estimates by Age Group

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
AL	1,126,337	2,740,965	579,798	1,212,702	3,057,540	909,065	1,284,828	3,069,518	1,195,921
AZ	1,371,099	3,091,693	667,839	1,832,737	4,351,254	1,309,943	2,117,749	4,708,843	1,812,966
AR	681,003	1,618,378	374,019	769,208	1,821,876	557,233	832,285	1,861,954	698,259
CA	9,254,212	21,021,768	3,595,658	10,105,474	25,308,061	5,717,329	10,895,067	26,084,324	7,681,464
CO	1,101,772	2,783,415	416,073	1,321,930	3,359,731	954,691	1,498,688	3,453,808	1,370,557
CT	839,051	2,096,330	470,183	821,773	2,110,504	588,222	845,210	1,991,695	728,973
DE	195,997	485,877	101,726	213,375	571,224	149,015	227,024	574,497	204,731
DC	120,659	381,502	69,898	95,389	337,146	103,401	93,833	305,565	123,728
FL	3,643,004	9,531,774	2,807,597	4,466,384	12,098,406	4,472,647	5,026,785	12,483,019	5,933,620
GA	2,176,259	5,224,918	785,275	2,600,100	6,296,967	1,328,722	2,851,139	6,620,751	1,778,194
ID	369,522	778,515	145,916	451,473	980,346	282,616	507,776	1,045,592	374,826
IL	3,247,904	7,671,362	1,500,025	3,286,653	8,148,579	2,069,429	3,425,612	7,962,757	2,669,430
IN	1,581,993	3,745,661	752,831	1,691,800	4,113,510	1,087,932	1,800,717	4,096,828	1,421,006
IA	737,415	1,752,696	436,213	734,433	1,820,333	593,034	766,374	1,750,358	755,945
KS	714,371	1,617,818	356,229	760,573	1,795,227	499,065	814,382	1,778,859	653,139
KY	998,042	2,538,933	504,793	1,077,101	2,762,379	801,696	1,154,120	2,750,564	1,052,988
LA	1,221,651	2,730,396	516,929	1,247,161	2,952,038	850,018	1,318,748	2,917,899	1,116,293
ME	299,691	791,830	183,402	284,880	852,466	289,399	297,507	807,626	394,873
MD	1,350,517	3,346,661	599,307	1,453,726	3,868,715	926,465	1,559,338	3,877,266	1,256,566
MA	1,508,818	3,980,116	860,162	1,533,618	4,071,543	1,144,857	1,604,543	3,871,104	1,469,089
MI	2,596,118	6,123,307	1,219,018	2,587,563	6,590,540	1,798,905	2,703,858	6,417,627	2,366,125
MN	1,285,100	3,040,113	594,266	1,413,120	3,525,458	948,035	1,547,597	3,524,435	1,298,319
MS	779,939	1,721,196	343,523	826,142	1,912,067	513,412	867,469	1,926,497	667,632
MO	1,428,853	3,410,978	755,379	1,526,846	3,830,433	1,062,471	1,631,969	3,798,554	1,404,065

Exhibit C-4. State-Level Population Estimates by Age Group (continued) *May 12, 2003*

	2000			2020			2030		
State	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***	***0-18***	***18-64***	***65+***
MT	228,916	552,330	120,949	234,129	612,736	241,971	258,376	604,179	322,696
NE	450,372	1,028,696	232,195	483,340	1,148,129	329,112	522,703	1,142,368	426,556
NV	510,633	1,268,694	218,929	757,488	1,921,749	477,249	904,840	2,150,250	654,589
NH	309,490	778,326	147,970	321,958	906,776	236,489	346,320	897,774	329,510
NJ	2,074,020	5,227,192	1,113,136	2,126,538	5,560,594	1,507,553	2,222,228	5,402,892	1,949,786
NM	506,558	1,100,262	212,225	583,389	1,372,898	412,394	644,935	1,420,580	572,907
NY	4,696,232	11,831,869	2,448,352	4,487,417	11,815,310	3,179,326	4,540,245	11,246,710	3,953,934
NC	1,977,387	5,102,877	969,048	2,399,345	6,081,807	1,588,246	2,646,039	6,337,401	2,079,430
ND	162,017	385,705	94,478	152,979	407,052	152,185	160,056	387,072	203,240
OH	2,889,207	6,956,175	1,507,757	2,894,902	7,316,549	2,031,922	3,021,197	7,084,923	2,597,112
OK	894,531	2,100,173	455,950	968,204	2,255,616	685,395	1,037,634	2,249,445	865,166
OR	846,233	2,136,988	438,177	1,028,841	2,593,792	824,166	1,161,142	2,731,400	1,065,084
PA	2,930,189	7,431,699	1,919,165	2,807,320	7,589,422	2,473,482	2,879,828	7,112,827	3,152,928
RI	252,438	643,479	152,402	248,650	664,840	185,270	253,697	626,436	236,588
SC	1,017,627	2,509,052	485,333	1,125,147	2,936,359	879,310	1,217,702	2,989,589	1,188,398
SD	202,496	444,217	108,131	209,379	498,258	159,468	222,092	487,168	215,460
TN	1,402,958	3,583,013	703,311	1,614,405	4,118,556	1,147,546	1,759,007	4,213,846	1,513,183
TX	5,891,741	12,887,542	2,072,532	7,108,830	15,994,222	3,802,007	7,929,363	16,840,990	5,236,651
UT	724,466	1,318,481	190,222	989,440	1,826,327	368,454	1,120,100	2,046,412	497,421
VT	147,949	383,368	77,510	137,590	414,505	138,315	144,053	397,442	190,941
VA	1,743,459	4,542,721	792,333	1,955,331	5,201,333	1,308,689	2,132,729	5,295,036	1,734,954
WA	1,511,831	3,720,140	662,148	1,785,937	4,644,371	1,174,213	2,006,978	4,857,761	1,581,410
WV	404,484	1,126,965	276,895	388,379	1,094,529	403,851	404,280	1,038,496	488,364
WI	1,369,215	3,291,907	702,553	1,413,693	3,680,062	1,070,942	1,511,982	3,612,218	1,450,806
WY	128,585	307,504	57,693	124,005	314,574	117,862	132,595	301,356	154,460

Woods & Poole Data

Woods & Poole (2001) developed county-level forecasts for each year from 2000 through 2025, for three racial groups "Black," "White," and "Other," and by age and by gender. For the Hispanic ethnic group, Woods and Poole developed forecasts just for the total population, and not by age and gender. As discussed in the section on population forecasts, BenMAP uses these forecasts to simply scale the 2000 Census block data, in order to estimate the population in the population grid-cells for any given year after 2000.

Aligning Woods & Poole FIPS Codes with BenMAP FIPS Codes

The county geographic boundaries used by Woods & Poole are somewhat more aggregated than the county definitions used in the 2000 Census (and BenMAP), and the FIPS codes used by Woods and Poole are not always the standard codes used in the Census. To make the Woods and Poole data consistent with the county definitions in BenMAP, we disaggregated the Woods and Poole data and changed some of the FIPS codes. Exhibit C-5 lists the discrepancies in the county definitions between Woods & Poole and those used in BenMAP.

To assign the population in the more aggregated Woods & Poole county definitions to the more disaggregated definitions used in BenMAP (and the U.S. Census), we used the total county population from the 2000 U.S. Census. We then assumed that the age and racial groups were distributed uniformly across the BenMAP counties contained within a Woods & Poole county definition. For example, in estimating the population of children ages 4-9 in county "c" contained within a more broadly defined Woods & Poole county, we would do the following:

$$age_{4-9,\,county_c} = age_{4-9,\,W\&P\ county} \cdot \frac{age_{all,\,county_c}}{age_{all,\,W\&P\ county}}$$

After this factor was applied, we rounded the estimates to the nearest integer so as to avoid having data with "partial people."

Exhibit C-5. Linkage Between Woods & Poole County Definitions and BenMAP County Definitions

Woods and Poole Counties (FIPS)	Counties in BenMAP (FIPS)
Northwest Arctic Borough, AK (02188)	Kobuk, AK (02140)
Remainder of Alaska, AK (02999)	Aleutian Islands, AK (02010), Aleutian Islands East Borough, AK (02013), Aleutian Islands West Census Area, AK (02016), Bethel Census Area, AK (02050), Denali Borough, AK (02068), Dillingham Census Area, AK (02070), Haines Borough, AK (02100), Kenai Peninsula Borough, AK (02122), Lake and Peninsula Borough, AK (02164), North Slope Borough, AK (02185), Prince of Wales-Outer Ketchikan, AK (02201), Sitka Borough, AK (02220), Skagway-Yukatat-Angoon, AK (02231), Skagway-Hoonah-Angoon Census Area, AK (02232), Southeast Fairbanks Census Area, AK (02240), Valdez-Cordova Census Area, AK (02261), Wrangell-Petersburg Census Area, AK (02280), Yakutat Borough, AK (02282), Yukon-Koyukuk, AK (02290)
Yuma + La Paz, AZ (04027)	La Paz, AZ (04012), Yuma, AZ (04027)
Miami-Dade, FL (12086)	Dade, FL (12025)
Maui + Kalawao, HI (15901)	Kalawao, HI (15005), Maui, HI (15009)
Fremont, ID (16043)	Fremont, ID (16043), Yellowstone Park, ID
Park, MT (30067)	Park, MT (30067), Yellowstone Park, MT (30113)
Valencia + Cibola, NM (35061)	Cibola, NM (35006), Valencia, NM (35061)
Halifax, VA (51083)	Halifax, VA (51083), South Boston City, VA (51780)
Albemarle + Charlottesville, VA (51901)	Albemarle, VA (51003), Charlottesville City, VA (51540)
Alleghany + Clifton Forge + Covington, VA (51903)	Alleghany, VA (51005), Clifton Forge City, VA (51560), Covington City, VA (51580)
Augusta + Staunton + Waynesboro, VA (51907)	Augusta, VA (51015), Staunton City, VA (51790), Waynesboro City, VA (51820)
Bedford + Bedford City, VA (51909)	Bedford, VA (51019), Bedford City, VA (51515)
Campbell + Lynchburg, VA (51911)	Campbell, VA (51031), Lynchburg City, VA (51680)
Carroll + Galax, VA (51913)	Carroll, VA (51035), Galax City, VA (51640)
Dinwiddie + Colonial Heights + Petersburg, VA (51918)	Dinwiddie, VA (51053), Colonial Heights City, VA (51570), Petersburg City, VA (51730)
Fairfax + Fairfax City + Falls Church City, VA (51919)	Fairfax, VA (51059), Fairfax City, VA (51600), Falls Church City, VA (51610)
Frederick + Winchester, VA (51921)	Frederick, VA (51069), Winchester City, VA (51840)
Greensville + Emporia, VA (51923)	Greensville, VA (51081), Emporia City, VA (51595)
Henry + Martinsville, VA (51929)	Henry, VA (51089), Martinsville City, VA (51690)
James City + Williamsburg, VA (51931)	James City County, VA (51095), Williamsburg City, VA (51830)

Woods and Poole Counties (FIPS)	Counties in BenMAP (FIPS)
Montgomery + Radford, VA (51933)	Montgomery, VA (51121), Radford City, VA (51750)
Pittsylvania + Danville, VA (51939)	Pittsylvania, VA (51143), Danville City, VA (51590)
Prince George + Hopewell, VA (51941)	Prince George, VA (51149), Hopewell City, VA (51670)
Prince William + Manassas + Manassas Park, VA (51942)	Prince William, VA (51153), Manassas City, VA (51683), Manassas Park City, VA (51685)
Roanoke + Salem, VA (51944)	Roanoke, VA (51161), Salem City, VA (51775)
Rockbridge + Buena Vista + Lexington, VA (51945)	Rockbridge, VA (51163), Buena Vista City, VA (51530), Lexington City, VA (51678)
Rockingham + Harrisonburg, VA (51947)	Rockingham, VA (51165), Harrisonburg City, VA (51660)
Southampton + Franklin, VA (51949)	Southampton, VA (51175), Franklin City, VA (51620)
Spotsylvania + Fredericksburg, VA (51951)	Spotsylvania, VA (51177), Fredericksburg City, VA (51630)
Washington + Bristol, VA (51953)	Washington, VA (51191), Bristol City, VA (51520)
Wise + Norton, VA (51955)	Wise, VA (51195), Norton City, VA (51720)
York + Poquoson, VA (51958)	York, VA (51199), Poquoson City, VA (51735)
Shawano (includes Menominee), WI (55901)	Menominee, WI (55078), Shawano, WI (55115)

Age, Gender, Race, and Ethnicity

We generated the same 38 age and gender categories developed from the 1990 and 2000 Census data. Since these projections are available for every year of age, it is a simple matter to sum the individual years to get the same age categories used by BenMAP.

However, the only racial categories available are "White," "Black," and "Other." Since we do not have an Asian or Native American group, or an Other group which is consistent with the definition used by the 1990 and 2000 Census data, we assume that projection data's Other category is representative of all 3 groups, and that they move together over time.

The county projections only forecast the Hispanic population of all ages, and does not have separate gender and age forecasts. Lacking further information, we use the ratio of future-year all age population to the year 2000 all age population when forecasting any particular age group of Hispanics. In effect, we assume for all forecast years the same distribution of age and gender as found in the 2000 Census.

Creating Growth Ratios from Absolute Population Values

For each year from 2000 through 2025 and for each of the 256 demographic groups listed in Exhibit C-1, BenMAP stores the ratio of the future-year to year 2000 county-level population projections. As described below, these ratios are used to forecast population levels in the population grid-cells used by BenMAP to health effects.

Note that there are a small number of cases were the 2000 county population for a specific demographic group is zero, so the ratio of any future year to the year 2000 data is undefined. In these relatively rare cases, we set the year 2000 ratio and all subsequent ratios to 1, assuming no growth.

There are an even smaller number of cases where a total population variable dwindles from some non-zero number to zero, creating ratios of zero. Variables which represent a subpopulation of the first variable may not be zero, however. In these cases, we set all subset population variables for that year to zero.

For instance, if a county only had one person in it for the year 2000 - a 79 year old black male - we set all variables (excluding total variables and BlackMale75to79) to a ratio of 1, because their 2000 values of 0 produce undefined ratios. If the man dies at age 82, the total black population variable for years 2003 and beyond is calculated as 0/1 = 0. Thus for each of those years where the total black population is listed as zero, we go back and set all black population variables to zero, to reflect the knowledge that the block is empty. For all variables except the BlackMale75to79 age group (already zero), 1 becomes 0.

APPENDIX D

PARTICULATE MATTER AND OZONE CONCENTRATION-RESPONSE FUNCTIONS

In this Appendix, we present the concentration-response (C-R) functions used to estimate adverse health effects related to PM and ozone. First, we discuss the concentration response functions for particulate matter, then we discuss then concentration response functions for ozone. Each sub-section has an Exhibit with a brief description of the C-R function and the underlying parameters. Following each Exhibit, we present a brief summary of each of the studies and any items that are unique to the study. Also, note that each citation in the text includes a numbered reference to a database that facilitates updating the citations.

Particulate Matter Concentration Response Functions

Long-term Mortality

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

Exhibit D-1 Concentration-Response (C-R) Functions for Particulate Matter and Long-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form	Notes
All Cause	$PM_{2.5}$	Krewski et al.	2000	63 cities	30+	All	All	None	Annual Avg	0.004626	0.001205	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	50 cities	30+	All	All	None	Annual Median	0.005348	0.001464	Log-linear	ACS reanalysis
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.010394	0.002902	Log-linear	ACS reanalysis; RE Ind Cities
All Cause	$PM_{2.5}$	Krewski et al.	2000	nationwide	30+	All	All	None	Annual Median	0.006058	0.003383	Log-linear	ACS reanalysis; RE Reg Adj
All Cause	$PM_{2.5}$	Krewski et al.	2000	6 cities	25+	All	All	None	Annual Avg	0.013272	0.004070	Log-linear	Six Cities reanalysis
All Cause	$PM_{2.5}$	Pope et al.	1995	50 cities	30+	All	All	None	Annual Median	0.006408	0.001509	Log-linear	
All Cause	$PM_{2.5}$	Dockery et al.	1993	6 cities	25+	All	All	None	Annual Avg	0.012425	0.004228	Log-linear	
All Cause	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.004018	0.001642	Log-linear	'79-'83 exposure
Cardiopulmonary	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.005733	0.002167	Log-linear	'79-'83 exposure
Lung Cancer	$PM_{2.5}$	Pope et al.	2002	61 cities	30+	All	All	None	Annual Avg	0.007881	0.003463	Log-linear	'79-'83 exposure
Infant	PM_{10}	Woodruff et al.	1997	86 cities	<1	All	All	None	Annual Avg	0.003922	0.001221	Logistic	

Mortality - Mean, All Cause [Krewski, 2000 #1805] - Reanalysis of Pope et al. [, 1995 #81]

The Krewski et al. [2000 #1805] reanalysis of Pope et al. [1995 #81] used a Cox proportional hazard model to estimate the impact of long-term PM exposure. The original investigation followed 295,223 individuals[1] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Krewski et al. [2000 #1805] independently estimated city-specific annual *mean* values from EPA's Inhalable Particle Monitoring Network (IPMN) for the same years (1979-1983). Krewski et al. [2000 #1805] followed Pope et al. [1995 #81, Table 2] and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths,[2] and found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Krewski et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. [1995 #81] is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. [1993 #20]. Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier and study population, in comparison to poorer minority populations. Krewski et al. [2000 #1805, Part II - Table 52] found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. In fact, much of the differences in magnitude of effect between the studies was made up when assessing risk across comparable levels of educational attainment.

Another source of downward bias is that intercity movement of cohort members was not considered in the original study and therefore could not be evaluated in the reanalysis. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

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[1]The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

[2]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.12) and 95% confidence interval (1.06-1.19) associated with a change in *annual mean* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 31].

Functional Form: Log-linear
Coefficient: 0.004626
Standard Error: 0.001205
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, All Cause [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] performed an analysis of Pope et al. [2000 #1805] using independently estimated city-specific annual *median* values as well. Fine particle estimates were obtained from EPA's Inhalable Particle Monitoring Network (IPMN) for the years 1979-1983 for the same 50 cities. Overall, the estimates showed good agreement with the median values used in the original investigation with one exception. The median fine particle concentration for Denver dropped from 16.1 to 7.8 $\mu g/m^3$, resulting in a larger range between the least and most polluted cities and a reduced relative risk. Since the original estimate could not be audited, Denver is included in the subsequent C-R function as there is no reason to believe that the monitoring data is invalid.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.06-1.22) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 31].

Functional Form: Log-linear
Coefficient: 0.005348
Standard Error: 0.001464
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, Random Effects with Regional Adjustment [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] also performed an analysis of Pope et al. [2000 #1805] using a random effects model to estimate a regionally-adjusted relative risk. The authors used an

indicator variable representing seven regions of the U.S. The regionally-adjusted estimate was comparable with the results from the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.16) and 95% confidence interval (0.99-1.37) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 46].

Functional Form: Log-linear
Coefficient: 0.006058
Standard Error: 0.003383
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality - Median, Random Effects with Independent Cities [Krewski, 2000 #1805] - Reanalysis of Pope et al. [1995 #81]

Krewski et al. [2000 #1805] also performed an analysis of Pope et al. [2000 #1805] using a random effects approach to estimate an independent cities model. This approach incorporates between-city variation into second-stage modeling weights, thereby avoiding the assumption of independent observations. However, potential regional patterns in mortality may be overlooked, because the approach assumes that city-specific mortality rates are statistically independent. The independent cities estimate is considerably larger than the standard Cox Proportional Hazards Model, which assumes that all observations are statistically independent.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.29) and 95% confidence interval (1.12-1.48) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ (based on the range from the original ACS study) [Krewski, 2000 #1805, Part II - Table 46].

Functional Form: Log-linear
Coefficient: 0.010394
Standard Error: 0.002902
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality [Krewski, 2000 #1805] - Reanalysis of Dockery et al. [1993 #20]

Krewski et al. [2000 #1805] performed a validation and replication analysis of Dockery et al. [1993 #20]. The originial investigators examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older; a higher proportion without a high school education), and a follow-up period nearly twice as long as that of Pope et al. [1995 #81]. Krewski et al. [2000 #1805, Part II - Table 52] found that educational status was a strong effect modifier of the PM - mortality relationship in both studies, with the strongest effect seen among the less educated. Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

After an audit of the air pollution data, demographic variables, and cohort selection process, Krewski et al. [2000 #1805] noted that a small portion of study participants were mistakenly censored early. The following C-R function is based on the risk estimate from the audited data, with the inclusion of those person-years mistakenly censored early.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.28) and 95% confidence interval (1.10-1.48) associated with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ to 29.6 $\mu g/m^3$ [Krewski et al.,2000 #1805, Part I - Table 19c].

Functional Form: Log-linear
Coefficient: 0.013272
Standard Error: 0.004070
Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older
Population: population of ages 25 and older

Mortality, All Cause [Pope, 1995 #81]

Pope et al. [1995 #81] used a Cox proportional hazard model to estimate the impact of long-term PM exposure. They followed 295,223 individuals[3] ages 30 and over in 50 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Pope et al. [1995 #81, Table 2] reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-

[3]The total study population was 552,138 in 151 cities, however, only 295,223 individuals resided in 50 cities with fine particle data.

519), and "all other" deaths,[4] and found that median $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Pope et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. [1995 #81] is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. [1993 #20]. Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier study population, in comparison to poorer minority populations. Another source of downward bias is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.17) and 95% confidence interval (1.09-1.26) associated with a change in *annual median* $PM_{2.5}$ exposure of 24.5 $\mu g/m^3$ [Pope, 1995 #81, Table 2].

Functional Form: Log-linear
Coefficient: 0.006408
Standard Error: 0.001509
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality, All Cause [Dockery, 1993 #20]

Dockery et al. [1993 #20] examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved

[4]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. [1995 #81]. Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

Single Pollutant Model

The coefficient and standard error are estimated from the relative risk (1.26) and 95% confidence interval associated (1.08-1.47) with a change in *annual mean* $PM_{2.5}$ exposure of 18.6 $\mu g/m^3$ [Dockery, 1993 #20, Tables 1 and 5].

Functional Form: Log-linear
Coefficient: 0.012425
Standard Error: 0.004228
Incidence Rate: county-specific annual all cause mortality rate per person ages 25 and older
Population: population of ages 25 and older

Mortality, All Cause [Pope, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

The Pope et al. [2002 #2240] analysis is a longitudinal cohort tracking study that uses the same American Cancer Society (ACS) cohort as the original Pope et al. [1995 #81] study, and the Krewski et al. [2000 #1805] reanalysis. Pope et al. [2002 #2240] analyzed survival data for the cohort from 1982 through 1998, 9 years longer than the original Pope study. Pope et al. [2002 #2240] also obtained $PM_{2.5}$ data in 116 metropolitan areas collected in 1999, and the first three quarters of 2000. This is more metropolitan areas with $PM_{2.5}$ data than was available in the Krewski reanalysis (61 areas), or the original Pope study (50 areas), providing a larger size cohort.

They used a Cox proportional hazard model to estimate the impact of long-term PM exposure using three alternative measures of $PM_{2.5}$ exposure; metropolitan area-wide annual mean PM levels from the beginning of tracking period (’79-’83 PM data, conducted for 61 metropolitan areas with 359,000 individuals), annual mean PM from the end of the tracking period (’99-’00, for 116 areas with 500,000 individuals), and the average annual mean PM levels of the two periods (for 51 metropolitan areas, with 319,000 individuals). PM levels were lower in ’99-00 than in ’79 - ’83 in most cities, with the largest improvements occurring in cities with the highest original levels.

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, Table 2] and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and “all other” deaths.[5] Like the

[5]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category “all other” deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

Pope et al. [2002 #2240] obtained ambient data on gaseous pollutants routinely monitored by EPA during the 1982-1998 observation period, including SO_2, NO_2, CO, and ozone. They did not find significant relationships between NO_2, CO, and ozone and premature mortality, but there were significant relationships between SO_2, and all-cause, cardiopulmonary, lung cancer and "all other" mortality.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.041) and 95% confidence interval (1.008-1.075) associated with a change in *annual mean* exposure of 10.0 $\mu g/m^3$. Pope et al. [2002 #2240, Table 2].[6]

Functional Form: Log-linear
Coefficient: 0.004018
Standard Error: 0.001642
Incidence Rate: county-specific annual all cause mortality rate per person ages 30 and older
Population: population of ages 30 and older

Mortality, Cardiopulmonary [Pope, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, Table 2] and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[7] Like the earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.059) and 95% confidence interval (1.015-1.105) associated with a change in

[6]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

[7]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

annual mean exposure of 10.0 μg/m^3. Pope et al. [2002 #2240, Table 2].[8]

Functional Form: Log-linear
Coefficient: 0.005733
Standard Error: 0.002167
Incidence Rate: county-specific annual cardiopulmonary mortality rate (ICD codes 401-440, 460-519) per person ages 30 and older
Population: population of ages 30 and older

Mortality, Lung Cancer [Pope, 2002 #2240] - Based on ACS Cohort: Mean $PM_{2.5}$

Pope et al. [2002 #2240] followed Krewski et al. [2000 #1805] and Pope et al. [1995 #81, Table 2] and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths.[9] Like the earlier studies, Pope et al. [2002 #2240] found that mean $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. [2002 #2240] found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with "all other" deaths.

'79-'83 Exposure

The coefficient and standard error for $PM_{2.5}$ using the '79-'83 PM data are estimated from the relative risk (1.082) and 95% confidence interval (1.011-1.158) associated with a change in *annual mean* exposure of 10.0 μg/m^3. Pope et al. [2002 #2240, Table 2].[10]

Functional Form: Log-linear
Coefficient: 0.007881
Standard Error: 0.003463
Incidence Rate: county-specific annual lung cancer mortality rate (ICD code 162) per person ages 30 and older
Population: population of ages 30 and older

[8]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

[9]All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

[10]Note that we used an unpublished, final version of the paper that presents the relative risks with one more significant digit than that found in the published version. We chose to use this extra information to increase the precision of our estimates.

Infant Mortality [Woodruff, 1997 #210]

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. [1997 #210] found a significant link between PM_{10} exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM_{10} exposure was significant for all-cause mortality. PM_{10} was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

In addition to the work by Woodruff et al., work in Mexico City [Loomis, 1999 #756], the Czech Republic [Bobak, 1992 #1130], Sao Paulo [Saldiva, 1994 #167; Pereira, 1998 #164], and Beijing [Wang, 1997 #1132] provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality.

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. [1995 #81], because the Pope function covers only the population over 30 years old.[11] However, the EPA Science Advisory Board recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies [U.S. EPA, 1999 #930, p. 12]. The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, and are not included in the primary analysis.

Single Pollutant Model

The coefficient and standard error are based on the odds ratio (1.04) and 95% confidence interval (1.02-1.07) associated with a 10 $\mu g/m^3$ change in PM_{10} [Woodruff, 1997 #210, Table 3].

Functional Form: Logistic
Coefficient: 0.003922
Standard Error: 0.001221
Incidence Rate: county-specific annual postneonatal[12] infant deaths per infant under the age of one
Population: population of infants under one year old

[11] Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages.

[12] Post-neonatal refers to infants that are 28 days to 364 days old.

Exhibit D-2 Concentration-Response (C-R) Functions for Particulate Matter and Short-Term Mortality

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.001433	0.00013	Log-linear	
Non-Accidental	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.002835	--	Log-linear	Lag Adjusted[2]
Chronic Lung	$PM_{2.5}$	Schwartz et al.	1996	6 cities	All	All	All	None	24-hr avg	0.006423	--	Log-linear	Lag Adjusted[2]

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.
2. Refer to the study summaries below for a discussion of the lag adjustment used for these functions.

Short-term Mortality

Short-term mortality studies are those that typically link daily air pollution levels with daily changes in mortality rates.

Short-Term Mortality, Non-Accidental [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[13] Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error [Abt Associates Inc., 1996 #238, p. 65].

Single Pollutant Model

Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient [Abt Associates Inc., 1996 #238, Exhibit 7.2] and its standard error [Abt Associates Inc., 1996 #238, Exhibit 7.2].

Functional Form: Log-linear
Coefficient: 0.001433
Standard Error: 0.000129
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental - Lag Adjusted [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[14] Abt Associates Inc. [1996 #239, p. 52] used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in

[13]Schwartz et al. [1996 #98, p. 929] defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

[14]Schwartz et al. [1996 #98, p. 929] defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error [Abt Associates Inc., 1996 #238, p. 65]. In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure [Schwartz, 2000 #1550; Samet, 2000 #1810]. These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz [2000 #1550] examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. [1996 #98] for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The distributed lag model coefficient is estimated by applying the distributed lag adjustment factor of 1.9784 to the pooled $PM_{2.5}$ coefficient (0.001433) estimated by Abt Associates Inc. [1996 #238, Exhibit 7.2] from the six $PM_{2.5}$ relative risks reported by Schwartz et al. [1996 #98].[15]

Functional Form: Log-linear
Coefficient: 0.002835
Incidence Rate: county-specific annual daily non-accidental mortality rate (ICD codes <800) per person

[15] The distributed lag adjustment C-R function is only run for the point estimate. The standard error of this coefficient has not been estimated.

Population: population of all ages

Short-Term Mortality, Chronic Lung Disease - Lag Adjusted [Schwartz, 1996 #98]

Schwartz et al. [1996 #98] evaluated the relationship between daily $PM_{2.5}$ levels and short-term mortality in six U.S. cities. Schwartz pooled results across the six cities and found statistically significant associations between daily $PM_{2.5}$ levels and non-accidental mortality (ICD codes <800), along with mortality for ischemic heart disease (ICD codes 410-414), COPD (ICD codes 490-496), and pneumonia (ICD codes 480-486). A smaller association was found for PM_{10} and no significant associations were reported for $PM_{10-2.5}$. The C-R function for chronic lung disease mortality is based on the results of a single pollutant model using a two-day average of $PM_{2.5}$ [Schwartz et al., 1996 #98, Table 7]. In order to estimate the impact of daily $PM_{2.5}$ levels on daily mortality if a distributed lag model had been fit, the $PM_{2.5}$ coefficient is adjusted as described below.

Recent studies have found that an increase in PM levels on a given day can elevate mortality for several days following the exposure [Schwartz, 2000 #1550; Samet, 2000 #1810]. These studies have reported the results of distributed lag models for the relationship between PM_{10} and daily mortality. Schwartz [2000 #1550] examined the relationship between PM_{10} and daily mortality and reported results both for a single day lag model and an unconstrained distributed lag model. The unconstrained distributed lag model coefficient estimate is 0.0012818 and the single-lag model coefficient estimate is 0.0006479. A distributed lag adjustment factor can be constructed as the ratio of the estimated coefficient from the unconstrained distributed lag model to the estimated coefficient from the single-lag model reported in Schwartz (2000). The ratio of these estimates is 1.9784. In order to estimate the full impact of daily PM levels on daily mortality, we applied this ratio to the coefficient obtained from Schwartz et al. [1996 #98] for the association between $PM_{2.5}$ and daily mortality.

In applying the ratio derived from a PM_{10} study to $PM_{2.5}$, we assume that the same relationship between the distributed lag and single day estimates would hold for $PM_{2.5}$. Effect estimates for the PM_{10}-daily mortality relationship tend to be lower in magnitude than for $PM_{2.5}$, because fine particles are believed to be more closely associated with mortality than the coarse fraction of PM. If most of the increase in mortality is expected to be associated with the fine fraction of PM_{10}, then it is reasonable to assume that the same proportional increase in risk would be observed if a distributed lag model were applied to the $PM_{2.5}$ data.

Single Pollutant Model

The $PM_{2.5}$ coefficient is based on a reported 3.3% increase in COPD mortality associated with a 10 $\mu g/m^3$ change in two-day average $PM_{2.5}$ levels [Schwartz, 1996 #98, Table 7]. This coefficient (0.003247) is then multiplied by the distributed lag adjustment factor of 1.9784 to estimate a distributed lag model coefficient.

Functional Form: Log-linear
Coefficient: 0.006423
Incidence Rate: county-specific annual daily chronic lung disease mortality rate (ICD codes 490-496)
Population: population of all ages

Exhibit D-3 Concentration-Response (C-R) Functions for Particulate Matter and Chronic Illness

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time	Beta	Std Error	Functional Form
Chronic Bronchitis	$PM_{2.5}$	Abbey et al.	1995	SF, SD, South Coast Air Basin	27+	All	All	None	Annual Avg	0.0137	0.00680	Logistic
Chronic Bronchitis	PM_{10}	Schwartz	1993	53 cities	30+	All	All	None	Annual Avg	0.0123	0.00434	Logistic

Chronic Illness

Schwartz [1993 #240] and Abbey et al. [1993 #245;, 1995 #452] provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. [1999 #1153] provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning [Abbey, 1998 #249; AckermannLiebrich, 1997 #117; Detels, 1991 #345].[16]

Chronic Bronchitis [Abbey, 1995 #452, California]

Abbey et al. [1995 #452] examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined. The C-R function is based on the results of the single pollutant model presented in Table 2.

Single Pollutant Model

The estimated coefficient (0.0137) is presented for a one $\mu g/m^3$ change in $PM_{2.5}$ [Abbey, 1995 #452, Table 2]. The standard error is calculated from the reported relative risk (1.81) and 95% confidence interval (0.98-3.25) for a 45 $\mu g/m^3$ change in $PM_{2.5}$ [Abbey, 1995 #452, Table 2].

Functional Form: Logistic
Coefficient: 0.0137
Standard Error: 0.00680
Incidence Rate: annual bronchitis incidence rate per person [Abbey, 1993 #245, Table 3] = 0.00378

[16] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

Population: population of ages 27 and older[17] without chronic bronchitis = 95.57%[18] of population 27+

Chronic Bronchitis [Schwartz, 1993 #240]

Schwartz [1993 #240] examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz [1993 #240, Table 3] reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[19], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea. The C-R function for PM_{10} is estimated from the results of the single pollutant model reported for TSP.

Single Pollutant Model

The estimated coefficient is based on the odds ratio (1.07) associated with 10 $\mu g/m^3$ change in TSP [Schwartz, 1993 #240, p. 9]. Assuming that PM_{10} is 55 percent of TSP[20] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient by 0.55. The standard error for the coefficient is calculated from the 95% confidence interval for the odds ratio (1.02 to 1.12) [Schwartz, 1993 #240, p. 9].

Schwartz [1993 #240] examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in

[17] Using the same data set, Abbey et al. [1995 #247, p. 140] reported that the respondents in 1977 ranged in age from 27 to 95.

[18] The American Lung Association [2002 #2357, Table 4] reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% [American Lung Association, 2002 #2357, Table 4].

[19] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

[20] The conversion of TSP to PM_{10} is from ESEERCO [1994 #323, p. V-5], who cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate. The scaling factor used in the C-R function is the ratio of chronic bronchitis incidence rate (estimated from Abbey et al. [1993 #245]) to chronic bronchitis prevalence rate (estimated from American Lung Association [2002 #2357, Table 4]).

Functional Form: Logistic
Coefficient: 0.0123
Standard Error: 0.00434
Incidence Rate: annual chronic bronchitis prevalence rate per person [American Lung Association, 2002 #2357, Table 4] = 0.0443
Population: population of ages 30 and older without chronic bronchitis = 95.57%[21] of population 30+
Adjustment Factor: ratio of chronic bronchitis incidence to chronic bronchitis prevalence = 0.00378/0.0443 = 0.085 [Abbey, 1993 #245, Table 3; American Lung Association, 2002 #2357, Table 4]

[21] The American Lung Association [2002 #2357, Table 4] reports a chronic bronchitis prevalence rate for ages 18 and over of 4.43% [American Lung Association, 2002 #2357, Table 4].

Exhibit D-4 Concentration-Response (C-R) Functions for Particulate Matter and Hospital Admissions

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Sheppard et al.	1999	Seattle, WA	<65	All	All	CO	24-hr avg	0.002505	0.001045	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001089	0.002420	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0008	0.001000	Log-linear
Chronic Lung Disease	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0020	0.000909	Log-linear
Chronic Lung Disease (less Asthma)	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002839	0.001351	Log-linear
Pneumonia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004480	0.001918	Log-linear
Pneumonia	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.002049	0.000570	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	65+	All	All	CO	24-hr avg	0.0005	0.000556	Log-linear
All Cardiovascular	$PM_{2.5}$	Moolgavkar	2000	Los Angeles, CA	18-64	All	All	CO	24-hr avg	0.0009	0.000500	Log-linear
All Cardiovascular	PM_{10}	Samet et al.	2000	14 cities	65+	All	All	None	24-hr avg	0.001183	0.000111	Log-linear
Dysrhythmia	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.002138	0.002525	Log-linear
Heart Failure	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.004668	0.001650	Log-linear
Ischemic Heart Disease	$PM_{2.5}$	Lippmann et al.	2000	Detroit, MI	65+	All	All	O_3	24-hr avg	0.001116	0.001339	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Hospitalizations

Hospital Admissions for Asthma [Sheppard, 1999 #792, Seattle]

Sheppard et al. [1999 #792] studied the relation between air pollution in Seattle and nonelderly (<65) hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM10_{10\text{-}2.5}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects.[22] They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, $PM_{10\text{-}2.5}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting co-pollutant models were found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. For the remaining pollutants, the best fitting models included $PM_{2.5}$ and CO. Results for other co-pollutant models were not reported. The $PM_{2.5}$ C-R function is based on the multipollutant model.

Multipollutant Model ($PM_{2.5}$ and CO)

The coefficient and standard error for the co-pollutant model with CO are calculated from a relative risk of 1.03 (95% CI 1.01-1.06) for an 11.8 $\mu g/m^3$ increase[23] in $PM_{2.5}$ [Sheppard, 1999 #792, p. 28].

Functional Form: Log-linear
Coefficient: 0.002505
Standard Error: 0.001045
Incidence Rate: region-specific daily hospital admission rate for asthma admissions per person <65 (ICD code 493)
Population: population of ages 65 and under

Hospital Admissions for Chronic Lung Disease [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10\text{-}2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-

[22] $PM_{2.5}$ levels were estimated from light scattering data.

[23] The reported IQR change in the abstract and text is smaller than reported in Table 3. We assume the change reported in the abstract and text to be correct because greater number of significant figures are reported.

486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.040 (95% CI 0.877-1.234) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 26].

Functional Form: Log-linear
Coefficient: 0.001089
Standard Error: 0.002420
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease [Moolgavkar, 2000 #2152]

Moolgavkar [2000 #2152] examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM_{10}. In Los Angeles, marginally significant associations were observed for $PM_{2.5}$, which were generally lower than for the gases. In co-pollutant models with CO, the $PM_{2.5}$ effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

The $PM_{2.5}$ C-R functions are based on the co-pollutant models ($PM_{2.5}$ and CO) reported for the 20-64 and 65+ age groups. Since the true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.8[24] and t-statistic of 0.8 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model [Moolgavkar, 2000 #2152, Table 3, p. 80].

Functional Form: Log-linear
Coefficient: 0.0008
Standard Error: 0.001000
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

Ages 18 to 64[25]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0[26] and t-statistic of 2.2 for a 10 μg/m^3 increase in $PM_{2.5}$ in the two-day lag model [Moolgavkar, 2000 #2152, Table 4, p. 81].

Functional Form: Log-linear
Coefficient: 0.0020
Standard Error: 0.000909
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD codes 490-492, 494-496)[27]
Population: population of ages 18 to 64

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[24] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.8 would result in a relative risk of 1.008 and coefficient of 0.000797. The "estimated" percent change, as reported by Moolgavkar, of 0.8 results in a relative risk of 1.008032 and coefficient of 0.0008.

[25] Although Moolgavkar [2000 #2152] reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[26] In a log-linear model, the percent change is equal to (RR - 1) * 100. In this study, Moolgavkar defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.0 would result in a relative risk of 1.020 and coefficient of 0.001980. The "estimated" percent change, as reported by Moolgavkar, of 2.0 results in a relative risk of 1.020201 and coefficient of 0.002.

[27] Moolgavkar [2000 #2152] reports results for ICD codes 490-496. In order to avoid double counting non-elderly asthma hospitalizations (ICD code 493) with Sheppard et al. [1999 #792] in a total benefits estimation, we have excluded ICD code 493 from the baseline incidence rate used in this function.

Hospital Admissions for Chronic Lung Disease (less Asthma) [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[28] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[29] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[30] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

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[28]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[29] Joel Schwartz (co-author), personal communication.

[30] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.88 percent increase (RR = 1.0288) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][31]. The standard error is estimated from the reported lower (0.19 percent) and upper bounds (5.64 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.002839
Standard Error: 0.001351
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the results of the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.175 (95% CI 1.026-1.345) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 26].

Functional Form: Log-linear
Coefficient: 0.004480
Standard Error: 0.001918

[31] The random effects estimate of the unconstrained distributed lag model was chosen for COPD admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

Incidence Rate: region-specific daily hospital admission rate for pneumonia admissions per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[32] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[33] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[34] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

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[32]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[33] Joel Schwartz (co-author), personal communication.

[34] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 2.07 percent increase (RR = 1.0207) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][35]. The standard error is estimated from the reported lower (0.94 percent) and upper bounds (3.22 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.002049
Standard Error: 0.000570
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for All Cardiovascular [Moolgavkar, 2000 #2029, Los Angeles]

Moolgavkar [2000 #2029] examined the association between air pollution and cardiovascular hospital admissions (ICD 390-448) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM_{10} or $PM_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $PM_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $PM_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $PM_{2.5}$ effect dropped out in co-pollutant models with CO. The $PM_{2.5}$ C-R functions are based on co-pollutant ($PM_{2.5}$ and CO) models.

Ages 65 and older

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.5[36] and t-statistic of 0.9 for a 10 μg/m^3 increase in $PM_{2.5}$ in the one day lag

[35] The random effects estimate of the unconstrained distributed lag model was chosen for pneumonia admissions since the chi-square test of heterogeneity was significant (see Samet et al., 2000, Part II - Table 15).

[36] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar [2000 #2152], he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.5 would result in a relative risk of 1.005 and coefficient of 0.000499. Assuming that the 0.5 is the "estimated" percent change described previously would result in a relative risk of 1.005013 and coefficient of 0.0005. We assume that the "estimated" percent changes reported in this study reflect the definition from [Moolgavkar, 2000 #2152].

model [Moolgavkar, 2000 #2029, Table 3, p. 1202].

Functional Form: Log-linear
Coefficient: 0.0005
Standard Error: 0.000556
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person 65+ (ICD codes 390-409, 411-459)[37]
Population: population of ages 65 and older

Ages 18 to 64[38]

Multipollutant Model ($PM_{2.5}$ and CO)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 0.9[39] and t-statistic of 1.8 for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ in the zero lag model [Moolgavkar, 2000 #2029, Table 4, p. 1203].

Functional Form: Log-linear
Coefficient: 0.0009
Standard Error: 0.000500
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD codes 390-409, 411-459)[40]
Population: population of ages 18 to 64

[37] Moolgavkar [2000 #2029] reports results for ICD codes 390-429. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

[38] Although Moolgavkar [2000 #2029] reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64.

[39] In a log-linear model, the percent change is equal to (RR - 1) * 100. In a similar hospitalization study by Moolgavkar [2000 #2152], he defines and reports the "estimated" percent change as (log RR * 100). Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 0.9 would result in a relative risk of 1.009 and coefficient of 0.000896. Assuming that the 0.9 is the "estimated" percent change described previously would result in a relative risk of 1.009041 and coefficient of 0.0009. We assume that the "estimated" percent changes reported in this study reflect the definition from [Moolgavkar, 2000 #2152].

[40] Moolgavkar [2000 #2029] reports results that include ICD code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Hospital Admissions for All Cardiovascular [Samet, 2000 #1810, 14 Cities]

Samet et al. [2000 #1810] examined the relationship between air pollution and hospital admissions for individuals of ages 65 and over in 14 cities across the country.[41] Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements. Hospital admissions were obtained from the Health Care Financing Administration (HCFA) for the years 1992 and 1993. Poisson regression was used in the analysis with unconstrained distributed lag models to examine the possibility that air pollution affects hospital admissions on not only the same day but on later days as well. The use of unconstrained distributed lags has the advantages of (1) not inappropriately biasing down risk estimates due to tight constraints (e.g. one day lag) and (2) not leaving the often arbitrary choice of lag period to the investigator's discretion. The C-R functions are based on the pooled estimate across all 14 cities, using the unconstrained distributed lag model and fixed or random effects estimates, depending on the results of a test for heterogeneity.

For this analysis, the unadjusted, base models for the effect of PM_{10} on hospital admissions were used. The authors performed a second-stage regression to estimate the impact of SO_2 and O_3 on the PM_{10} - hospitalization effect. For ozone, the PM_{10} effect in each city was regressed on the correlation between ozone and particulate matter (the slope of a PM_{10} vs. O_3 regression) in that city. The fitted line for this regression will have a slope of zero if there is no relationship, meaning that the effect of PM_{10} is not dependent on the correlation between PM_{10} and O_3. The adjusted point estimate was obtained by determining the PM_{10} effect when the correlation between the pollutants is zero (i.e. the y-intercept of the fitted line). The effect of O_3 adjustment on the PM_{10} - hospitalization relationship appeared to be minimal except for the case of COPD. In this case, adjustment increased the point estimate of the independent particulate matter effect. The variance of this estimate, however, was quite large and the confidence intervals of the adjusted and unadjusted estimates overlapped substantially. For these reasons, there appeared to be little impact of O_3 adjustment.[42] Furthermore, the statistical power and robustness of this second-stage approach to co-pollutant adjustment are in question because of the small number of observations used in the regression (14 cities) and the potential for one or two observations to dramatically impact the results.[43] Finally, for the case of COPD, adjustment led to an increased PM_{10} independent effect, meaning that if the adjustment is valid, the impact on hospital admissions will be underestimated rather than overestimated.

[41]The cities under investigation include: Birmingham, Boulder, Canton, Chicago, Colorado Springs, Detroit, Minneapolis/St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown.

[42] Joel Schwartz (co-author), personal communication.

[43] Commentary from the Health Review Committee (Samet et al., 2000, p.77) states that "[w]hile the approach used in the morbidity analysis is novel...the question arises as to the adequacy of statistical power for performing these analyses."

Single Pollutant Model

The estimated PM_{10} coefficient is based on a 1.19 percent increase (RR = 1.0119) in admissions due to a PM_{10} change of 10.0 μg/m^3 [Samet, 2000 #1810, Part II - Table 14][44]. The standard error is estimated from the reported lower (0.97 percent) and upper bounds (1.41 percent) of the percent increase [Samet, 2000 #1810, Part II - Table 14].

Functional Form: Log-linear
Coefficient: 0.001183
Standard Error: 0.000111
Incidence Rate: region-specific daily hospital admission rate for all cardiovascular disease per person 65+ (ICD codes 390-459)
Population: population of ages 65 and older

Hospital Admissions for Dysrhythmia [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.080 (95% CI 0.904-1.291) for a 36 μg/m^3 increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.002138

[44] The fixed effects estimate of the unconstrained distributed lag model was chosen for CVD admissions since the chi-square test of heterogeneity was non-significant (see Samet et al., 2000, Part II - Table 15).

Standard Error: 0.002525
Incidence Rate: region-specific daily hospital admission rate for dysrhythmia admissions per person 65+ (ICD code 427)
Population: population of ages 65 and older

Hospital Admissions for Heart Failure [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.183 (95% CI 1.053-1.329) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.004668
Standard Error: 0.001650
Incidence Rate: region-specific daily hospital admission rate for heart failure admissions per person 65+ (ICD code 428)
Population: population of ages 65 and older

Hospital Admissions for Ischemic Heart Disease [Lippmann, 2000 #2328, Detroit]

Lippmann et al. [2000 #2328] studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ in a Poisson regression model with generalized

additive models (GAM) to adjust for nonlinear relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), $PM_{10-2.5}$ and PM_{10} were significant for ischemic heart disease (ICD code 410-414), and $PM_{2.5}$ and PM_{10} were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO_2, NO_2, or CO, the results were generally comparable. The $PM_{2.5}$ C-R function is based on the co-pollutant model with ozone.

Multipollutant Model ($PM_{2.5}$ and ozone)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.041 (95% CI 0.947-1.144) for a 36 $\mu g/m^3$ increase in $PM_{2.5}$ [Lippmann, 2000 #2328, Table 14, p. 27].

Functional Form: Log-linear
Coefficient: 0.001116
Standard Error: 0.001339
Incidence Rate: region-specific daily hospital admission rate for ischemic heart disease admissions per person 65+ (ICD codes 411-414)[45]
Population: population of ages 65 and older

[45] Lippmann et al. [2000 #2328] reports results for ICD codes 410-414. In the benefits analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. [2001 #2157]. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Exhibit D-5 Concentration-Response (C-R) Functions for Particulate Matter and Emergency Room Visits

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Asthma	$PM_{2.5}$	Norris et al.	1999	Seattle, WA	<18	All	All	NO_2, SO_2	24-hr avg	0.016527	0.004139	Log-linear
Asthma	PM_{10}	Schwartz et al.	1993	Seattle, WA	<65	All	All	None	24-hr avg	0.00367	0.00126	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Emergency Room Visits

Emergency Room Visits for Asthma [Norris, 1999 #1263]

Norris et al. [1999 #1263] examined the relation between air pollution in Seattle and childhood (<18) hospital admissions for asthma from 1995 to 1996. The authors used air quality data for PM_{10}, light scattering (used to estimate fine PM), CO, SO_2, NO_2, and O_3 in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to $PM_{2.5}$), PM_{10}, and CO. No association was found between O_3, NO_2, or SO_2 and asthma ER visits, although O_3 had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM_{10}) and NO_2 and SO_2, the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits. The $PM_{2.5}$ C-R function is on the multipollutant model reported.

Multipollutant Model ($PM_{2.5}$, NO_2, and SO_2)

In a model with NO_2 and SO_2, the $PM_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ [Norris, 1999 #1263, p. 491].
Functional Form: Log-linear
Coefficient: 0.016527
Standard Error: 0.004139
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <18 (ICD code 493)
Population: population of ages under 18

Emergency Room Visits for Asthma [Schwartz, 1993 #860, Seattle]

Schwartz et al. [1993 #680] examined the relationship between air quality and emergency room visits for asthma (ICD codes 493,493.01,493.10,493.90,493.91) in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

Single Pollutant Model

The PM_{10} coefficient and standard error are reported by Schwartz et al. [1993 #860, p. 829] for a unit $\mu g/m^3$ increase in four-day average PM_{10} levels.

Functional Form: Log-linear
Coefficient: 0.00367

Standard Error: 0.00126
Incidence Rate: region-specific daily emergency room rate for asthma admissions per person <65 (ICD code 493)
Population: population of ages under 65

Exhibit D-6 Concentration-Response (C-R) Functions for Particulate Matter and Acute Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form
Acute Bronchitis	$PM_{2.5}$	Dockery et al.	1996	24 communities	8-12	All	All	None	Annual Avg	0.027212	0.017096	Logistic
Acute Myocardial Infarction, Nonfatal	$PM_{2.5}$	Peters et al.	2001	Boston, MA	18+	All	All	None	24-hr avg	0.024121	0.009285	Logistic
Any of 19 Respiratory Symptoms	PM_{10}	Krupnick	1990	Los Angeles, CA	18-64	All	All	O_3	24-hr avg	0.000461	0.000239	Linear
Lower Respiratory Symptoms	$PM_{2.5}$	Schwartz and Neas	2000	6 cities	7-14	All	All	$PM_{10-2.5}$	24-hr avg	0.016976	0.006680	Logistic
Minor Restricted Activity Days	$PM_{2.5}$	Ostro and Rothschild	1989	nationwide	18-64	All	All	O_3	24-hr avg	0.00741	0.00070	Log-linear
Work Loss Days	$PM_{2.5}$	Ostro	1987	nationwide	18-64	All	All	None	24-hr avg	0.0046	0.00036	Log-linear

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Acute Effects

Acute Bronchitis [Dockery, 1996 #25]

Dockery et al. [1996 #25] examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[46] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that thestudy examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes. Earlier work, by Dockery et al. [1989 #327], based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al. [1996 #25] study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis.

Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" [Dockery, 1996 #25, p. 501]. It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis. The C-R function is based on results of the single pollutant model reported in Table 1.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (1.50) and 95% confidence interval (0.91-2.47) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$) versus the least polluted city ($PM_{2.1}$ = 5.8 $\mu g/m^3$) [Dockery, 1996 #25, Tables 1 and 4]. The original study used $PM_{2.1}$, however, we use the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

Functional Form: Logistic
Coefficient: 0.027212
Standard Error: 0.017096
Incidence Rate: annual bronchitis incidence rate per person = 0.043 [American Lung Association, 2002 #2354, Table 11]
Population: population of ages 8-12

[46] The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable.

Acute Myocardial Infarction (Heart Attacks), Nonfatal [Peters, 2001 #2157]

Peters et al. [2001 #2157] studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$,"black carbon", O_3, CO, NO_2, and SO_2 in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and $PM_{2.5}$ levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour $PM_{2.5}$ concentrations before onset. Significant associations were observed for PM_{10} as well. None of the other particle measures or gaseous pollutants were significantly associated with acute myocardial infarction for the two hour or twenty-four hour period before onset.

The patient population for this study was selected from health centers across the United States. The mean age of participants was 62 years old, with 21% of the study population under the age of 50. In order to capture the full magnitude of heart attack occurrence potentially associated with air pollution and because age was not listed as an inclusion criteria for sample selection, we apply an age range of 18 and over in the C-R function. According to the National Hospital Discharge Survey, there were no hospitalizations for heart attacks among children <15 years of age in 1999 and only 5.5% of all hospitalizations occurred in 15-44 year olds [Popovic, 2001 #2374, Table 10].

Single Pollutant Model

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 $\mu g/m^3$ increase in twenty-four hour average $PM_{2.5}$ [Peters, 2001 #2157, Table 4, p. 2813].

Functional Form: Logistic
Coefficient: 0.024121
Standard Error: 0.009285
Incidence Rate: region-specific daily nonfatal heart attack rate per person 18+ = 93% of region-specific daily heart attack hospitalization rate (ICD code 410) [47]
Population: population of ages 18 and older

[47]This estimate assumes that all heart attacks that are not instantly fatal will result in a hospitalization. In addition, Rosamond et al. [1999 #2373] report that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We applied a factor of 0.93 to the number of hospitalizations to estimate the number of nonfatal heart attacks per year.

Any of 19 Respiratory Symptoms [Krupnick, 1990 #35]

Krupnick et al. [1990 #35] estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. [1990 #35] listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included COH, ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (PM_{10} and ozone)

The C-R function used to estimate the change in ARD2 associated with a change in daily average PM_{10} concentration is based on Krupnick et al. [1990 #35, p. 12]:[48]

$$\Delta ARD2 \cong \beta^{*}_{PM_{10}} \cdot \Delta PM_{10} \cdot pop\,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000461
Standard Error: 0.000239
Population: population of ages 18-64 years[49]

The logistic regression model used by Krupnick et al. [1990 #35] takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

[48]Krupnick and Kopp [1988 #318, p. 2-24] and ESEERCO [1994 #323, p. V-32] used the same C-R functional form as that used here.

[49]Krupnick et al. [1990 #35, Table 1] reported the age distribution in their complete data, but they did not report the ages of individuals that were considered "adult." This analysis assumes that individuals 18 and older were considered adult. Only a small percentage (0.6%) of the study population is above the age of 60, so the C-R function was limited to the adult population. up through the age of 65.

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 | sickness\ or\ not_{t-1}) = p_i = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of COH (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in COH and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[50] Rather than use the full suite of coefficient values, the impact of COH on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to COH:

$$\frac{\partial probability(ARD2)}{\partial COH} = \frac{p_0 \cdot (1 - p_1) \cdot \beta_{COH} \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^*_{COH},$$

where β_{COH} is the reported logistic regression coefficient for COH. Since COH data are not available for the benefits analysis, an estimated PM_{10} logistic regression coefficient is used based on the following assumed relationship between PM_{10}, COH, and TSP:

$$COH = 0.116 \cdot TSP$$

$$PM_{10} = 0.55 \cdot TSP$$

[50]The model without NO_2 [Krupnick, 1990 #35, Table V equation 3] was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. [1990 #35, Table IV] reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially [see \Krupnick, 1990 #35, Table IV]. Both the ozone and COH coefficients dropped by about a factor of two or more.

$$\Rightarrow COH = 0.2109 \cdot PM_{10}$$

$$\Rightarrow \beta_{PM_{10}} = 0.2109 \cdot \beta_{COH} = 0.2109 \cdot 0.0088 = 0.001856.$$

This analysis uses $\beta_{COH} = 0.0088$ [Krupnick, 1990 #35, Table V equation 3]. The conversion from COH to TSP is based on study-specific information provided to ESEERCO [1994 #323, p. V-32]. The conversion of TSP to PM_{10} is from also from ESEERCO [1994 #323, p. V-5], which cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

The change in the incidence of ARD2 associated with a given change in COH is then estimated by:

$$\frac{\partial ARD2}{\partial PM_{10}} \cong \frac{\Delta ARD2}{\Delta PM_{10}}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta PM_{10}} \cong \beta^*_{PM_{10}}$$

$$\Rightarrow \Delta ARD2 \cong \beta^*_{PM_{10}} \cdot \Delta PM_{10}\ .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO [1994 #323, p. V-32], for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$\beta^*_{PM_{10}} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.001856 \cdot \left[0.7775_1 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000461.$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. [1990 #35, Table V]:

$$\Rightarrow \beta_{PM_{10},\,high} = 0.2109 \cdot \beta_{COH,\,high} = 0.2109 \cdot \left(0.0088 + (1.96 \cdot 0.0046)\right) = 0.003757$$

$$\Rightarrow \beta^*_{PM_{10},\,high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.003757 \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000934$$

$$\sigma_{\beta,\,high} = \frac{\beta_{PM_{10},\,high} - \beta_{PM_{10}}}{1.96} = \frac{(0.000934 - 0.000461)}{1.96} = 0.000236$$

$$\beta_{PM_{10},low} = 0.2109 \cdot \beta_{COH,low} = 0.2109 \cdot (0.0088 - (1.96 \cdot 0.0046)) = -4.555 \cdot 10^{-5}$$

$$\Rightarrow \beta^*_{PM_{10},low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot (-4.555 \cdot 10^{-5}) \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = -1.132 \cdot 10^{-5}$$

$$\Rightarrow \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{(0.000461 + 1.132 \cdot 10^{-5})}{1.96} = 0.000241$$

$$\sigma_{\beta} = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.000239 .$$

Lower Respiratory Symptoms [Schwartz, 2000 #1657]

Schwartz et al. [2000 #1657] replicated a previous analysis [Schwartz, 1994 #96] linking PM levels to lower respiratory symptoms in children in six cities in the U.S. The original study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14. The previous study focused on PM_{10}, acid aerosols, and gaseous pollutants, although single-pollutant $PM_{2.5}$ results were reported. Schwartz et al. [2000 #1657] focused more on the associations between $PM_{2.5}$ and $PM_{10-2.5}$ and lower respiratory symptoms. In single and co-pollutant models, $PM_{2.5}$ was significantly associated with lower respiratory symptoms, while $PM_{10-2.5}$ was not. $PM_{10-2.5}$ exhibited a stronger association with cough than did $PM_{2.5}$. The $PM_{2.5}$ C-R function for lower respiratory symptoms is based on the results of the reported co-pollutant model ($PM_{2.5}$ and $PM_{10-2.5}$).

Multipollutant Model ($PM_{2.5}$ and $PM_{10-2.5}$)

In a model with $PM_{10-2.5}$, the $PM_{2.5}$ coefficient and standard error are calculated from the reported odds ratio (1.29) and 95% confidence interval (1.06-1.57) associated with a 15 $\mu g/m^3$ change in $PM_{2.5}$ [Schwartz , 2000 #1657, Table 2].

Functional Form: Logistic
Coefficient: 0.016976
Standard Error: 0.006680
Incidence Rate: daily lower respiratory symptom incidence rate per person = 0.0012 [Schwartz, 1994 #96, Table 2]
Population: population of ages 7 to 14

Minor Restricted Activity Days: Ostro and Rothschild [1989 #60]

Ostro and Rothschild [1989 #60] estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[51] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years. The C-R function for PM is based on this co-pollutant model.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. The elderly appear more likely to die due to PM exposure than other age groups [e.g., \Schwartz, 1994 #149, p. 30;] and a number of studies have found that hospital admissions for the elderly are related to PM exposures [e.g., \Schwartz, 1994 #147; Schwartz, 1994 #144].

Multipollutant Model ($PM_{2.5}$ and ozone)

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient is a weighted average of the coefficients in Ostro and Rothschild [1989 #60, Table 4] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.00741 .$$

[51] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981}\frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981}\frac{1}{\sigma_{\beta_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981}\frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma}\right) = \sum_{i=1976}^{1981}\text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2\cdot\gamma}\right).$$

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00070.$$

Functional Form: Log-linear
Coefficient: 0.00741
Standard Error: 0.00070
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 [Ostro and Rothschild , 1989 #60, p. 243]
Population: adult population ages 18 to 64

Work Loss Days [Ostro, 1987 #456]

Ostro [1987 #456] estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[52] The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels[53] were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function presented here is a weighted average of the coefficients in Ostro [1987 #456, Table III] using the inverse of the variance as the weight.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals under 65. The elderly appear more likely to die due

[52] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

[53] The study used a two-week average pollution concentration; the C-R function uses a daily average, which is assumed to be a reasonable approximation.

to PM exposure than other age groups [e.g., \Schwartz, 1994 #149, p. 30;] and a number of studies have found that hospital admissions for the elderly are related to PM exposures [e.g., \Schwartz, 1994 #147; Schwartz, 1994 #144]. On the other hand, the number of workers over the age of 65 is relatively small; it was approximately 3% of the total workforce in 2001 [U.S. Bureau of the Census, 2002 #2387, Table 561].

Single Pollutant Model

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro [1987 #456, Table III] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0046 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00036 .$$

Functional Form: Log-linear
Coefficient: 0.0046
Standard Error: 0.00036
Incidence Rate: daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 [Adams, 1999 #2355, Table 41; U.S. Bureau of the Census, 1997 #447, No. 22]
Population: adult population ages 18 to 64

Exhibit D-7 Concentration-Response (C-R) Functions for Particulate Matter and Asthma-Related Effects

Endpoint Name	Pollutant	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Beta	Std Error	Functional Form	Notes
Acute Bronchitis	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.022431	0.015957	Logistic	
Asthma Exacerbation, Asthma Attacks	PM_{10}	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	O_3	24-hr avg	0.001436	0.000558	Logistic	
Asthma Exacerbation, Cough	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001156	Logistic	New onset of symptoms
Asthma Exacerbation, Cough	PM_{10}	Vedal et al.	1998	Vancouver, CAN	6-13	All	All	None	24-hr avg	0.007696	0.003786	Logistic	
Asthma Exacerbation, Moderate or Worse	$PM_{2.5}$	Ostro et al.	1991	Denver, CO	All	All	All	None	24-hr avg	0.0006	0.0003	Linear (log of pollutant)	
Asthma Exacerbation, One or More Symptoms	PM_{10}	Yu et al.	2000	Seattle, WA	5-13	All	All	CO, SO_2	24-hr avg	0.004879	0.005095	Logistic	
Asthma Exacerbation, Shortness of Breath	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.003177	0.001550	Logistic	New onset of symptoms
Asthma Exacerbation, Wheeze	$PM_{2.5}$	Ostro et al.	2001	Los Angeles, CA	8-13	Black	All	None	24-hr avg	0.002565	0.001030	Logistic	New onset of symptoms
Chronic Phlegm	$PM_{2.5}$	McConnell et al.	1999	Southern California	9-15	All	All	None	Annual Avg	0.063701	0.025580	Logistic	
Upper Respiratory Symptoms	PM_{10}	Pope et al.	1991	Utah Valley	9-11	All	All	None	24-hr avg	0.0036	0.0015	Logistic	

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Asthma-Related Effects

Acute Bronchitis [McConnell, 1999 #1900]

McConnell et al. [1999 #1900] examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[54] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Bronchitis was defined in the study by the question: "How many times in the past 12 months did your child have bronchitis?" [McConnell, 1999 #1900, p. 757]. It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. McConnell et al. found a relationship between PM and chronic phlegm but none with chronic cough, each of which may be indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on McConnell et al. is measuring acute bronchitis. The $PM_{2.5}$ C-R function for bronchitis among asthmatics is based on the results of the single pollutant model reported in Table 3.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (1.4) and 95% confidence interval (0.9-2.3) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. [McConnell, 1999 #1900, Table 3]

Functional Form: Logistic
Coefficient: 0.022431
Standard Error: 0.015957
Incidence Rate: annual incidence rate of one or more episodes of bronchitis per asthmatic = 0.326 [McConnell, 1999 #1900, Table 2]
Population: population of asthmatics ages 9 to 15 = 5.67%[55] of population ages 9 to 15

[54] Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

[55] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Asthma Attacks [Whittemore and Korn, 1980 #634]

Whittemore and Korn [1980 #634] examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

Multipollutant Model (PM_{10} and ozone)

The PM_{10} C-R function is based on the results of a co-pollutant model of TSP and ozone [Whittemore, 1980 #634, Table 5]. Assuming that PM_{10} is 55 percent of TSP[56] and that particulates greater than ten micrometers are harmless, the coefficient is calculated by dividing the TSP coefficient (0.00079) by 0.55. The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn [1980 #634, Table 5], which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001436
Standard Error: 0.000558
Incidence Rate: daily incidence of asthma attacks = 0.0550[57]
Population: population of asthmatics of all ages = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

Asthma Exacerbation, Cough [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.[58] Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms

[56]The conversion of TSP to PM_{10} is from ESEERCO [1994 #323, p. V-5], who cited studies by EPA [1986 #236] and the California Air Resources Board [1982 #329].

[57] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

[58] The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions.

followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The $PM_{2.5}$ C-R function is based on the results of the single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.03-1.18) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration.

The C-R function based on this model will estimate the number of new onset episodes of cough avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For cough, this ratio is 2.2 (14.5% divided by 6.7%) [Ostro, 2001 #2317, p.202].

In addition, not all children are at-risk for a new onset of cough, as defined by the study. On average, 14.5% of African-American asthmatics have cough on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.145 = 85.5%). As a result, a factor of 85.5% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new cough episode.

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001156
Incidence Rate: daily new onset cough (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.067
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of cough = 6.21% of African-American population ages 8 to 13 multiplied (85.5% at-risk[59] times 7.26% asthmatic[60])
Adjustment Factor: average number of consecutive days with a cough episode (days) = 2.2

Asthma Exacerbation, Cough [Vedal, 1998 #416]

Vedal et al. [1998 #416] studied the relationship between air pollution and respiratory symptoms among asthmatics and non-asthmatic children (ages 6 to 13) in Port Alberni, British Columbia, Canada. Four groups of elementary school children were sampled from a prior cross-

[59] On average, 17.3% of African-American asthmatics have cough episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.145 = 85.5%) are at-risk for a new onset episode.

[60] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

sectional study: (1) all children with current asthma, (2) children without doctor diagnosed asthma who experienced a drop in FEV after exercise, (3) children not in groups 1 or 2 who had evidence of airway obstruction, and (4) a control group of children with matched by classroom. The authors used logistic regression and generalized estimating equations to examine the association between daily PM_{10} levels and daily increases in various respiratory symptoms among these groups. In the entire sample of children, PM_{10} was significantly associated with cough, phlegm, nose symptoms, and throat soreness. Among children with diagnosed asthma, the authors report a significant association between PM_{10} and cough symptoms, while no consistent effects were observed in the other groups. Since the study population has an over-representation of asthmatics, due to the sampling strategy, the results from the full sample of children are not generalizeable to the entire population. The C-R function presented below is based on results among asthmatics only.

Single Pollutant Model

The PM_{10} coefficient and standard error are based on an increase in odds of 8% (95% CI 0-16%) reported in the abstract for a 10 $\mu g/m^3$ increase in daily average PM_{10}.

Functional Form: Logistic
Coefficient: 0.007696
Standard Error: 0.003786
Incidence Rate: daily cough rate per person [Vedal, 1998 #416, Table 1, p. 1038] = 0.086
Population: asthmatic population ages 6 to 13 = 5.67%[61] of population ages 6 to 13

Asthma Exacerbation, Moderate or Worse [Ostro, 1991 #64]

Ostro et al. [1991 #64] examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. Sulfate was linked to shortness of breath. No effects seen for other pollutants. The C-R function is based on a single-pollutant linear regression model where the log of the pollutant is used.

Single Pollutant Model

Two $PM_{2.5}$ coefficients are presented, both equal 0.0006, however only one is significant. The coefficient based on data that does not include estimates of missing $PM_{2.5}$ values is not

[61] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children 5-17 at 5.67% (based on data from the 1999 National Health Interview Survey).

significant (std error = 0.0053); the coefficient that includes estimates of missing $PM_{2.5}$ values (estimated using a function of sulfate and nitrate) is significant at $p < 0.5$ (std error = 0.0003). The latter coefficient is used here.

The C-R function to estimate the change in the number of days with moderate (or worse)

$$\Delta Days\ Moderate / Worse\ Asthma = -\beta \cdot \ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right) \cdot pop\,,$$

asthma

Functional Form: Linear (using log of the pollutant)
Coefficient: 0.0006
Standard Error: 0.0003
Population: population of asthmatics of all ages[62] = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

Asthma Exacerbation, One or More Symptoms [Yu, 2000 #2112]

Yu et al. [2000 #2112] examined the association between air pollution and asthmatic symptoms among mild to moderate asthmatic children ages 5-13 in Seattle. They collected air quality data for CO, SO_2, PM_{10}, and $PM_{1.0}$ and asked study subjects to record symptoms daily. They used logistic regression models with generalized estimating equations in two different approaches. A "marginal approach" was used to estimate the impact of air pollution on asthma symptoms and a "transition approach" was used to estimate the association conditioned on the previous day's outcome. The primary endpoint, odds of at least one asthma symptom, was significantly associated with CO, PM_{10}, and $PM_{1.0}$ in single pollutant models. In multipollutant models, CO remained significant while PM effects declined slightly. The magnitude of the effects were similar between the "marginal" and "transition" approaches. The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable.

Multipollutant Model (PM_{10}, CO, SO_2)

The C-R function is based on the results of the "transition approach," where the previous day's symptoms is an explanatory variable. The multipollutant PM_{10} coefficient and standard error are based on the odds ratio (1.05) and 95% confidence interval (0.95-1.16) for a 10 $\mu g/m^3$ increase in one-day lagged daily average PM_{10} [Yu, 2000 #2112, Table 4, p. 1212].

[62] The C-R function is applied to asthmatics of all ages, although the study population consists of asthmatics between the ages of 18 and 70. It seems reasonable to assume that individuals over the age of 70 are at least as susceptible as individuals in the study population. It also seems reasonable to assume that individuals under the age of 18 are also susceptible. For example, controlling for oxidant levels, Whittemore and Korn [1980 #634] found TSP significantly related to asthma attacks in a study population comprised primarily (59 percent) of individuals less than 16 years of age.

Functional Form: Logistic
Coefficient: 0.004879
Standard Error: 0.005095
Incidence Rate: daily rate of at least one asthma episode per person [Yu, 2000 #2112, Table 2, p. 1212] = 0.60
Population: asthmatic population ages 5 to 13 = 5.67%[63] of population ages 5 to 13

Asthma Exacerbation, Shortness of Breath [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] studied the relationship between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and ozone in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "new onset of a symptom episode". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.10 (95% CI 1.00-1.20) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration [Ostro, 2001 #2317, Table 5, p.204].

The C-R function based on this model will estimate the number of new onset episodes of shortness of breath avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For shortness of breath, this ratio is 2.0 (7.4% divided by 3.7%) [Ostro, 2001 #2317, p.202].

In addition, not all children are at-risk for a new onset of shortness of breath, as defined by the study. On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.074 = 92.6%). As a result, a factor of 92.6% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new shortness of breath episode.

[63] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Functional Form: Logistic
Coefficient: 0.003177
Standard Error: 0.001550
Incidence Rate: daily new onset shortness of breath (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.037
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of shortness of breath = 6.72% of African-American population ages 8 to 13 multiplied (92.6% at-risk[64] times 7.26% asthmatic[65])
Adjustment Factor: average number of consecutive days with a shortness of breath episode (days) = 2.0

Asthma Exacerbation, Wheeze [Ostro, 2001 #2317]

Ostro et al. [2001 #2317] studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found both the prevalence and incidence of wheeze associated with $PM_{2.5}$ PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics. The $PM_{2.5}$ C-R function is based on the results of a single pollutant model looking at the onset of new symptoms.

Single Pollutant Model

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.01-1.14) for a 30 $\mu g/m^3$ increase in 12-hour average $PM_{2.5}$ concentration [Ostro, 2001 #2317, Table 5, p.204].

The C-R function based on this model will estimate the number of new onset episodes of wheeze avoided. In order to convert this estimate to the total number of episodes avoided, the results are adjusted by an estimate of the duration of symptom episodes. The average duration can be estimated from Ostro et al. [2001 #2317] using the ratio of the probability of a symptom episode to the probability of a new onset episode. For wheeze, this ratio is 2.3 (17.3% divided by 7.6%) [Ostro, 2001 #2317, p.202].

[64] On average, 7.4% of African-American asthmatics have shortness of breath episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.074 = 92.6%) are at-risk for a new onset episode.

[65] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

In addition, not all children are at-risk for a new onset of wheeze, as defined by the study. On average, 17.3% of African-American asthmatics have wheeze on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day are at-risk for a new onset episode (1-0.173 = 82.7%). As a result, a factor of 82.7% is used in the function to estimate the population of African-American 8 to 13 year old children at-risk for a new wheeze episode.

Functional Form: Logistic
Coefficient: 0.002565
Standard Error: 0.001030
Incidence Rate: daily new onset wheeze (incidence) rate per person [Ostro, 2001 #2317, p.202] = 0.076
Population: asthmatic African-American population ages 8 to 13 at-risk for a new episode of wheeze = 6.00% of African-American population ages 8 to 13 multiplied (82.7% at-risk[66] times 7.26% asthmatic[67])
Adjustment Factor: average number of consecutive days with a wheeze episode (days) = 2.3

Chronic Phlegm [McConnell, 1999 #1900]

McConnell et al. [1999 #1900] examined the relationship between air pollution and bronchitic symptoms among asthmatic 4th, 7th, and 10th grade children in southern California.[68] The authors collected information on the prevalence of bronchitis, chronic cough, and chronic phlegm among children with and without a history of asthma and/or wheeze. They used annual measurements of ozone, PM_{10}, $PM_{2.5}$, NO_2, and acids in a logistic regression model with adjustments for personal covariates. Neither bronchitis, cough, or phlegm were associated with any of the pollutants among children with no history of wheeze or asthma or a history of wheeze without diagnosed asthma. Among asthmatics, PM_{10} was significantly associated with bronchitis and phlegm; $PM_{2.5}$ was significantly associated with phlegm and marginally associated with bronchitis; NO_2 and acids were both significantly associated with phlegm; and ozone was not significantly associated with any of the endpoints.

Phlegm was defined in the study by the question: "Other than with colds, does this child usually seem congested in the chest or bring up phlegm?" [McConnell, 1999 #1900, p. 757]. The authors refer to this definition as "chronic phlegm" and we also assume that the term "usually" refers to chronic, rather than acute, phlegm. The PM C-R functions for chronic phlegm among asthmatics are based on the results of the single pollutant model reported in Table 3.

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[66] On average, 17.3% of African-American asthmatics have wheeze episodes on a given day [Ostro, 2001 #2317, p.202]. Only those who are symptom-free on the previous day (1-0.173 = 82.7%) are at-risk for a new onset episode.

[67] The American Lung Association [2002 #2358, Table 9] estimates asthma prevalence for African-American children ages 5 to 17 at 7.26% (based on data from the 1999 National Health Interview Survey).

[68] Assuming that a child enters kindergarten at age 5, 4th grade corresponds to age 9 and 10th grade corresponds to age 15. We therefore applied the results of this study to children ages 9 to 15.

Single Pollutant Model

The estimated logistic coefficient and standard error are based on the odds ratio (2.6) and 95% confidence interval (1.2-5.4) associated with an increase in yearly mean 2-week average $PM_{2.5}$ of 15 $\mu g/m^3$. [McConnell, 1999 #1900, Table 3]

Functional Form: Logistic
Coefficient: 0.063701
Standard Error: 0.025580
Incidence Rate: annual incidence rate of phlegm per asthmatic = 0.257 [McConnell, 1999 #1900, Table 2]
Population: population of asthmatics ages 9 to 15 = 5.67%[69] of population ages 9 to 15

Upper Respiratory Symptoms [Pope, 1991 #77]

Using logistic regression, Pope et al. [1991 #77] estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' [Pope, 1991 #77, p. 669]." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample [Pope, 1991 #77, Table 5] show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Single Pollutant Model

The coefficient and standard error for a one $\mu g/m^3$ change in PM_{10} is reported in Table 5.

Functional Form: Logistic

[69] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Coefficient: 0.0036
Standard Error: 0.0015
Incidence Rate: daily upper respiratory symptom incidence rate per person = 0.3419 [Pope, 1991 #77, Table 2]
Population: asthmatic population ages 9 to 11 = 5.67%[70] of population ages 9 to 11

Ozone Concentration-response Functions

Short-term Mortality

Exhibit D-8 summarizes the C-R functions used to estimate the relationship between ozone and short-term mortality. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

[70] The American Lung Association [2002 #2358, Table 7] estimates asthma prevalence for children ages 5 to 17 at 5.67% (based on data from the 1999 National Health Interview Survey).

Exhibit D-8 Concentration-Response (C-R) Functions for Ozone and Short-Term Mortality

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Non-Accidental	Ito and Thurston	1996	Chicago, IL	All	All	All	PM_{10}	1-hr max	Log-linear	0.000634	0.000251
Non-Accidental	Kinney et al.	1995	Los Angeles, CA	All	All	All	PM_{10}	1-hr max	Log-linear	0	0.000214
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	All	All	All	SO_2, TSP	24-hr avg	Log-linear	0.000611	0.000216
Non-Accidental	Samet et al.	1997	Philadelphia, PA	All	All	All	CO, NO_2, SO_2, TSP	24-hr avg	Log-linear	0.000936	0.000312

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Short-Term Mortality, Non-Accidental [Ito, 1996 #187, Chicago]

Ito and Thurston [1996 #187] examined the relationship between daily non-accidental mortality and air pollution levels in Cook County, Illinois from 1985 to 1990. They examined daily levels of ozone, PM_{10}, SO_2, and CO, and found a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. In single pollutant models the effects were slightly larger. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a co-pollutant model with PM_{10}, the coefficient (0.000634) and standard error (0.000251) were obtained directly from the author because the published paper reported incorrect information.

Functional Form: Log-linear
Coefficient: 0.000634
Standard Error: 0.000251
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Kinney, 1995 #191, Los Angeles]

Kinney et al. [1995 #191] examined the relationship between daily non-accidental mortality and air pollution levels in Los Angeles, California from 1985 to 1990. They examined ozone, PM_{10}, and CO, and found a significant relationship for each pollutant in single pollutant models. The effect for ozone dropped to zero with the inclusion of PM_{10} in the model, while the effect for CO and PM_{10} appeared co-pollutant ozone models. The C-R function for ozone is based on the results of the co-pollutant model.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are based on the relative risk (1.00) and 95% confidence interval (0.94-1.06) reported for a 143 ppb increase in daily one-hour maximum ozone concentration [Kinney, 1995 #191, Table 2, p. 64].

Functional Form: Log-linear
Coefficient: 0
Standard Error: 0.000214
Incidence: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Moolgavkar, 1995 #49, Philadelphia]

Moolgavkar et al. [1995 #49] examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant. In season-specific models, ozone was significantly associated with mortality only in the summer months. The C-R function for ozone is based on the full-year three-pollutant model reported in Table 5 [Moolgavkar et al., 1995 #49, p. 482].

Multipollutant Model (ozone, SO_2, TSP)

The coefficient and standard error are based on the relative risk (1.063) and 95% confidence interval (1.018-1.108) associated with a 100 ppb increase in daily average ozone [Moolgavkar et al., 1995 #49, p. 482, Table 5].

Functional Form: Log-linear
Coefficient: 0.000611
Standard Error: 0.000216
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Short-Term Mortality, Non-Accidental [Samet, 1997 #685, Philadelphia]

Samet et al. [1997 #685] examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They examined ozone, TSP, SO_2, NO_2, and CO in a Poisson regression model. In single pollutant models, ozone, SO_2, TSP, and CO were significantly associated with mortality. In a five-pollutant model, they found a positive statistically significant relationship for each pollutant except NO_2. The C-R function for ozone is based on the five-pollutant model (ozone, CO, NO_2, SO_2, and TSP) reported in Table 9 [Samet, 1997 #685, p. 20].

Multipollutant Model (ozone, CO, NO_2, SO_2, and TSP)

In a model with CO, NO_2, SO_2, and TSP, the ozone coefficient and standard error are based on the percent increase (1.91) and t-statistic (3) associated with a 20.219 ppb increase in two-day average ozone [Samet, 1997 #685, p. 20, Table 9].

Functional Form: Log-linear
Coefficient: 0.000936
Standard Error: 0.000312
Incidence Rate: county-level daily non-accidental mortality rate (ICD codes <800) per person
Population: population of all ages

Exhibit D-9 Concentration-Response (C-R) Functions for Ozone and Chronic Illness

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Chronic Asthma	McDonnell et al.	1999	SF, SD, South Coast Air Basin	27+	All	Male	None	annual avg 8-hr avg	Logistic	0.0277	0.0135

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Chronic Illness

Exhibit D-9 summarizes the C-R function ([McDonnell, 1999 #1153]) used to estimate the relationship between ozone and chronic asthma. A more detailed summary of McDonnell et al. [1999 #1153], and the parameters used in the function, is described below.

Chronic Asthma [McDonnell, 1999 #1153]

McDonnell et al. [1999 #1153] used the same cohort of Seventh-Day Adventists as Abbey et al. [1991 #242;, 1993 #245], and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4. The C-R function for ozone is based on the single pollutant model for males reported in Table 5 [McDonnell, 1999 #1153, 1999, p. 117].

Single Pollutant Model

The coefficient and standard error for males is reported in Table 5 for a unit increase in annual average eight-hour ozone concentrations.[71]

Functional Form: Logistic
Coefficient: 0.0277
Standard Error: 0.0135
Incidence Rate: annual asthma incidence rate per person = 0.00219 [McDonnell, 1999 #1153, 1999, Table 4]
Population: non-asthmatic males age 27 and over = 97.9%[72] of males 27+

[71] The eight-hour ozone concentration is defined as 9:00 A.M. to 4:59 P.M. The study used the 1973-1992 mean 8-hour average ambient ozone concentration [McDonnell, 1999 #1153, p. 113].

[72] The prevalence of asthma among males 27 and older (2.10 percent) was estimated from the 2000 National Health Interview Survey (NHIS) public use data, available at ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHIS/2000.

Exhibit D-10 Concentration-Response (C-R) Functions for Ozone and Hospital Admissions

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
All Respiratory	Burnett et al.	2001	Toronto, CAN	<2	All	All	$PM_{2.5}$	1-hr max	Log-linear	0.006309	0.001834
All Respiratory	Schwartz	1995	New Haven, CT	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.002652	0.001398
All Respiratory	Schwartz	1995	Tacoma, WA	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.007147	0.002565
Chronic Lung Disease	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	CO, PM_{10}	24-hr avg	Log-linear	0.002743	0.001699
Chronic Lung Disease (less Asthma)	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00549	0.00205
Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	65+	All	All	NO_2, PM_{10}, SO_2	24-hr avg	Log-linear	0.003696	0.001030
Pneumonia	Schwartz	1994	Detroit, MI	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.00521	0.0013
Pneumonia	Schwartz	1994	Minneapolis, MN	65+	All	All	PM_{10}	24-hr avg	Log-linear	0.003977	0.001865

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Hospital Admissions

Exhibit D-10 summarizes the C-R functions used to estimate the relationship between ozone and hospital admissions. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Hospital Admissions for All Respiratory [Burnett, 2001 #2202, Toronto]

Burnett et al. [2001 #2202] studied the association between air pollution and acute respiratory hospital admissions (ICD codes 493, 466, 464.4, 480-486) in Toronto from 1980-1994, among children less than 2 years of age. They collected hourly concentrations of the gaseous pollutants, CO, NO_2, SO_2, and ozone. Daily measures of particulate matter were estimated for the May to August period of 1992-1994 using TSP, sulfates, and coefficient of haze data. The authors report a positive association between ozone in the May through August months and respiratory hospital admissions, for several single days after elevated ozone levels.

The strongest association was found using a five-day moving average of ozone. No association was found in the September through April months. In co-pollutant models with a particulate matter or another gaseous pollutant, the ozone effect was only slightly diminished. The effects for PM and gaseous pollutants were generally significant in single pollutant models but diminished in co-pollutant models with ozone, with the exception of CO. The C-R function for ozone is based on a co-pollutant model with $PM_{2.5}$, using the five-day moving average of one-hour max ozone.

Multipollutant Model (ozone and $PM_{2.5}$)

In a model with $PM_{2.5}$, the coefficient and standard error are based on the percent increase (33.0) and t-statistic (3.44) associated with a 45.2 ppb increase in the five-day moving average of one-hour max ozone [Burnett, 2001 #2202, Table 3].

Functional Form: Log-linear
Coefficient: 0.006309
Standard Error: 0.001834
Incidence Rate: region-specific daily hospital admission rate for all respiratory admissions per person less than 2 years of age (ICD codes 464, 466, 480-487, 493)
Population: population less than 2 years of age

Hospital Admissions for All Respiratory [Schwartz, 1995 #153, New Haven]

Schwartz [1995 #153] examined the relationship between air pollution and respiratory hospital admissions (ICD codes 460-519) for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In a co-pollutant model with ozone and PM_{10}, both pollutants were significant. PM_{10} remained significant in a model with SO_2,

while ozone was marginally significant when adjusted for SO_2. SO_2 was significant in a co-pollutant model with PM_{10} but not with ozone. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.15) for a 50 μg/m^3 increase in average daily ozone levels [Schwartz,1995 #153, Table 3, p. 534].[73]

Functional Form: Log-linear
Coefficient: 0.002652
Standard Error: 0.001398
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

Hospital Admissions for All Respiratory [Schwartz, 1995 #153, Tacoma]

Schwartz [1995 #153] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. Ozone remained significant in separate co-pollutant models with PM_{10} and SO_2. PM_{10} remained significant in a co-pollutant model with SO_2, but not in a co-pollutant model with ozone. SO_2 was not significant in either of the co-pollutant models. The ozone C-R function is based on results from the co-pollutant model with PM_{10}.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.06-1.37) for a 50 μg/m^3 increase in average daily ozone levels [Schwartz, 1995 #153, Table 6, p. 535].[74]

Functional Form: Log-linear
Coefficient: 0.007147

[73] To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

[74]To calculate the coefficient, a conversion of 1.96 μg/m^3 per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million μg in a gram, this density means that there are 1.96 billion μg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 μg of ozone (i.e., one ppb = 1.96 μg/m^3).

Standard Error: 0.002565
Incidence Rate: region-specific daily hospital admission rate for respiratory admissions per person 65+ (ICD codes 460-519)
Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease [Moolgavkar, 1997 #53, Minneapolis]

Moolgavkar et al. [1997 #53] examined the relationship between air pollution and hospital admissions (ICD codes 490-496) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a Poisson regression, they found no significant effect for any of the pollutants (PM_{10}, ozone, or CO). The effect for ozone was marginally significant. The model with a 100 df smoother was reported to be optimal (p. 368). The C-R function is based on the results from a three-pollutant model (ozone, CO, PM_{10}) using the 100 df smoother.

Multipollutant Model (ozone, CO, PM_{10})

In a model with CO and PM_{10}, the estimated coefficient and standard error are based on the percent increase (4.2) and 95% confidence interval of the percent increase (-1.0-9.4) associated with a change in daily average ozone levels of 15 ppb [Moolgavkar, 1997 #53, Table 4 and p. 366].

Functional Form: Log-linear
Coefficient: 0.002743
Standard Error: 0.001699
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-496)
Population: population of ages 65 and older

Hospital Admissions for Chronic Lung Disease (less Asthma) [Schwartz, 1994 #144, Detroit]

Schwartz [1994 #144] examined the relationship between air pollution and hospital admissions (ICD codes 491-492, 494-496) for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The C-R function for chronic lung disease incidence is based on the results of the "basic" co-pollutant model (ozone and PM_{10}) presented in Table 4 (p. 651).[75]

[75] Schwartz [1994 #144] also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

Multipollutant Model (ozone and PM_{10})

The coefficient and standard error for the "basic" model are reported in Table 4 [Schwartz , 1994 #144, p.651] for a one ppb change in daily average ozone.

Functional Form: Log-linear
Coefficient: 0.00549
Standard Error: 0.00205
Incidence Rate: region-specific daily hospital admission rate for chronic lung disease per person 65+ (ICD codes 490-492, 494-496)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Moolgavkar, 1997 #53, Minneapolis]

Moolgavkar et al. [1997 #53] examined the relationship between air pollution and pneumonia hospital admissions (ICD 480-487) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant Poisson model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. The model with a 130 df smoother was reported to be optimal (p. 368). The ozone C-R function is based on the results from the four-pollutant model with a 130 df smoother.

Multipollutant Model (ozone, NO_2, PM_{10}, SO_2)

In a model with NO_2, PM_{10},and SO_2, the estimated coefficient and standard error are based on the percent increase (5.7) and 95% confidence interval of the percent increase (2.5-8.9) associated with an increase in daily average ozone levels of 15 ppb [Moolgavkar, 1997 #53, Table 4 and p. 366].

Functional Form: Log-linear
Coefficient: 0.003696
Standard Error: 0.00103
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Schwartz, 1994 #144, Detroit]

Schwartz [1994 #144] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were

relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The PM_{10} C-R function for pneumonia incidence is based on results of the "basic" co-pollutant model (ozone and PM_{10}).[76]

Multipollutant Model (ozone and PM_{10})

The ozone C-R function for pneumonia incidence is based on the coefficient and standard error for the "basic" co-pollutant model presented in Table 4 [Schwartz, 1994 #144, p. 651].

Functional Form: Log-linear
Coefficient: 0.00521
Standard Error: 0.0013
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

Hospital Admissions for Pneumonia [Schwartz, 1994 #143, Minneapolis]

Schwartz [1994 #143] examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In single-pollutant Poisson regression models, both ozone and PM_{10} were significantly associated with pneumonia admissions. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. The results were not sensitive to the methods used to control for seasonal patterns and weather. The ozone C-R function is based on the results of the two-pollutant model (PM_{10} and ozone) with spline smoothing for temporal patterns and weather.

Multipollutant Model (ozone and PM_{10})

In a model with PM_{10} and spline functions to adjust for time and weather, the coefficient and standard error are based on the relative risk (1.22) and 95% confidence interval (1.02, 1.47) for a 50 ppb increase in daily average ozone levels [Schwartz, 1994 #143, Table 4].

Functional Form: Log-linear
Coefficient: 0.003977
Standard Error: 0.001865
Incidence Rate: region-specific daily hospital admission rate for pneumonia per person 65+ (ICD codes 480-487)
Population: population of ages 65 and older

[76] Schwartz [1994 #144] also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

Exhibit D-11 Concentration-Response (C-R) Functions for Ozone and Emergency Room Visits

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma	Cody et al.	1992	New Jersey (Northern)	All	All	All	SO_2	5-hr avg	Linear	0.0203	0.00717
Asthma	Stieb et al.	1996	New Brunswick, CAN	All	All	All	None	1-hr max	Quadratic	0.00004	0.00002
Asthma	Weisel et al.	1995	New Jersey (Northern and Central)	All	All	All	None	5-hr avg	Linear	0.0443	0.00723

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Emergency Room Visits

Exhibit D-11 summarizes the C-R functions used to estimate the relationship between ozone and emergency room visits. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Emergency Room Visits for Asthma [Cody, 1992 #914, Northern NJ]

Cody et al. [1992 #914] examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1988-1989. In a two pollutant multiple linear regression model, ozone was linked to asthma visits, and no effect was seen for SO_2. They modeled PM_{10} in separate analysis because of limited (every sixth day) sampling. No significant effect was seen for PM_{10}. The C-R function for ozone is based on results of a co-pollutant model with SO_2 [Cody, 1992 #914, Table 6, p. 191].

Multipollutant Model (ozone and SO_2)

The ozone coefficient and standard error are reported per 1 ppm increment of five-hour ozone levels, which are converted to a 1 ppb increment by dividing by 1,000 [Cody, 1992 #914, Table 6, p. 191].

Functional Form: Linear
Coefficient: 0.0203
Standard Error: 0.00717

Baseline Population: baseline population of Northern New Jersey[77] = 4,436,976
Population: population of all ages

Emergency Room Visits for Asthma [Stieb, 1996 #218, New Brunswick]

Stieb et al. [1996 #218] examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb. The authors reported results from a linear model, quadratic model, and linear-quadratic model using daily average and 1-hour maximum ozone. In the linear model, ozone was borderline significant. In the quadratic and linear-quadratic models, ozone was highly significant. This is consistent with the author's conclusion that "only ozone appeared to have a nonlinear relationship with visit rates" (p. 1356) and that "quadratic, linear-quadratic, and indicator models consistently fit the data better than the linear model ..." (p. 1358). The linear term in the linear-quadratic model is negative, implying that at low ozone levels, increases in ozone are associated with decreases in risk. Since this does not seem biologically plausible, the ozone C-R function described here is based on the results of the quadratic regression model presented in Table 2 [Stieb et al., 1996 #218, p. 1356], for a change in one-hour maximum ozone levels.

Single Pollutant Model

The coefficient and standard error of the quadratic model are reported in Table 2 [Stieb et al., 1996 #218, p. 1356] for a 1 ppb increase in 1-hour daily maximum ozone levels. The C-R function to estimate avoided emergency visits derived from a quadratic regression model is shown below:

$$\Delta\, Asthma\ ERVisits = \frac{\beta}{BasePop} \cdot [(O_{3,baseline})^2 - (O_{3,control})^2] \cdot pop,$$

Functional Form: Quadratic
Coefficient: 0.00004
Standard Error: 0.00002
Baseline Population: baseline population of St. John, New Brunswick [Stieb, 1996 #218, p. 1354] = 125,000
Population: population of all ages

[77] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. [1992 #914, Figure 1] presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

Emergency Room Visits for Asthma [Weisel, 1995 #688, Northern NJ]

Weisel et al. [1995 #688] examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1986-1990. A significant relationship was reported for ozone. The C-R function is based on the results of the single pollutant models reported by Weisel et al. [1995 #688, Table 2].

Single Pollutant Model

The coefficient (β) used in the C-R function is a weighted average of the coefficients in Weisel et al. [1995 #688, Table 2] using the inverse of the variance as the weight:

$$\beta = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.0443 .$$

The standard error of the coefficient (σ_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = \text{var}\left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{\sigma_{\beta_i}^2}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1986}^{1990} \text{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This eventually reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.00723 .$$

Functional Form: Linear
Coefficient: 0.0443
Standard Error: 0.00723
Baseline Population: baseline population of Northern New Jersey[78] = 4,436,976
Population: population of all ages

[78] The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. [1992 #914, Figure 1] presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

Exhibit D-12 Concentration-Response (C-R) Functions for Ozone and Acute Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Any of 19 Respiratory Symptoms	Krupnick	1990	Los Angeles, CA	18-64	All	All	COH	1-hr max	Linear	0.000137	0.000070
Minor Restricted Activity Days	Ostro and Rothschild	1989	nationwide	18-64	All	All	$PM_{2.5}$	24-hr avg	Log-linear	0.0022	0.000658
School Loss Days, All Cause	Chen et al.	2000	Washoe Co, NV	6-11	All	All	CO, PM_{10}	1-hr max	Linear	0.013247	0.004985
School Loss Days, All Cause	Gilliland et al.	2001	Southern California	9-10	All	All	None	8-hr avg	Log-linear	0.00755	0.004527
Worker Productivity	Crocker and Horst	1981	nationwide	18-64	All	All	None	24-hr avg	Linear	0.14	–

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Acute Morbidity

Exhibit D-12 summarizes the C-R functions used to estimate the relationship between ozone and acute morbidity. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Any of 19 Respiratory Symptoms: Krupnick [1990 #35]

Krupnick et al. [1990 #35] estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. [1990 #35] listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included coefficient of haze (COH, a measure of particulate matter concentrations), ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily ozone, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

Multipollutant Model (ozone and coefficient of haze)

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone[79] is based on Krupnick et al. [1990 #35, p. 12]:[80]

$$\Delta ARD2 \cong \beta^* \cdot \Delta O_3 \cdot pop \,,$$

Functional Form: Linear
Coefficient: first derivative of the stationary probability = 0.000137
Standard Error: 0.0000697

[79]Krupnick et al. [1990 #35] used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[80]Krupnick and Kopp [1988 #318, p. 2-24] and ESEERCO [1994 #323, p. V-32] used the same C-R functional form as that used here.

Population: population of ages 18-64 years[81]

The logistic regression model used by Krupnick et al. [1990 #35] takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$p_i = probability(ARD2 | sickness\ or\ not_{t-1}) = \frac{1}{1 - e^{\beta_0 + \beta_1 \cdot ARD2_{t-1} + X \cdot \beta}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[82] Rather than use the full suite of coefficient values, the impact of ozone on the probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[83]

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[81]The coefficient estimates are based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. [1990 #35, Table 1], about 0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals 65 years of age and older.

[82]The model without NO_2 [Krupnick, 1990 #35, Table V equation 3] was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[83]The derivative result is reported by Krupnick et al. [1990 #35, p. 12].

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot \beta \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = \beta^* ,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong \beta^*$$

$$\Rightarrow \Delta ARD2 \cong \beta^* \cdot \Delta O_3 .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO [1994 #323, p. V-32] for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$\beta^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137 .$$

The *standard error* for the coefficient is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. [1990 #35, Table V]:

$$\beta_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow \beta^*_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$\sigma_{\beta, high} = \frac{\beta_{high} - \beta}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$\beta_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow \beta^*_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{\left(0.000137 + 5.17 \cdot 10^{-6}\right)}{1.96} = 0.0000725$$

$$\sigma_{\beta} = \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} = 0.0000697 .$$

Minor Restricted Activity Days: Ostro and Rothschild [1989 #60]

Ostro and Rothschild [1989 #60] estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas.[84] The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone had a highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years. The C-R function for ozone is based on the co-pollutant model with $PM_{2.5}$.

The study is based on a "convenience" sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to ozone as individuals under 65. A number of studies have found that hospital admissions for the elderly are related to ozone exposures [e.g., \Schwartz,1995 #153; Schwartz , 1994 #144].

Multipollutant Model (ozone and $PM_{2.5}$)

The coefficient and standard error used in the C-R function are based on a weighted average of the coefficients in Ostro and Rothschild [1989 #60, Table 4]. The derivation of these estimates is described below.

Functional Form: Log-linear
Coefficient: 0.00220
Standard Error: 0.000658
Incidence Rate: daily incidence rate for minor restricted activity days (MRAD) = 0.02137 [Ostro and Rothschild , 1989 #60, p. 243]
Population: adult population ages 18 to 64

[84] The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild [1989 #60, Table 4] using the inverse of the variance as the weight:[85]

$$\beta = \left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = 0.00220 .$$

The standard error of the coefficient is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$\sigma_\beta^2 = VAR\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} \right) = VAR\left(\frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} VAR\left(\frac{\beta_i}{\sigma_{\beta_i}^2 \cdot \gamma} \right).$$

This reduces down to:

$$\sigma_\beta^2 = \frac{1}{\gamma} \Rightarrow \sigma_\beta = \sqrt{\frac{1}{\gamma}} = 0.000658 .$$

School Loss Days, All Cause [Chen, 2000 #2101]

Chen et al. [2000 #2101] studied the association between air pollution and elementary school absenteeism (grades 1-6)[86] in Washoe County, Nevada. Daily absence data were available for all elementary schools in the Washoe Country School District. The authors regressed daily total absence rate on the three air pollutants, meteorological variables, and indicators for day of the week, month, and holidays. They reported statistically significant associations between both ozone and CO and daily total absence rate for grades one through six. PM_{10} was negatively associated with absence rate, after adjustment for ozone, CO, and meteorological and temporal variables. The C-R function for ozone is based on the results from a multiple linear regression model with CO, ozone, and PM_{10}.

[85] The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro [1987 #456].

[86] Assuming that most children start kindergarten at age 5, the corresponding ages for grades 1 through 6 would be 6 through 11.

Multipollutant Model (ozone, CO, and PM_{10})

The coefficient and standard error are presented in Table 3 [Chen, 2000 #2101, p. 1008] for a unit ppm increase in the two-week average of daily one-hour maximum ozone concentration. This is converted to unit ppb increase by dividing by 1,000.

The reported coefficient represents an *absolute* increase in absenteeism rate for a unit increase in ozone. If we apply this study to other locations, we assume that the same absolute increase will occur for a unit increase in ozone, regardless of the baseline rate. If the study location has a particularly high baseline rate, we may be overestimating decreases in absenteeism nationally, and vice-versa. As an example, consider if the baseline absenteeism rate were 10% in the study and 5% nationally. An absolute increase in absence rate of 2% associated with a given increase in ozone reflects a relative increase in absence rate of 20% for the study population. However, in the national estimate, we would assume the same absolute increase of 2%, but this would reflect a relative increase in the absenteeism rate of 40%.

An alternative approach is to estimate apply the *relative* increase in absenteeism rate in the C-R function by adjusting the results by the ratio of the national absenteeism rate to the study-specific rate. As a result, the percent increase in absenteeism rate associated with an increase in ozone is extrapolated nationally rather than the absolute increase in absenteeism rate. The incidence derivation section above describes the data used to estimate national and study-specific absence rates.

In addition to this scaling factor, there are two other scaling factors which are applied to the function. A scaling factor of 0.01 is used to convert the beta from a percentage (x 100) per unit increase of ozone to a proportion per unit increase of ozone. As a result it can be applied directly to the national population of school children ages 6 through 11 to estimate the number of absences avoided.

The final scaling factor adjusts for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7). The C-R function parameters are shown below.

Functional Form: Linear
Coefficient: 0.013247
Standard Error: 0.004985
Population: population of children ages 6-11

Scaling Factor 1: Ratio of national school absence rate to study-specific school absence rate[87] = 1.081
Scaling Factor 2: Convert beta in percentage terms to a proportion = 0.01
Scaling Factor 3: Proportion of days that are school days in the ozone season[88] = 0.393

School Loss Days, All Cause [Gilliland, 2001 #2151]

Gilliland et al. [2001 #2151] examined the association between air pollution and school absenteeism among 4th grade school children (ages 9-10) in 12 southern Californian communities. The study was conducted from January through June 1996. The authors used school records to collect daily absence data and parental telephone interviews to identify causes. They defined illness-related absences as respiratory or non-respiratory. A respiratory illness was defined as an illness that included at least one of the following: runny nose/sneezing, sore throat, cough, earache, wheezing, or asthma attack. The authors used 15 and 30 day distributed lag models to quantify the association between ozone, PM_{10}, and NO_2 and incident school absences. Ozone levels were positively associated with all school absence measures and significantly associated with all illness-related school absences (non-respiratory illness, respiratory illness, URI and LRI). Neither PM_{10} nor NO_2 was significantly associated with illness-related school absences, but PM_{10} was associated with non-illness related absences. The C-R function for ozone is based on the results of the single pollutant model.

Gilliland et al. [2001 #2151] defines an incident absence as an absence that followed attendance on the previous day and the incidence rate as the number of incident absences on a given day over the population at risk for an absence on a given day (i.e. those children who were not absent on the previous day). Since school absences due to air pollution may last longer than one day, an estimate of the average duration of school absences could be used to calculated the total avoided school loss days from an estimate of avoided new absences. A simple ratio of the total absence rate divided by the new absence rate would provide an estimate of the average duration of school absences, which could be applied to the estimate of avoided new absences as follows:

$$Duration = \frac{totalAbsences}{newAbsences}$$

$$\Delta TotalAbsences = -\left[incidence \cdot \left(e^{-\beta \cdot \Delta O_3} - 1\right)\right] \cdot duration \cdot pop$$

[87] National school absence rate of 5.5% obtained from the U.S. Department of Education [1996 #2377, Table 42-1]. Study-specific school absence rate of 5.09% obtained from Chen et al. [2000 #2101, Table 1].

[88] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

Since the function is log-linear, the baseline incidence rate (in this case, the rate of new absences) is multiplied by duration, which reduces to the total school absence rate. Therefore, the same result would be obtained by using a single estimate of the total school absence rate in the C-R function. Using this approach, we assume that the same relationship observed between pollutant and new school absences in the study would be observed for total absences on a given day. As a result, the total school absence rate is used in the function below. The derivation of this rate is described in the section on baseline incidence rate estimation.

Single Pollutant Model

For all absences, the coefficient and standard error are based on a percent increase of 16.3 percent (95% CI -2.6 percent, 38.9 percent) associated with a 20 ppb increase in 8-hour average ozone concentration [2001 #2151, Table 6, p. 52].

A scaling factor is used to adjust for the number of school days in the ozone season. In the modeling program, the function is applied to every day in the ozone season (May 1 - September 30), however, in reality, school absences will be avoided only on school days. We assume that children are in school during weekdays for all of May, two weeks in June, one week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

In addition, not all children are at-risk for a new school absence, as defined by the study. On average, 5.5% of school children are absent from school on a given day [U.S. Department of Education, 1996 #2377, Table 42-1]. Only those who are in school on the previous day are at risk for a new absence (1-0.055 = 94.5%). As a result, a factor of 94.5% is used in the function to estimate the population of school children at-risk for a new absence.

Functional Form: Log-linear
Coefficient: 0.007550
Standard Error: 0.004527
Incidence Rate: daily school absence rate = 0.055 [U.S. Department of Education, 1996 #2377, Table 42-1]
Population: population of children ages 9-10 not absent from school on a given day[89] = 94.5% of children ages 9-10
Scaling Factor: Proportion of days that are school days in the ozone season[90] = 0.393

Worker Productivity: Crocker and Horst [1981 #636]

[89] The proportion of children not absent from school on a given day (5.5%) is based on 1996 data from the U.S. Department of Education [1996 #2377, Table 42-1].

[90] Ozone is modeled for the 5 months from May 1 through September 30. We assume that children are in school during weekdays for all of May, 2 weeks in June, 1 week in August, and all of September. This corresponds to approximately 2.75 months out of the 5 month season, resulting in an estimate of 39.3% of days (2.75/5*5/7).

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst [1981 #636] and summarized in EPA [1994 #637]. Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[91] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the national median daily income for outdoor workers engaged in strenuous activity reported by the U.S. Census Bureau [2002 #2387], $68 per day (2000$),[92] a ten percent reduction in ozone yields about $0.97 in increased daily wages. We adjust the national median daily income estimate to reflect regional variations in income using a factor based on the ratio of county median household income to national median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

Single Pollutant Model

The C-R function for estimating changes in worker productivity is shown below:

$$\Delta productivity = \beta \cdot \frac{Q_1 - Q_0}{Q_1} \cdot dailyincome \cdot pop \cdot,$$

Functional Form: Linear
Coefficient: 0.1427
Daily Income: median daily income for outdoor workers[93]
Population: population of adults 18 to 64 employed as farm workers

[91] The relationship estimated by Crocker and Horst between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of the log of ozone in the model.

[92] The national median daily income for workers engaged in "farming, forestry, and fishing" from the U.S. Census Bureau [2002 #2387, Table 621, p. 403] is used as a surrogate for outdoor workers engaged in strenuous activity.

[93] The national median daily income for workers engaged in "farming, forestry, and fishing" was obtained from the U.S. Census Bureau [2002 #2387, Table 621, p. 403] and is used as a surrogate for outdoor workers engaged in strenuous activity. This national median daily income ($68) is then scaled by the ratio of national median income to county median income to estimate county median daily income for outdoor workers.

Exhibit D-13 Concentration-Response (C-R) Functions for Ozone and Asthma-Related Effects

Endpoint Name	Author	Year	Location	Age	Race	Gender	Other Pollutants	Averaging Time[1]	Functional Form	Beta	Std Error
Asthma Exacerbation, Asthma Attacks	Whittemore and Korn	1980	Los Angeles, CA	All	All	All	TSP	1-hr max	Logistic	0.001843	0.000715

1. The averaging time refers to the metric used in the benefits model. This may differ slightly from the averaging time used in the study. Refer to the study summaries below for more detail on the specific averaging time used in the study.

Asthma-Related Effects

Exhibit D-13 summarizes the C-R functions used to estimate the relationship between ozone and asthma-related effects. Detailed summaries of each of the studies used to generate the functions are described below, along with the parameters used in each of the functions.

Asthma Attacks [Whittemore and Korn, 1980 #634]

Whittemore and Korn [1980 #634] examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted so it may be used with ozone data.

Multipollutant Model (ozone and PM_{10})

The daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm. The coefficient is converted to ppb by dividing by 1,000 and to ozone by multiplying by 1.11.[94] The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn [1980 #634, Table 5], which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

Functional Form: Logistic
Coefficient: 0.001843
Standard Error: 0.000715
Incidence Rate: daily incidence of asthma attacks = 0.0550[95]
Population: population of asthmatics of all ages = 3.86% of the population of all ages [American Lung Association, 2002 #2358, Table 7]

[94] The study used oxidant measurements in ppm [Whittemore, 1980 #634, p. 688]; these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper [U.S. EPA, 1996 #455, p.164] is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for ozone at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that ozone remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."

[95] Based on an analysis of the 1999 National Health Interview Survey, the daily incidence of wheezing attacks for adult asthmatics is estimated to be 0.0550. In the same survey, wheezing attacks for children were examined, however, the number of wheezing attacks per year were censored at 12 (compared to censoring at 95 for adults). Due to the potential for underestimation of the number of children's wheezing attacks, we used the adult rate for all individuals.

APPENDIX E

UPDATING THE FIRST PROSPECTIVE STUDY'S TITLE VI ANALYSIS

This appendix presents a revised approach for a benefit/cost analysis of the Clean Air Act's Title VI (regulation of ozone depleting substances). Updating the first prospective study's analysis of Title VI costs and benefits is necessary for several reasons.

First, a few new ozone-depleting substances (ODS) regulations are now in place – or are expected to be promulgated – that will impose costs on and offer benefits to U.S. citizens. The 812 control scenario will be updated to incorporate these new regulations.

Second, the first prospective study relied extensively on regulatory impact analysis (RIA) estimates of benefits and costs prepared at various times in the past for specific stratospheric ozone protection regulations. Much has changed since the preparation of the background RIAs used in the 812 study regarding ozone depletion science, the response of health effects due to ultraviolet (UV) exposure, and many other factors central to estimating the benefits of addressing ozone depletion and recovery. In response to these changes, EPA has updated input data, such as population projections and ODS emissions, and has incorporated several advancements into its model of ozone depletion and health impacts, including improvements in the following areas:

- measurement of stratospheric ozone concentrations;
- forecasts of the impact of emissions of certain ODS on stratospheric ozone concentrations;
- predictions of the impact of changing ozone concentrations on ultraviolet (UV) radiation intensity at the earth's surface; and
- the roles of different spectra of UV radiation, behavior, age of exposure, and year of birth in producing skin cancers and other human health effects.

Finally, in their review of the first prospective study, the SAB raised a number of methodological, technical, and empirical issues for the 812 project team to consider. Many of these issues are related to the advancements in linkages between ODS emissions and ozone depletion; between ozone depletion and UV changes; and between UV changes and health effects. The SAB also recommended that the next 812 study provide an enhanced analysis of the uncertainty associated with Title VI benefits and costs.

To update the first prospective study's summary of stratospheric ozone protection costs and benefits – especially in light of the SAB's and others' comments and questions – we plan to make several revisions to the Title VI analysis methodology, primarily to the benefits assessment. The remainder of this appendix discusses this revised methodology First we describe the proposed ODS emissions scenarios for the analysis. Next, we provide an overview of the approaches to estimating

costs and benefits for Title VI. Finally, we list the major comments on the previous Title VI analysis and describe how these comments are addressed in the second prospective study.

SCENARIOS

As in the rest of the 812 analysis, we propose to develop a pre-CAAA baseline scenario without CAAA-related ODS regulations and a post-CAAA scenario that includes Title VI ODS controls. The Clean Air Act Amendments ODS Title VI phase out scenario ("post-CAAA scenario") will reflect compliance by the United States and all of the rest of the world with the cumulative controls required under the major international agreements reached over the past fifteen years. These agreements include the original Montreal Protocol, the London Amendments, the Copenhagen Amendments, and the latest agreement, the Montreal Adjustments. All of these control programs except the Montreal Adjustments were reflected in the control scenario of the first prospective study. The increasing stringency of the ODS restrictions imposed by each of these policies is summarized in Exhibit E-1.

Exhibit E-1: ODS Restrictions Mandated by Four International Agreements

Policy/Emission Scenario	Description
Montreal Protocol (1987)	Developed countries subject to a freeze on CFCs in 1989, declining to a 50% cap in 1998; Freeze on halons in 1992. Developing countries subject to the same restrictions with a 10-year delay.
London Amendments (1990)	Developed countries subject to a phase out of CFCs, halons, and carbon tetrachloride by 2000, and methyl chloroform by 2005. Developing countries subject to the same restrictions with a 10-year delay.
Copenhagen Amendments (1992)	Developed countries subject to an accelerated phase out for CFCs (1996), halons (1994), carbon tetrachloride (1996), and methyl chloroform (1996); methyl bromide freeze in 1995, and HCFC controls beginning with a freeze in 1996, declining to a full phaseout in 2030.
Montreal Adjustments (1997)	Developed countries subject to all existing controls and a methyl bromide phase out by 2005. Developing countries subject to all existing controls, a freeze on HCFCs in 2016 with an eventual phaseout in 2040, and a methyl bromide freeze in 2002, declining to a full phaseout in 2015.

The second prospective analysis will measure all costs and benefits of Title VI provisions relative to a baseline Pre-CAAA scenario. Under the pre-CAAA scenario, the United States will be assumed to comply with ODS controls only for the original Montreal Protocol, which predates the Clean Air Act Amendments by several years. This means that the United States will only be

subject to a freeze on CFCs in 1989, which then drops to an 80% cap in 1993, and to a final 50% cap in 1998. Halons will be subject only to a freeze in 1992. All other U.S. ODS uses will be uncontrolled for this scenario. Under the Post-CAAA scenario, the United States will also comply with the ODS phaseout controls as specified in Title VI, Sections 604-606. In addition, the Pre-CAAA scenario will assume that all countries other than the United States will comply with all of the restrictions embodied in the various international agreements up to and including the Montreal Adjustments. Thus, the rest of the world's emissions will be the same in the Pre-CAAA and the Post-CAAA scenarios. Exhibit E-2 presents a brief description of each scenario to be used in the analysis.

Exhibit E-2: Summary of Scenarios for the Title VI Cost/Benefit Analysis

Title VI Scenario Summary		
	Assumptions/Requirements	
	United States	**All Other Countries**
"Pre-CAAA" Baseline Scenario	No ODS controls beyond those mandated prior to the 1990 Clean Air Act Amendments (i.e., only the Montreal Protocol controls on CFCs and halons)	Full compliance with ODS reductions in accordance with the Montreal Protocol, the London Amendments, the Copenhagen Amendments, and the most recent Montreal Adjustments international agreements
"Post-CAAA" Control Scenario	Implementation of Sections 604-606 (ODS Phaseout) Implementation of Sections 608-609 (ODS product servicing, recycling and disposal) Implementation of Section 611 (ODS labeling)	Full compliance with ODS reductions in accordance with the Montreal Protocol, the London Amendments, the Copenhagen Amendments, and the most recent Montreal Adjustments international agreements

COST ESTIMATION APPROACH

The approach to estimating costs of Title VI provisions is essentially the same as the one used in the previous prospective analysis. Existing regulatory impact assessments (RIAs) for individual provisions of Title VI will be the source of social cost data for the phasing out of ODS. The total cost estimate of Title VI comprises the costs of Sections 604 and 606 and the incremental costs of sections 608, 609, and 611.

To update the cost analysis, EPA plans to retrieve original cost data from each RIA for use in the second prospective analysis. Costs are evaluated between 1990 and 2075, as in the first prospective. The proposed discount rate for the primary cost estimate is three percent, with a sensitivity analysis using a rate of seven percent.

BENEFITS ESTIMATION APPROACH

The Title VI benefits approach will provide estimates for human health and ecological effects based on a comparison of the baseline Pre-CAAA and Post-CAAA control scenarios. The primary difference between the benefits estimation approach in the first and second prospective analyses is that the second prospective will not rely on RIAs for health benefit estimates but instead will generate new estimates using EPA's Atmospheric Health Effects Framework (AHEF) model. This model consists of several modules that compute stratospheric ozone concentrations from past and predicted future emissions of ODS, forecast ground-level UV resulting from the predicted stratospheric ozone concentrations, and predict future health effects due to increased UV exposure. The AHEF is the centerpiece of the Title VI benefits analysis for the second prospective study.

To calculate monetary values of quantified Title VI benefits, EPA will multiply the physical effects estimates by appropriate unit values for each effect. EPA plans to re-evaluate and update, if necessary, the unit values used in the first prospective analysis. Monetized benefits for each effect category will be expressed as a net present value, using a discount rate for primary estimates of three percent. (A rate of seven percent will be used for a sensitivity analysis.) Total monetized Title VI benefits will be estimated by summing net present value benefits across effect categories.

In addition to the benefits estimated by AHEF, EPA will include in its analysis a qualitative discussion of health and ecological benefits that scientists have identified but that cannot yet be quantified. A search of the available literature reveals very little conclusive information regarding quantified health and ecological benefits from the reduction of future emissions of ODS. The available information does indicate that any currently unquantified health and ecological benefits are minimal compared to the benefits estimated by AHEF.

AHEF Modeling Approach

This section first summarizes the main steps associated with the AHEF modeling approach and describes the inputs for each step in the model approach. This is followed by an outline of the uncertainties associated with the AHEF model and its inputs and a discussion of the proposed changes to the model that are currently being developed by EPA.

Figure E-1 presents a flow diagram of the steps and inputs to the AHEF model.

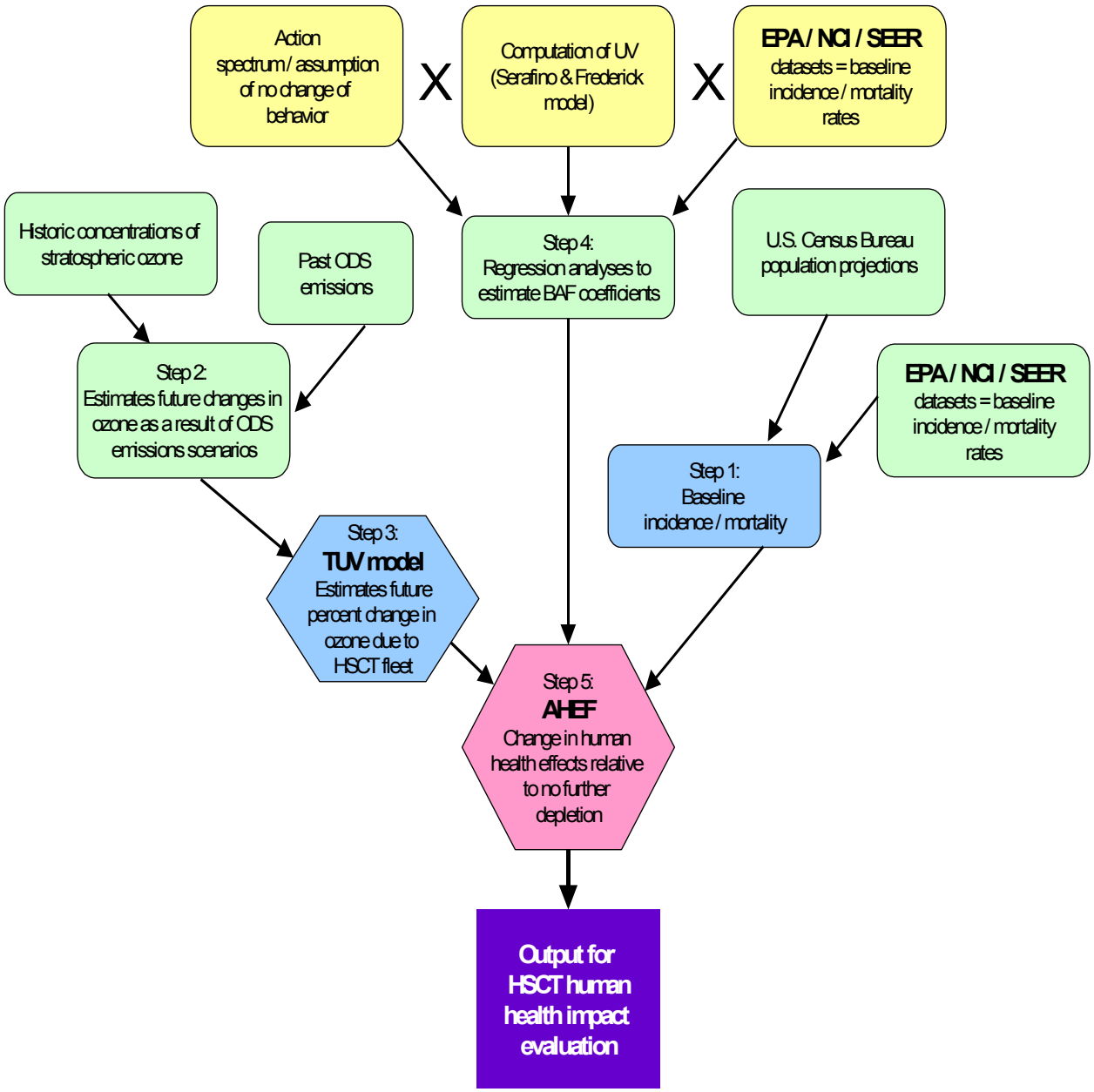

Figure E-1. Relationship between the models used for this evaluation of human health impacts. The symbols used in this diagram do not correspond to traditional flow chart notation. BAF = biological amplification factor. NCI = National Cancer Institute. SEER = Surveillance, Epidemiology, and End Results Program.

The AHEF has five main steps incorporated into the modeling approach. These steps lead to a prediction of incremental changes in incidence and mortality estimates for various UV-related health effects based on ODS emission scenarios. These steps are described below:

Step 1. Compute baseline projections of incidence and mortality assuming no further depletion of the ozone layer.

The AHEF defines an initial estimate of incidence and/or mortality for skin cancer and cataracts that would be expected to occur in the future if the concentration of stratospheric ozone were fixed at 1979-1980 levels (the first two-year period for which satellite measurements of stratospheric ozone exist). This is defined as the "no further depletion" scenario. Future skin cancer and cataracts incidence and skin cancer mortality that would have occurred in the absence of ozone depletion are assumed to be associated with these 1979-1980 ozone concentrations. Establishing these values provides a standard against which to evaluate changes in the mortality and/or incidence of these health effects resulting from future ODS emission scenarios (in the case of Title VI, the "Pre-CAAA" and "Post-CAAA" scenarios). The AHEF performs the following calculations to create initial estimates of incidence and mortality:

- Data on past cases of skin cancer and cataract incidence and mortality are used to derive rates for UV-related health effects in the US population. Rates are based on age, gender, and in some cases, birth year. The historical data was collected from the Surveillance, Epidemiology, and End Results Program (SEER) within the Cancer Control Research Program at the National Cancer Institute (NCI) (Ries *et al*, 1999). The ratio of SEER-based incidence to mortality is calculated and then applied to EPA/NCI mortality rates to generate comprehensive future incidence rates ((Scotto *et al*, 1991) and (Pitcher and Longstreth, 1991)).

- Future US population is estimated by age and gender groupings. Data are gathered from U.S. Census Bureau population projections.

- The number of people in each age and gender group is multiplied by the appropriate incidence and/or mortality rate to produce an estimated baseline number of future skin cancer and cataract cases per year.

Because skin cancer and cataract rates as well as ozone depletion vary across latitudes, the initial US health effects data are stratified into three latitude regions based on specific population estimates from U.S. Census Bureau. Furthermore, because skin cancer incidence and mortality among non-white populations is not well understood in terms of baseline rates of responsiveness to increased UV exposures, currently only white populations are examined in this framework. Once the required information becomes available, non-white populations will be integrated into the model; however, this is not expected to be accomplished within the timeframe of the second prospective analysis.

Step 2. Model the impacts of future emissions of ODS on stratospheric ozone concentrations.

Since 1978, satellites have been providing measurements of stratospheric ozone by latitudinal band. Data from the first of these satellites, the Nimbus 7, indicate that during the satellite's lifespan from 1978 to 1993, ozone measurements have declined in a manner that appears to be related to an increase in the concentration of stratospheric chlorine and bromine. This relationship enables the AHEF to use ODS emissions to predict decreases in stratospheric ozone. First, the model uses regression coefficients to quantify the relationship between past ODS emissions and past changes in ozone concentration, as follows:

- Measurements of historical concentrations of stratospheric ozone are obtained from satellite data.

- The amount and type of past emissions of ODS are combined with the information on each species' degree of dissociation and rate of transport to the stratosphere. Using this information, ODS emissions are expressed in terms of equivalent effective stratospheric chlorine, or EESC, for each year for which satellite-based ozone measurements are available.

- Statistical linear regressions are performed to obtain a measure of the correlation between ODS emissions expressed as EESC and satellite measurements of stratospheric ozone. These regressions are performed by month and by latitudinal band for each year.

To predict future changes in ozone as a result of different ODS emission scenarios (in this case for the "Pre-CAAA" and "Post-CAAA" scenarios), the AHEF converts the hypothesized emissions into EESC and multiplies by the regression coefficients obtained above to estimate future ozone depletion by month and latitude.

Step 3. Estimate changes in ground-level UV based on ozone depletion projections.

After future ozone concentrations have been estimated for a given ODS emissions scenario, future ground-level UV intensities can be calculated. The AHEF uses the results of the Tropospheric Ultraviolet-Visible (TUV) model to predict UV irradiance at ground level (Madronich 1992, 1993b; Madronich and de Gruijl 1993; Madronich *et al.* 1996, 1998). TUV estimates surface UV levels based on total column ozone at different latitudes, the solar zenith angle, the relative weights placed on different portions of the UV spectrum, and other atmospheric characteristics. Thus, the modeling framework can use projected ozone concentrations to calculate the UV dose at any given location and for any given time period (e.g., peak intensity day of the year or the sum of exposures incurred over the entire year). Several studies that test the accuracy of TUV against direct measurements of surface UV levels have been completed (e.g., Shetter *et al.* 1992, 1996; Kirk *et al.* 1994; Lantz *et al.* 1996).

The TUV estimate of the spectral UV irradiance, $F(\lambda, x, t)$, at time t, location x, and wavelength λ, may be represented as the product of the solar spectral irradiance at the top of the atmosphere, $F_{toa}(\lambda)$, and an atmospheric transmission factor, T.

$$F(\lambda, x, t) = F_{toa}(\lambda)\ T(\lambda, x, t; \Theta_0, O_3, \text{clouds, aerosols, } \ldots) \qquad \text{Equation E-1}$$

$F_{toa}(\lambda)$ is based on direct measures of the sun by satellite, balloon, and ground-based instruments. The value of T is impacted by a variety of factors including the solar zenith angle (Θ_0), the earth-sun distance, and a number of atmospheric optical properties (e.g. absorption by ozone, pollutant gases, scattering by air molecules). The calculation may also optionally include values for atmospheric particles such as clouds and aerosols that can affect absorption and scattering. Finally, TUV includes surface reflections, as they can contribute to the radiation incident at the surface (see for example McKenzie *et al.* 1998).

TUV then uses vertical profiles of air density, temperature, and ozone from the United States Standard Atmosphere to calculate the transmission factor (T). The spectral irradiance at any location and time, F(l, x, t), is then calculated by solving for radiative transfer within uniform layers of the atmosphere using an accurate numerical scheme, the discrete ordinates method developed by Stamnes *et al.* (1998) and modified by Madronich *et al.* (1999).

Step 4. Derive dose-response relationships for the incidence and mortality of skin cancer and cataracts from primary data or are obtained from the most up-to-date literature.

When estimating dose-response relationships for human health effects and UV exposure, controversy exists regarding which portion of the spectrum of UV radiation is the best measure of the "dose" an individual receives from the sun. This decision is critical because ozone depletion primarily alters the amount of biologically active UV-B radiation that reaches the ground, leaving the less harmful UV-A portion of the spectrum largely unchanged. In the attempt to quantify this "dose," scientists have created mathematical expressions describing the amount of UV-B and UV-A wavelength radiation that may cause health effects in mice and fish (e.g., DNA damage, skin cancer, and cataract). Based on these studies, a number of different "action spectra," as these weighting schemes are called, have been proposed as predictors of the dose of UV radiation needed to induce skin cancer and cataracts in humans.

Once a particular action spectrum for a human health effect is selected, the second component in developing dose-response relationships for UV exposure requires determination of the degree to which incidence of skin cancer and cataract increases with more intense UV exposure. These dose-response relationships, known as biological amplification factors (BAFs), are usually estimated for cutaneous malignant melanoma (CMM); for the two non-melanoma skin cancers (NMSC), basal cell carcinoma (BCC) and squamous cell carcinoma (SCC); and for cataracts based on actual human incidence and mortality data. Information on skin cancer incidence and mortality rates among populations at different latitudes is combined with the difference in the intensity of UV exposure across those latitudes (e.g., southern latitudes are exposed to higher levels of UV radiation

than northern locations) to estimate BAFs. This latitude gradient can also be used to estimate the way in which skin cancer and cataract incidence and/or mortality will change over time as ozone depletion occurs and UV doses at all latitudes rise.

The current peer-reviewed version of the AHEF uses two action spectra in its estimates of dose-response relationships for UV exposure (U.S. EPA, 2001). The SCUP-h[1] action spectrum is used to predict the dose of UV exposure needed to induce melanoma incidence and mortality as well as NMSC incidence and mortality (de Gruijl *et al.* 1993). The DNA-h action spectrum is used to predict the dose of UV exposure needed to induce cataract incidence (Setlow, 1993). In addition, data on the baseline incidence and mortality rates for human health endpoints are found in EPA, NCI, and/or SEER datasets.

Step 5. Combine the above inputs to project future levels of skin cancer and cataract incidence and skin cancer mortality.

The final step in the modeling framework incorporates the previously discussed inputs to project future incremental skin cancers and cataracts generated under a particular emissions scenario as compared to the no-further-depletion scenario. For all scenarios, the base case relative to which incremental health effects are computed is the future skin cancers and cataracts that would have occurred in the absence of ozone depletion from the 1979-1980 concentrations. Declines in future health effects due to tightening ODS emissions reduction targets are then calculated as the difference between the Pre-CAAA and Post-CAAA scenarios. Benefits will be calculated over the period of 1990 to 2165, to reflect the long time period during which stratospheric ozone depletion occurs and the health effects become manifest in the population.

The final step begins when the model calculates the future annual percentage change in UV dose for a given action spectrum across the three latitudinal bands of interest. Multiplying the percentage change in UV exposure in a future year by the appropriate BAF (both specific to a given UV action spectrum) yields the percentage change in future skin cancer incidence and mortality as well as cataract incidence attributable to the future change in ozone concentrations. These percentages are then multiplied by the "no further depletion" incidence and/or mortality for that health effect to obtain the incremental changes in incidence and/or mortality for a particular ODS emission scenario relative to no further ozone depletion.

[1] SCUP-h = Skin Cancer Utrecht Philadelphia action spectrum, adjusted for human skin transmission.

Future updates to the AHEF model

EPA is currently preparing a new version of the AHEF model set for peer review by the end of the current (2003) fiscal year. This version of the model is expected to include the following improvements:

- *The addition of newly developed action spectrum for cataracts by Oriowo et al. (2001).*

- *A CMM weighting scheme for early age exposure.* As discussed above, development of melanoma is more closely associated with UV exposure during childhood than with cumulative UV exposure. In the new version of AHEF the results for CMM mortality using annual and peak day exposures are computed either by weighting all exposures equally over a person's lifetime, or by weighting only the exposures received between age one and age 20. More specifically, the following approach was used for estimating whole life versus early life exposures:
  - *For whole life esposure:* exposures throughtout the individual's lifetime are given equal weighting (i.e., each year's exposure is counted in the results).
  - *For early life exposure:* only exposures received between the ags of one and 20 are considered (i.e., later life exposures do not contribute to the results).

The peer review process for the version of the AHEF model including these improvements is expected to be completed in time to use the newer version of the model in estimating Title VI benefits for the second prospective study.

Uncertainties associated with the AHEF model

There are several important uncertainties associated with the AHEF model. This section describes these uncertainties and, where possible, an estimate of the potential magnitude and expected direction of possible bias. Exhibit E-3 presents a summary of these key uncertainties and their expected effect on the AHEF model estimates.

Exhibit E-3: Factors Contributing to Uncertainty

Factor	Parameter	Bias of Current Estimate
Change in UV Estimates	Atmospheric parameters assumption	unknown
	Long-term Systematic Changes in Atmospheric Opacity (e.g. clouds, aerosols, other pollutants)	unknown
Changes in Health Effect Estimates	Action Spectrum Choice	unknown
	Action Spectrum Derivation	unknown
	Future Population Composition and Size	unknown
	Latency	overestimate
	Changes in Human UV Exposure Behavior	unknown
	Improvements in Medical Care/Increased Longevity	overestimate

There is a small amount of uncertainty introduced in the TUV model (Step 3). The uncertainty stems from the assumption that relative to the specific change in ozone all other factors (air pollution, cloud cover, etc.) remain constant. The direction of the bias created by this uncertainty is unknown, however it is expected to be a small effect compared to other uncertainties associated with the AHEF methodology.

The composition of the future atmosphere is unknown. Impacts from ODS phaseout scenarios as well as future climate changes could result in increases in atmospheric water vapor and cloud cover. In addition the impact of global warming on future atmospheric composition is unknown. These factors introduce an unknown factor of uncertainty into the model estimates of cataract incidence and skin cancer incidence and/or mortality.

A degree of uncertainty is present in the estimates of the dose-response relationships or BAFs. This uncertainty stems from the assumption that the SCUP-h action spectrum is an adequate predictor of NMSC in addition to the CMM for which it was developed. The BAFs developed for SCC and BCC using SCUP-h are estimated at 2.5 +/- 0.7 and 1.4 +/- 0.4 respectively (de Gruijl *et al.* 1993, Longstreth *et al.* 1998). The degree of uncertainty associated with these BAF estimates may also be expressed as a range of approximately 30 percent.

An additional factor of uncertainty is associated with the action spectra, stemming from the laboratory techniques and instrumentation used for their derivation. The potential for inconsistencies between the wavelengths of UV received by the subject and the intended wavelengths can affect the measured result, sometimes by orders of magnitude. While the potential for a large uncertainty is present, we cannot predict in what direction the bias would occur, as the wavelengths received could be greater or less than those measured. Also, the action spectra are estimated using monochromatic light sources, which are not fully representative of the polychromatic light received directly from the sun.

Steps 1 and 4 of the model incorporate future population predictions from the U.S. Census Bureau. AHEF uses estimates of population grouped by race, gender, age, and location. The Census

Bureau does present an uncertainty factor of +/- 0.1% for its overall population estimates, however a similar estimate of uncertainty is not predicted for the population groups used in AHEF. There is a possibility that the uncertainty factors for each of these groups could be more or less than the overall estimate, therefore it is not useful to apply the overall factor to the population groupings used in the AHEF model.

In the case of the AHEF model, latency refers to the lag time between UV exposure and the manifestation of a given health effect. Both skin cancer latency and early life exposure have been identified as potential risk factors associated with increased susceptibility to CMM. Skin cancer latency is a potential risk factor because the manifestation of skin cancer may not appear for a length of time, during which a person may continue practices that expose the skin to harmful UV. However, the current peer-reviewed version of the AHEF does not model lag time due to difficulties caused by the limited state of knowledge about latency and its mechanisms prevailing at the time of the peer review (Madronich, 1999). Therefore, a quantitative estimate of this source of uncertainty is unavailable. If there is a significant lag and it is not included in the model, then benefits are likely overestimates since the benefit stream has not been properly discounted.

In the case of the proposed weighting scheme for early age exposure, there is uncertainty associated with the timing of the incremental effects and who will bear them. More specifically, for the cumulative lifetime exposure assumption, the risks of ozone depletion are borne primarily by the present population of adults who will experience these health effects as they age. It is children and future generations who will experience increased early life UV exposures and the associated incremental health effects later in their lives. It should be noted that this shift of health risks does not reflect a formal modeling of CMM latency, which would involve an elaborate method for assigning different weights to exposures incurred at different ages or some other yet-to-be-developed approach.

There are a number of factors that may have an additional, unknown effect on future incidence and/or mortality rates for skin cancer and cataracts. These include future population composition and size, future UV exposure behavior, improvements in medical care and predictions of increased longevity. The expected increase in Hispanic populations could lead to a decrease in incidence and mortality rates due to the higher pigmentation found in these populations. The bias of future UV exposure is more difficult to predict and innovations and increased awareness could lead to decreased exposure to UV, however an increased sense of protection could lead to longer periods of exposure, thus negating the positive effects of sunglasses and sunscreens.

However, most of these confounding factors are assumed to be constant in the AHEF model. Therefore sunbathing frequency and attire, the use of sunscreens and sunglasses, the detection and treatment capacities of the medical system, and other conditions are assumed to remain unchanged in the future. The health effects modeling does account for the gradual shift from outdoor work earlier in the last century to factory and office occupations through the 1950s, and it does take into account the evolving demographics of the United States over time. It remains true that significant

changes in UV-related behavior patterns could generate outcomes different from those predicted by the existing model.

Benchmarking the results of a model against available observed data can help to predict the accuracy of the model as well as determine the direction of potential biases. The majority of inputs to the AHEF model are derived statistically using real data (e.g., EESC to ozone, BAFs), therefore calibration is not an issue. The results of the model, however, have yet to be benchmarked against observed data. Several activities can be undertaken to assess actual column ozone measurements compared to AHEF predictions, though all will require substantial time and monetary commitments. First, the AHEF output of column ozone concentrations (in Dobson units) by year and latitudinal band can be compared to observed data, as available by region. Second, ground level UV monitoring can be obtained and assessed to help improve modeling estimates, particularly in urban areas. It should be noted that the AHEF model and all of its individual inputs have been peer-reviewed.

MAJOR COMMENTS ON STRATOSPHERIC OZONE ANALYSIS FROM FIRST PROSPECTIVE STUDY

This section lists the major comments from SAB and others on the Title VI analysis performed for the first prospective and discusses how the approach to Title VI benefits and costs for the second prospective will address these issues.

1. Incorporate tropospheric ozone concentration reductions expected under other regulations as negative benefits.

The great majority of shielding provided by the ground level ozone against the harmful effects of UV-b radiation results from naturally occurring ozone in the stratosphere, but the 10 percent of total "column"ozone present in the troposphere also contributes (NAS, 1991). A variable portion of this tropospheric fraction of UV-B shielding is derived from ground level or "smog" ozone related to anthropogenic air pollution. Therefore, strategies that reduce ground level ozone could, in some small measure, increase exposure to UV-B from the sun.

While EPA's analyses demonstrate it is possible to provide quantitative estimates of benefits associated with globally based strategies to restore the far larger and more spatially uniform stratospheric ozone layer, the changes in UV-B exposures associated with ground level ozone reduction strategies are much more complicated and uncertain. Smog ozone strategies, such as mobile source controls, are focused on decreasing peak ground level ozone concentrations, and it is reasonable to conclude that they produce a far more complex and heterogeneous spatial and temporal pattern of ozone concentration and UV-B exposure changes than do stratospheric ozone protection programs. In addition, the changes in long-term total column ozone concentrations are far smaller from ground-level programs. To properly estimate the change in exposure and impacts, it would be necessary to match the spatial and temporal distribution of the changes in ground-level

ozone to the spatial and temporal distribution of exposure to ground level ozone and sunlight. More importantly, it is long-term exposure to UV-B that is associated with effects. Intermittent, short-term, and relatively small changes in ground-level ozone and UV-B are not likely to measurably change long-term risks of these adverse effects.

For all of these reasons, EPA believes we will continue to be unable to provide reliable estimates of the changes in UV-B shielding associated with ground-level ozone changes. This inability lends an upward bias to the net monetized benefits of tropospheric ozone reduction that will be presented in second prospective criteria pollutant analysis. It is likely that the adverse health effects associated with increases in UV-B exposure from decreased tropospheric ozone would, however, be relatively very small from a public health perspective because 1) the expected long-term ozone change resulting from the CAAA is likely to be small in comparison to the sum of total column natural stratospheric and tropospheric ozone; 2) air quality management strategies are focused on decreasing peak ozone concentrations and thus may change exposures over limited areas for limited times; 3) people often receive peak exposures to UV-B in coastal areas where sea or lake breezes reduce ground level pollution concentrations regardless of strategy; and 4) ozone concentration changes are greatest in urban areas and areas immediately downwind of urban areas, where people are more likely to spend most of their time indoors or in the shade of buildings, trees or vehicles.

EPA has also explored this issue recently through collaboration with Dr. Sasha Madronich of the National Center for Atmospheric Research. His methods and data, while somewhat preliminary in nature, result in predictions of health effects from tropospheric ozone decreases that are far lower than those cited by the SAB. In brief, his modeling accounts more realistically for the geographical areas and the seasons in which people receive their UV exposures. These are then compared to realistic estimates of where and when tropospheric ozone depletion might occur. His analysis is thus more credible than others in which tropospheric ozone depletion is assumed to be distributed evenly across the entire nation.

2. Examine the uncertainties in the cost and benefit estimates.

In a recent project that is nearing completion, EPA explored the many sources of uncertainty concerning the health effects predicted by the AHEF. This study was conducted by EPA for NASA's hypersonic commercial transport (HSCT) project. It was extensively peer reviewed by leading atmospheric researchers (U.S. EPA, 2001).

The major sources of uncertainty in the AHEF examined in the HSCT analysis include the following:

- computing projected future stratospheric ozone concentrations from projected EESC,
- calculating ground-level UV based on projected stratospheric ozone concentrations,

- using alternative action spectrum weighting schemes for human health effects, and

- computing health effects using specific action spectra, incremental UV exposure, and the associated BAFs.

The HSCT analysis generated distributions for some pieces of the model (e.g., BAFs and atmospheric regression coefficients) and plus/minus factors for other parts (e.g., stratospheric ozone to ground UV estimation factors). EPA is investigating whether the analyses in the HSCT study can be applied to the AHEF results to be used in the revised 812 study.

3. Include the costs and benefits of new ozone-protection regulations.

The revised 812 study will incorporate current (and anticipated future) stratospheric protection rules, such as the methyl bromide freeze and eventual ban. EPA has developed several RIAs in the past few years that provide a source of cost estimates for rules not included in the first prospective. In addition, emission reductions associated with these rules will be incorporated into the control scenario for the benefits analysis.

4. Standardize the Value of a Statistical Life (VSL) for human health benefits and discounting for costs and benefits.

EPA plans to employ the same VSL estimate used for all mortality benefits from all CAA Titles, including Title VI. We also propose performing a sensitivity test that considers the age distribution of avoided skin cancer mortality and a distribution of VSL by age cohort to generate monetized benefits estimates for avoided mortality.

Adjusting the discount rate for the costs of the various stratospheric ozone protection rules to conform to the three percent discount rate for the primary analysis may be difficult unless the underlying original output information can be retrieved. For recent regulations, these data are expected to be available. For older rules with limited data on components of the benefit and cost streams, EPA will investigate the merits of applying adjustment factors.

5. Revisit Non-Melanoma Skin Cancer (NMSC) Mortality Estimates

Actual data on NMSC mortality have been incorporated in the revised AHEF over the past several years – including baseline incidence by age and gender, and estimated BAFs. This information was not available for the first prospective analysis. The result of using these data is that NMSC mortality is about 60% lower than the original 1% of incidence assumption. Hence, the new version of the AHEF will be used to address this issue.

6. Examine possible noncompliance with projected ODS-controls by other nations.

EPA and ICF recently evaluated a limited number of scenarios involving non-compliance of developing countries with future ODS restrictions to explore the impact of non-compliance on U.S. citizens. The empirical results of these and other simulations will be evaluated for possible incorporation in the second prospective study.

7. Directly measure social costs of ODS controls instead of using ex ante estimates.

As explained in the Appendix G of the first prospective study, EPA presents estimates for Title VI as net present values of the strams of annual costs and benefits due to the long-term nature of the mechanisms of stratospheric ozone depletion and measures taken to avoid depletion. For EPA as a whole, systematic ex post cost or benefits measurement/survey efforts have rarely been undertaken except for very broad categories of costs of pollution control. In EPA's *Cost of Clean* report, the primary source of information was the Census Bureau's MA200 survey of industry pollution control costs. Because these data were so aggregated and because imputing costs to EPA's regulations vs. other regulatory authorities, much less to voluntary expenditures, was not possible, this report and its underlying data are not suitable for estimating the on-going costs of EPA's stratospheric ozone protection efforts.

8. Review the discussion of the interaction between stratospheric ozone recovery and global climate change.

EPA's original calculations of stratospheric chlorine and bromine concentrations associated with changes in emissions incorporated the ability of CFCs, halons, and other ozone depleting chemicals to act as greenhouse gases. The atmospheric chemistry model adjusts column ozone and temperature so that they are consistent with consensus ozone-depleting potential and global warming potential estimates. The model also reflects radiative and chemical feedback from water vapor, ocean absorption, atmospheric circulation effects, and chemical interactions between substances. Some more recent studies have suggested that climate change could have a significant impact on the recovery of stratospheric ozone, beyond what was accounted for in original modeling work. It has been hypothesized, for example, that increased temperatures caused by climate forcing of greenhouse gases and aerosols could cool the stratosphere, thus increasing the time it takes for ozone to recover. At this point, however, little is known about the degree to which climate change may affect stratospheric ozone recovery and conversely, any relationship that may exist between stratospheric ozone recovery and climate change. Unless improved three-dimensional models of atmospheric chemistry and climate processes are developed in a sufficiently timely and rigorous manner, the second prospective study will not attempt to further assess the potential effects of climate change on ozone under alternative scenarios.

9. Update the human health benefits of stratospheric ozone recovery to reflect changes during the last decade in the sciences concerning ODS emissions and ozone depletion/recovery, ozone-to-UV exposures at the ground, and the complex relationship between human UV exposures (duration, action spectrum, latency, and so forth) and skin cancer and cataracts.

As outlined in the previous section, the revised AHEF uses the most up-to-date atmospherics inputs for a 1D model, a revised methyl bromide "alpha factor" of 55 (instead of 40), and a new statistically-estimated set of stratospheric atmospheric EESC-to-ozone depletion parameters. The column ozone-to-UV on the ground model is current (Dr. Madronich's TUV), and various new health effect action spectra and exposure assumptions have all been explored and incorporated into the AHEF. EPA plans to address these issues by re-running the various policy scenarios of interest using the revised AHEF. This is also necessary because recent stratospheric ozone protection rules' benefits have all been estimated using the new modeling framework.

With respect to modeling latency, although the epidemiological literature strongly suggests this may be appropriate, especially for cutaneous malignant melanoma, there is no widely accepted methodology that directly incorporates latency. The approach adopted in the AHEF is normally to assume that it is cumulative lifetime exposure that results in skin cancers of all types. The AHEF can use cumulative peak day exposures instead and it can weight exposures received at different ages unequally. The latter does shift the incremental health effects farther into the future and shifts the bulk of these health effects from individuals living today to future generations, thus roughly simulating a latency relationship. EPA plans to inviestigate the merits of this approximation approach for use in the second prospective study, and seeks the advice of the Council regarding its potential utility and technical merit.

Finally, the incorporation of advances in detection and treatment of skin cancers in recent years would be a very useful task to undertake. Unfortunately, this would require a substantial amount of new data collection and analysis, and the results would probably be difficult to integrate into the current framework of inter-related health effects inputs to the AHEF. The AHEF necessarily relies on historical data to calculate baseline and incremental health effects from UV exposure, so attempting an approximate adjustment based on more recent, and arguably non-comprehensive, information would likely add to the uncertainties of the results rather than reduce them. EPA seeks the advice of the Council regarding this issue.

10. Do the reported Title VI Costs and Benefits Represent World-Wide or U.S.-Specific Costs and Benefits?

The benefits and costs reported in all of the RIAs and related analyses are for the United States only. This analytical scope is consistent with the rest of the second prospective study.

11. Different timeframes for costs and benefits.

The results for stratospheric ozone protection included in the first prospective study reported costs through 2075. In order to capture all of the health benefits of ODS controls between 1985 and 2075, the benefits model must track anyone in the United States who experiences ozone depletion and increased UV up through 2075 until their deaths. Thus, in the extreme, this is 90 years after 2075. Clearly, the incremental incidence and mortality decline as the years get more distant from 2075 because fewer and fewer people are alive who experienced decrease UV up to 2075 relative to depletion. They nevertheless remain beneficiaries of ozone recovery effects prior to 2076 as long as they were alive during those years.

12. Sensitivity of 1D atmospheric modeling to geography and season.

Using a 1D atmospheric modeling framework does not imply that the outputs are insensitive to latitude and month of the year. While it does assume that the EESC is the same world-wide, the statistically-estimated parameters that translate EESC (based on ODS emissions and other factors) into stratospheric ozone concentration estimates are latitude- and month-specific because they were estimated using latitude- and month-specific ozone measurements. Thus, the column ozone estimates are sensitive to latitude and time of year.

Furthermore, the TUV model – which is used for predicting ground-level UV intensities by location and time – is similarly sensitive to latitude and time (indeed, to the hour of the day). Thus, UV intensities at different latitudes are very different due to the angle of the sun and amount of the atmosphere through which the sun's rays travel.

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Appendix F

SUMMARY OF MAJOR ONGOING EPA-FUNDED PROJECTS FOR NITROGEN DEPOSITION ECOLOGICAL EFFECTS VALUATION

Project	Affiliated EPA Office	General Information	URL
Chesapeake Bay Program	Office of Water, and Office of Air Quality Planning and Standards	• The objectives of the Chesapeake Bay Program include collecting information regarding the Bay's environmental quality, and determining appropriate measures to improve the Bay and protect it's multiple resources. • Development of a nitrogen loading model at Chesapeake Bay that may be applied to estuaries nationwide is currently a subject of research under EPA's Great Waters Program. This program is also examining the potential for developing comprehensive integrated models to assess ecological and economic impacts.	http://www.epa.gov/r3chespk/ http://www.chesapeakebay.net/
Waquoit Bay Watershed Ecological Risk Assessment	National Center for Environmental Assessment (NCEA), Office of Water, and Office of Research and Development	• The Waquoit Bay Ecological Risk Assessment intends to predict how changes in land use and human activity in the watershed will impact eelgrass growth in order to enable resource mangers to make decisions based on more information. • Nitrogen deposition is one of the key stressors being evaluated. Specifically, the risk analysis focuses on the relationship between nutrient enrichment and loss of eelgrass habitat, and the resulting effects on scallop abundance in the Bay.	http://cfpub.epa.gov/ncea/cfm/waquoit.cfm?ActType=default
Tampa Bay Atmospheric Deposition Study	Office of Water	• EPA's Great Waters Program and the Tampa Bay Estuary Program are partnering with local environmental agencies to develop a nitrogen loading model for the Bay including examination of the contribution of atmospheric deposition to eutrophication. This model will then be used to develop a cost-benefit study of management options. The Estuary Program anticipates that this loading model will be complete by September of 2003.	http://www.tbep.org/tbep.html http://www.epa.gov/projectxl/tampa/ http://www.hsc.usf.edu/publichealth/EOH/BRACE/TBADS.htm

Project	Affiliated EPA Office	General Information	URL
Albemarle-Pimlico National Estuary Program (APNEP)	Office of Water	• Ongoing research under the APNEP's Comprehensive Conservation and Management Plan addresses multiple water quality and habitat issues within the Estuary. In the past, a review of economic models was conducted to help determine the value of recreational fishing, and measure the impact of increasing the quality of fisheries in the Estuary. This review was applied to a cost-benefit study of the Program's management plan.	http://www.epa.gov/owow/estuaries/programs/aps.htm http://h2o.enr.state.nc.us/nep/
Casco Bay National Estuary Program (CBNEP)	Office of Water, and the National Center for Environmental Economics (NCEE)	• The Casco Bay Air Toxics Deposition Monitoring Program is an ongoing study at the Bay that models seasonal and annual deposition of airborne toxics, including nitrogen. • NCEE has completed an economic profile of the Estuary and determined that the health of the Estuary has a substantial effect on tourism and recreation revenues.	http://www.epa.gov/owow/estuaries/programs/cb.htm
Sarasota Bay National Estuary Program (SBNEP)	Office of Water	• A main focus of the SBNEP is reduction of nitrogen deposition in the Bay. A nitrogen loading model has been developed to determine the impact of atmospheric deposition. This information was used to target management objectives and evaluate the impact of nitrogen reductions on Bay resources. • Ongoing research at the Mote Marine Laboratory focuses on the effects of atmospheric nitrogen deposition at the Bay on algal assemblages.	http://www.sarasotabay.org/
Social and Ecological Transferability of Integrated Ecological Assessment Models	National Center for Environmental Research	• This project is designed to benefit urban/suburban coastal communities interested in protecting estuarine ecosystems from nitrogen loading. The main objective is to build an integrated assessment model using an existing watershed N-loading model and extend an estuarine ecological model to include a new and socially important management endpoint - fish and shellfish. A unique aspect of this project is the opportunity to use data collected to test the ecological transferability of the model as a treatment in the social experiment by involving citizens in data collection. • This research will result in an integrated ecological model of the consequences of coastal land-use change on estuarine systems and, perhaps more importantly, better information on how to apply that model in new environmental and social settings.	http://cfpub.epa.gov/ncer_abstracts/index.cfm/fuseaction/display.abstractDetail/abstract/847/report/0

Appendix G

PARTIAL LITERATURE REVIEW TO SUPPORT CHARACTERIZATION OF ECONOMIC VALUE OF ECOLOGICAL EFFECTS

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May 12, 2003

APPENDIX H

META-ANALYSIS FOR VALUE OF STATISTICAL LIFE

May 12, 2003

An Empirical Bayes Approach to Combining and Comparing Estimates of the Value of a Statistical Life for Environmental Policy Analysis*

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* The authors thank David Higdon for comments on an earlier draft of this manuscript. Please do not cite without permission.

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Abstract

An empirical Bayes pooling method is used to combine and compare estimates of the Value of a Statistical Life (VSL). The data come from 40 selected studies published between 1974 and 2000, containing 196 VSL estimates. The estimated composite distribution of empirical Bayes adjusted VSL has a mean of $5.4 million and a standard deviation of $2.4 million. The empirical Bayes method greatly reduces the variability around the pooled VSL estimate. The pooled VSL estimate is sensitive to the choice of valuation method and study location, but not to the source of data on occupational risk.

Key words: Value of a Statistical Life (VSL), empirical Bayes estimate, environmental policy, health policy, contingent valuation method, hedonic wage method

JEL subject category number: J17, C11, Q28

The value of a statistical life is one of the most controversial and important components of any analysis of the benefits of reducing environmental health risks. Health benefits of air pollution regulations are dominated by the value of premature mortality benefits. In recent analyses of air pollution regulations (United States Environmental Protection Agency (USEPA), 1999), benefits of reduced mortality risks accounted for well over 90 percent of total monetized benefits. The absolute size of mortality benefits is driven by two factors, the relatively strong concentration-response function, which leads to a large number of premature deaths predicted to be avoided per microgram of ambient air pollution reduced, and the value of a statistical life (VSL), estimated to be about \$6.3 million[1]. In addition to the contribution of VSL to the magnitude of benefits, the uncertainty surrounding the mean VSL estimate accounts for much of the measured uncertainty around total benefits. Thus, it is important to obtain reliable estimates of both the mean and variance of VSL.

The VSL is the measurement of the sum of society's willingness to pay (WTP) for one unit of fatal risk reduction (i.e. one statistical life). Rather than the value for any particular individual's life, the VSL represents what a whole group is willing to pay for reducing each member's risk by a small amount (Fisher et al. 1989). For example, if each of 100,000 persons is willing to pay \$10 for the reduction in risk from 2 deaths per 100,000 people to 1 death per 100,000 people, the VSL is \$1 million (\$10 × 100,000). Since fatal risk is not directly traded in markets, non-market valuation methods are applied to determine WTP for fatal risk reduction. The two most common methods for obtaining estimates of VSL are the revealed preference approach including hedonic wage and hedonic price analyses, and the stated preference approach including contingent valuation, contingent ranking, and conjoint methods. EPA does not conduct

original studies but relies on existing VSL studies to determine the appropriate VSL to use in its cost-benefit analyses. The primary source for VSL estimates used by EPA in recent analyses has been a study by Viscusi (1992). Based on the VSL estimates recommended in this study, EPA fit a Weibull distribution to the estimates to derive a mean VSL of $6.3 million, with a standard deviation of $4.2 million (U.S. EPA, 1999).

We extend Viscusi's study by surveying recent literature to account for new VSL studies published between 1992 and 2001. This is potentially important because the more recent studies show a much wider variation in VSL than the studies recommended by Viscusi (1992). The estimates of VSL reported by Viscusi range from 0.8 to 17.7 million. More recent estimates of VSL reported in the literature range from as low as $0.1 million per life saved (Dillingham, 1985), to as high as $87.6 million (Arabsheibani and Marin, 2000). Careful assessment is needed to determine the plausible range of VSL, taking into account these new findings.

There are several potential methods that can be used to obtain estimates of the mean and distribution of VSL. In a study prepared under section 812 of the Clean Air Act Amendments of 1990 (henceforth called the EPA 812 report), it was assumed that each study should receive equal weight, although the reported mean VSL in each study differs in its precision. For example, Leigh and Folson (1984) estimate a VSL of $10.4 million with standard error of $5.2 million, while Miller (1997) reports almost the same VSL ($10.5 million) but with a much smaller standard error ($1.5 million)[2]. As Marin and Psacharopoulos (1982) suggested, more weight should be given to VSL estimates that have smaller standard errors.

Our focus is to develop a more statistically robust estimate of the mean and distribution of VSL using the empirical Bayes estimation method in a two-stage pooling model. The first stage groups individual VSL estimates into homogeneous subsets to provide representative

sample VSL estimates. The second stage uses an empirical Bayes model to incorporate heterogeneity among sample VSL estimates. This approach allows the overall mean and variance of VSL to reflect the underlying variability of the individual VSL estimates, as well as the observed variability between VSL estimates from different studies. Our overall findings suggest the empirical Bayes method provides a pooled estimate of the mean VSL with greatly reduced variability. In addition, we conduct sensitivity analyses to examine how the pooled VSL is affected by valuation method, study location, source of occupational risk data and the addition of estimates with missing information on standard errors. This sensitivity analysis allows us to systematically compare VSL estimates to determine how they are influenced by study design characteristics.

1. Methodology

1.1 Study selection

We obtained published and unpublished VSL studies by examining previously published meta-analysis or review articles, citations from VSL studies and by using web searches and personal contacts.

The data were prepared as follows. First, we selected qualified studies based on a set of selection criteria applied in Viscusi (1992). Second, we computed and recorded all possible VSL estimates and associated standard errors in each study. Third, we made subsets of homogeneous VSL estimates and calculated the representative VSL for each subset by averaging VSLs and their standard errors[3]. Each step is discussed in detail below.

Since the empirical Bayes estimation method (pooled estimate model) does not control for the overall quality of the underlying studies, careful examination of the studies is required for

selection purposes. In order to facilitate comparisons with the EPA 812 report, we applied the same selection criteria that were applied in that report, based largely on the criteria proposed in Viscusi (1992).

Viscusi (1992) examined 37 hedonic wage (HW), hedonic price (HP) and contingent valuation (CV) studies of the value of a statistical life, and listed four criteria for determining the value of life for policy applications. The first criterion is the choice of risk valuation method. Viscusi (1992) found that all the HP studies evaluated failed to provide an unbiased estimate of the dollar side of the risk-dollar tradeoff, and tend to underestimate VSL. Therefore only HW studies and CV studies are included in this study.

The second criterion is the choice of the risk data source for HW studies. Viscusi argues that actuarial data reflect risks other than those on the job, which would not be compensated through the wage mechanism, and tend to bias VSL downward. Therefore some of the initial HW studies that used actuarial data are removed from this analysis. The third criterion is the model specification in HW studies. Most studies apply a simple regression of the natural log of wage rates on risk levels. However, a few of the studies estimate the tradeoff for discounted expected life years lost rather than simply risk of death. This estimation procedure is quite complicated, and the VSL estimates tend to be less robust than in a simple regression estimation approach. Only studies using the simple regression approach are used in this analysis.

The fourth criterion is the sample size for CV studies. Viscusi argues that the two studies he considered whose sample sizes were 30 and 36 respectively were less reliable and should not be used. In this study, a threshold of 100 observations was used as a minimum sample size[4].

There are several other selection criteria that are implicit in the 1992 Viscusi analysis[5]. The first is based on sample characteristics. In the case of HW studies, he only considered studies that examined the wage-risk tradeoff among general or blue-collar workers. Some recent studies only consider samples from extremely dangerous jobs, such as police officer. Workers in these jobs may have different risk preferences and face risks much higher than those evaluated in typical environmental policy contexts. As such, we exclude those studies to prevent likely downward bias in VSL relative to the general population. In the case of CV studies, Viscusi only considered studies that used a general population sample. Therefore we also exclude CV studies that use a specific subpopulation or convenience sample, such as college students.

The second implicit criterion is based on the location of the study. Viscusi (1992) considered only studies conducted in high income countries such as U.S., U.K. and Japan. Although there are increasing numbers of CV or HW studies in developing countries such as Taiwan, Korea and India, we exclude these from our analysis due to differences between these countries and the U.S. Miller (2000) found that income level has a significant impact on VSL, and because we are seeking a VSL applicable to U.S. policy analysis, inclusion of VSL estimates from low-income countries may bias VSL downward. In addition, there are potentially significant differences in labor markets, health care systems, life expectancy, and preferences for risk reductions between developed and developing countries. Thus, our analysis only includes studies in high-income OECD member countries[6]. Finally, our analysis only uses studies that estimate people's WTP for immediate risk reduction due to concerns about comparisons between risks with long latency periods with inherent discounting or uncertainty about future baseline health status.

1.2 Data preparation

In VSL studies, authors usually report the results of a hedonic wage regression analysis, or WTP estimates derived from a CV survey. In the studies we reviewed, a few authors reported all of the VSL that could be estimated based on their analysis, but most authors reported only selected VSL estimates and provided recommended VSL estimates based on their professional judgment. This judgment subjectively takes into account the quality of analysis, such as the statistical significance of the result, the target policy to be evaluated, or judgments based on comparative findings. Changes in statistical methods and best practices for study design during the period covered by our analysis may invalidate the subjective judgments used by authors to recommend a specific VSL. To minimize potential judgment biases, as well as make use of all available information, we re-estimate all possible VSLs based on the information provided in each study and included them in our analysis as long as they met the basic criteria laid out by Viscusi (1992)[7]. For certain specifications some authors found a negative VSL. However, in every case the authors rejected the plausibility of the negative estimates. We agree that negative VSL are highly implausible and exclude them from our primary data set. However, we do present sensitivity analysis showing the effects of excluding the negative estimates.

Estimation of VSL from HW studies

Most of the selected HW studies use the following equation to estimate the wage-risk premium:

$$LnY_i = a_1 p_i + a_2 q_i + a_3 p_i^2 + X_i\beta + \varepsilon_i \quad (1)$$

where Y_i is equal to earnings of individual i, p_i and q_i are job related fatal and non-fatal risk faced by i (q_i often omitted), X_i is a vector of other relevant individual and job characteristics (plus a

constant) and ε_i is an error term. In many cases, the wage equation will also include fatal risk squared and interactions between risk and variables such as union status. Based on equation (1), the VSL is estimated as follows.

$$VSL = (dlnY/dp_i) \times mean\ annual\ wage^{8} \times unit\ of\ fatal\ risk^{9} \qquad (2)$$

Note that $dlnY/dp_i$ may include terms other than a_1 if there are squared or interaction terms.

VSL is usually evaluated at the mean annual wage of the sample population. The unit of fatal risk is the denominator of the risk statistic, i.e. 1000 if the reported worker's fatal risk is 0.02 per 1000 workers. If there is an interaction term between fatal risk and human capital variables such as "Fatal Risk" × "Union Status", the VSL is evaluated at the mean values of the union status variable. If there is a squared risk term, the VSL is evaluated at the mean value of fatal risk.

Estimation of standard error of VSL from HW studies

The standard error of the VSL (SE(VSL)) from a HW study is $Var(VSL) = \left(unit\ of\ risk\right)^2 Var\left(\partial \ln Y/\partial p \times \overline{Y}\right)$, where $\overline{Y}$ is the average wage for the sample. For example, if the wage equation is specified as $LnY = a_1 p_i + a_2 q_i + a_3 p_i^2 + a_4 p_i UNION + \varepsilon_i$, then

$$Var(VSL) = \left(unit\ of\ risk\right)^2 \left[Var\left(a_1\overline{Y}\right) + 4Var\left(a_2\overline{p}\,\overline{Y}\right) + Var\left(a_3\overline{Y}\,\overline{UNION}\right) + 2Cov\left(a_1\overline{Y}, a_2\overline{p}\,\overline{Y}, a_3\overline{Y}\,\overline{UNION}\right)\right]$$

To calculate the full variance, allowing for the observed variability in wages and fatal risk, one needs to calculate the variance of the product of the regression coefficients and the wage, risk, and interaction terms. We use the formula for the exact variance of products provided by Goodman (1960). For the first variance term above, this formula would be

$$Var\left(a_1\overline{Y}\right) = \overline{Y}^2 \frac{s^2\left(a_1\right)}{n} + a_1^2 \frac{s^2\left(\overline{Y}\right)}{n} - \frac{s^2\left(a_1\right)s^2\left(\overline{Y}\right)}{n^2}$$

Most of the studies included in our analysis do not report the variance of annual wage or the covariance matrix (either for the parameter estimates or the variables), so we calculated the standard error of VSL based on the available information, usually consisting of the standard errors of the estimated parameters of the wage equation. In this case the variance formula reduces to

$$Var(VSL) = \left(unit\ of\ risk\right)^2\left[\overline{Y}^2 Var\left(a_1\right) + 4\overline{p}^2\overline{Y}^2 Var\left(a_2\right) + \overline{Y^2}\,\overline{UNION^2} Var\left(a_3\right)\right]$$ To assess the impact of treating mean annual wage as a constant, we estimate the standard error with and without the wage variance for the 45 VSL estimates for which information on the variance of wage was available. We find that the differences between the two estimates of standard error are fairly small, within $0.2 million for most estimates. In no case does the standard error differ by more than 10 percent. We also assess the impact of omitting the covariance term by comparing the reported standard error of Scotton and Taylor (2000) providing a "full" variance estimate for the estimated VSL with our estimated standard error, which does not include the covariance term. We find that the difference in standard error is quite small. Note that the published standard error from this study treats mean annual wage as fixed, so the comparison shows only the effect of excluding the covariance term. These results suggest the impact of omitting the covariance terms and treating mean annual wage as fixed in our calculation of standard errors should not have a significant effect on our results.

Estimation of VSL and standard error from CV studies

For most of the CV surveys, we could not estimate the VSL and its standard error unless the author provided mean or median WTP and a standard error for a certain amount of risk reduction. When this information is available, the VSL and its standard error are simply

calculated as WTP divided by the amount of risk reduction, and SE(WTP) divided by the amount of risk reduction, respectively.

Estimation of representative VSL for each study

Most studies reported multiple VSL estimates. For the empirical Bayes approach, which we use in our analysis, each estimate is assumed to be an independent sample, taken from a random distribution of the conceivable population of studies. This assumption is difficult to support given the fact that there are often multiple observations from a single study. To solve this problem, we constructed a set of homogeneous (and more likely independent) VSL estimates by employing the following approach.

We arrayed individual VSL estimates by study author (to account for the fact that some authors published multiple articles using the same underlying data). We then examined homogeneity among sub-samples of VSL estimates for each author by using Cochran's Q-statistics. The test statistic Q is the sum of squares of the effect about the mean where the i_{th} square is weighted by the reciprocal of the estimated variance. Under the null hypothesis of homogeneity, Q is approximately a χ^2 statistic with n -1 degrees of freedom (DerSimonian and Laird, 1986). If the null hypothesis was not rejected, we take the average of the VSL for the subset and the standard error to estimate the representative mean VSL for that author.

If the hypothesis of homogeneity was rejected, we further divided the samples into subsets according to their different characteristics such as source of risk data and type of population (i.e. white collar or blue collar), and tested for homogeneity again. We repeated this process until all subsets were determined to be homogeneous.

1.3 The empirical Bayes estimation model

In general, the empirical Bayes estimation technique is a method that adjusts the estimates of study-specific coefficients (β's) and their standard errors by combining the information from a given study with information from all the other studies to improve each of the study-specific estimates. Under the assumption that the true β's in the various studies are all drawn from the same distribution of β's, an estimator of β for a given study that uses information from all study estimates is generally better (has smaller mean squared error) than an estimator that uses information from only the given study (Post et al. 2001).

The empirical Bayes model assumes that

$$\beta_i = \mu_i + e_i \qquad (6)$$

where β_i is the reported VSL estimate from study *i,* μ_i is the true VSL, e_i is the sampling error and $N(0, s_i^2)$ for all i = 1,…, n. The model also assumes that

$$\mu_i = \mu + \delta_i \qquad (7)$$

where μ is the mean population VSL estimate, δ_i captures the between study variability, and $N(0, \tau^2)$, τ^2 represents both the degree to which effects vary across the study and the degree to which individual studies give biased assessments of the effects (Levy et al., 2000; DerSimonian and Laird, 1986).

The weighted average of the reported β_i is described as μ_w. The weight is a function of both the sampling error (s_i^2) and the estimate of the variance of the underlying distribution of β's (τ^2). These are expressed as follows;

$$\mu_w = \frac{\sum w_i^* \beta_i}{\sum w_i^*} \qquad (8)$$

$$s.e.\ (\mu_w) = (\sum w_i^*)^{-1/2} \qquad (9)$$

where $w_i^* = \dfrac{1}{w_i^{-1} + \tau^2}$ and $w_i = \dfrac{1}{s_i^2}$

τ^2 can be estimated as

$$\tau^2 = max \left(0, \left(\frac{(Q-(n-1))}{\sum w_i - \frac{\sum w_i^2}{\sum w_i}} \right) \right) \qquad (10)$$

where $Q = \sum w_i (\beta_i - \beta^*)^2$ (Cochran's Q-statistic) and $\beta^* = \dfrac{\sum w_i \beta_i}{\sum w_i}$

The adjusted estimate of the β_i is estimated as

$$Adjusted\ \beta_i = \frac{\frac{\beta_i}{e_i} + \frac{\mu_w}{\tau^2}}{\frac{1}{e_i} + \frac{1}{\tau^2}} \qquad (11)$$

This adjustment, as illustrated in Figure 1, pulls the reported estimates of β_i towards the pooled estimate. The more within-study variability, the less weight the β_i receives relative to the pooled estimate, and the more it gets adjusted towards the pooled estimate. The adjustment also reduces the variance surrounding the β_i by incorporating information from all β's into the estimate of β_i. (Post et al. 2001). In our analysis, β_i corresponds to the VSL of the *i*th study.

In order to visually compare the distributions, we used kernel density estimation to develop smooth distributions based on the empirical Bayes estimate. The kernel estimation provides a smoother distribution than the histogram approach. The kernel estimator is defined by $f(x) = \dfrac{1}{nh} \sum_{i=1}^{n} K\left(\dfrac{x - X_i}{h} \right)$. The kernel function, $\int_{-\infty}^{\infty} K(x)dx = 1$, is usually a symmetric probability density function, e.g. the normal density, and *h* is window width. The kernel function K determines the shape of the bumps, while *h* determines their width. The kernel estimator is a

sum of 'bumps' placed at observations and the estimate *f* is constructed by adding up the bumps (Silverman 1986). We assumed a normal distribution for K and a window width *h* equal to 0.7, which was wide enough to give a reasonably smooth composite distribution while still preserving the features of the distribution (e.g. bumps). The choice of window width is arbitrary, but has no impact on the statistical comparison, which is described below.

To compare the different distributions of VSL, we applied the bootstrap method, which is a nonparametric method for estimating the distribution of statistics. Bootstrapping is equivalent to random sampling with replacement. The infinite population that consists of the n observed sample values, each with probability 1/n, is used to model the unknown real population (Manly 1997). We first conducted re-sampling 1000 times, and compared the distributions in terms of mean, median and interquartile range.

2. Results and sensitivity analyses

In total, we collected 47 HW studies and 29 CV studies. A data summary for each stage of analysis is shown in Table 1. After applying the selection criteria outlined in section 2.1, there were 31 HW studies and 14 CV studies left for the analysis. In our final list, there are 22 new studies published between 1990 and 2000. We re-estimated all possible VSL for the selected studies, and obtained 232 VSL estimates.[10] [11] There were 23 VSL estimates from five studies for which standard errors were not available, and thus they are excluded from our primary analysis, although we examine the impact of excluding those studies in a sensitivity analysis. After testing for homogeneity among sub-samples, we obtained 60 VSL subsets, and estimated a representative VSL and standard error for each subset. Finally, we applied the empirical Bayes method and obtained an adjusted VSL value for each subset.

It is worthwhile to note how the empirical Bayes approach reduces the unexplained variability among VSL estimates. Our 196 VSL estimates show an extremely wide range from \$0.1 million to \$95.5 million with a coefficient of variation of 1.3 (in 2000 constant dollars). The VSL estimates from the 60 subsets range from \$0.3 million to \$43.1 million with a coefficient of variation of 1.2, and the adjusted VSL estimates range from \$0.7 million to \$13.9 million with a coefficient of variation of 0.4.

2.1 The distribution of VSL

Figure 2 shows the kernel density estimates of the composite distribution of the empirical Bayes adjusted VSL (using the 60 representative VSL estimates) and the Weibull distribution for the 26 VSL estimates as reported in the EPA 812 report. The summary results are shown in Table 2. The composite distribution of adjusted VSL has a mean of \$5.4 million

with a standard error of $2.4 million. This mean value is smaller than that based on the EPA 812 Weibull distribution and has less variance (EPA 812's coefficient of variation is 0.7) even though our VSL sample has a range more than five times as wide as the EPA 812 sample.

2.2 Sensitivity analyses

2.2.1 Sensitivity to choice of valuation method

Many researchers argue that the VSL is sensitive to underlying study characteristics (Viscusi 1992, Carson, et al. 2000, Mrozek and Taylor 2002). One of the most interesting differences is in the choice of valuation method. To determine if there is a significant difference between the empirical Bayes adjusted distributions of VSL using HW and CV estimates, we used bootstrapping to test the hypothesis that HW and CV estimates of VSL are from the same underlying distribution.

We divided the set of VSL studies into HW and CV and applied the homogeneity subsetting process and empirical Bayes adjustment method to each group. The kernel density estimates of the distributions for HW and CV sample are shown in Figure 3. The HW distribution has a mean value of $9.4 million with a standard error of $4.7 million while the CV distribution has much smaller mean value of $2.8 million with a standard error of $1.3 million (see Table 2). Bootstrap tests of significance show the VSL based on HW is significantly larger than that of CV ($p<0.001$), comparing means, medians and interquartile ranges between the distributions.

2.2.2 Sensitivity to study location

Because of differences in labor markets, health care systems, and societal attitudes towards risk, VSL estimates from HW studies may potentially be sensitive to the country in which the study was conducted (this may also be true for CV studies, however there were too few CV estimates to conduct similar comparisons). Empirical Bayes estimation was applied to HW samples from the U.S. and U.K separately. (Comparisons with Canada and Australia were not conducted because of small sample sizes for those countries.) The distribution for the U.S. sample has a mean value of $8.5 million with a standard error of $4.9 million, while the distribution for the U.K. sample has a mean value of $22.6 million with a standard error of $4.9 million. Bootstrap tests of significance show that the U.S. estimates are significantly different from UK estimates based on comparing means and medians between distributions.

2.2.3 Sensitivity to source of occupational risk data

Moore and Viscusi (1988) found that VSL was sensitive to choice of source of occupational risk data. According to their results, the VSL estimated based on Bureau of Labor Statistics (BLS) death-risk data is significantly smaller than that estimated based on National Institute of Occupational Safety and Health (NIOSH) death risk data. We estimated the empirical Byes adjusted VSL distribution for each risk data source, and we did not find a significant difference between the two distributions. However, the reliability of our result is limited due to the small number of studies based on the BLS risk data.

2.2.4 Sensitivity to excluded VSL estimates

We also examined the sensitivity of our results to excluded estimates. To do this, we added to the sample the VSL estimates that were excluded from the primary analysis due to the lack of a standard error. We assumed for this test that all reported VSL estimates should have passed at least a 95 percent significance test, and estimate the corresponding standard error at this significance level for each VSL. This added nine averaged VSL estimates to the set of 60 representative estimates, including four estimates from HW studies and five from CV studies.

The distribution of the enhanced sample has a mean value of $4.7 million with a standard error of $2.2 million. Compared with the result of our main analysis, the mean value is reduced by $0.7 million. This is because we have added more estimates from CV, which tends to produce relatively lower VSL. Bootstrap tests of significance show the VSL from HW studies is still significantly different from that from CV studies ($p<0.0001$), comparing means, medians and interquartile ranges.

We also report a 5% trimmed mean that increases the combined mean from both valuation methods from $5.4 million to $5.8 million with no effect on the coefficient of variation. Finally, we consider the impact of including negative estimates. Since these estimates were all associated with HW studies, the HW mean drops from $9.4 million to $6.6 million. This also has a noticeable effect on the combined mean dropping it from $5.4 million to $4.1 million. The difference between the CV and HW estimates remains significant based on bootstrap tests of the means and medians.

3. Conclusions

The meta analysis we have used results in a composite distribution of empirical Bayes adjusted VSL with a mean of $5.4 million and a standard deviation of $2.4 million. This is a somewhat lower mean than previous pooled estimates, and because of the Bayesian adjustment process, there is greatly reduced variability as evidenced by the coefficient of variation even though our dataset has a much wider range than previous studies.

Starting from a baseline of the literature used in Viscusi (1992), our approach has generated a set of hypotheses that may challenge some previously held assumptions. It is clear that VSL analysts need to look closely at study location; our estimates show significant differences in VSL even between developed countries with relatively similar income levels. It is also important to look at valuation method as we found quite different VSL estimates in the hedonic wage versus contingent valuation datasets. Our finding that the hedonic method generates significantly larger estimates than the CV approach is consistent with a comparison of CV and revealed preference approaches to valuing quasi-public goods reported by Carson (1996).

Theoretically, the two valuation methods should not necessarily provide the same results because the HW approach is estimating a local trade-off, while the CV approach approximates a movement along a constant expected utility locus (Viscusi and Evans 1990, Lanoie, Pedro and Latour 1995). However, the impact and direction of this difference had not been systematically investigated prior to this analysis

Our sensitivity analysis found no significant difference on average in the VSL estimates between studies using BLS or NIOSH data. Additional research into appropriate measures of risk is needed. Recent work by Black (2001) suggests that measurement errors in

estimates of fatal risk can lead to large downward biases in estimates of VSL.

Aggregate level comparisons as we have done in this paper are useful in comparing the overall distribution of VSL estimates from each method, however the resulting comparisons might be significantly affected by differences in the design of each study, as the large variance in the HW distribution suggests. This problem could be addressed by applying meta-regression analysis, which can determine the impact of specific study factors by taking into consideration study characteristics such as sample population, study location, or sources of risk data (Levy et al., 2000; Mrozek and Taylor, 2002; Viscusi and Aldy, 2002).

Study location does seem to matter, but additional investigation is necessary to identify why there are differences. Simply lumping countries together as developed or developing may not be the best way to account for potential differences in VSL. Differences in health care system may be a potential factor, as there are a number of differences in insurance coverage and access to health care across developed countries (Anderson and Hussey, 2000). There may be numerous other socio-cultural factors that can cause VSL estimates to diverge.

As the excluded studies sensitivity analysis indicates, our results are sensitive to the addition of small magnitude VSL estimates with low variances. For example, Krupnick et al. (2000) estimated the VSL as $1.1 million with a standard error of $0.05 million. If we remove this estimate from our main analysis, the overall mean VSL is increased to $5.9 million, implying that one study reduces the overall mean by $0.5 million. It is thus especially important to determine the reliability of CV studies very carefully by assessing any potential questionnaire and scope effects (Hammitt and Graham, 1999). Also, it may be important to investigate why the VSL estimates from CV studies are so similar despite the differences in type of risk, study location and survey method.

In addition to the application of the empirical Bayes method, our analysis demonstrates the importance of adopting a two-stage procedure for combining evidence from the literature when multiple estimates are available from a single source of data. The first stage sorting process using the Cochran's Q test for homogeneity seems a reasonable approach to control for over-representation of any one dataset. From the original set of 40 studies, we obtained 196 VSL estimates and then classified these into 60 homogeneous subsets. This suggests that there was a high probability of assigning too much weight to some estimates if a single stage process were used, treating each of the 196 estimates as independent. Also, the two-stage approach does not discard information from each study. Instead it uses all the available information in an appropriate manner.

As in the field of epidemiology, the economics profession should consider developing protocols for combining estimates from different studies for policy purposes. Consistent reporting of both point estimates of VSL and standard errors, or variance-covariance matrices would enhance the ability of future researchers to make use of all information in constructing estimates of VSL for policy analysis. Additional research is needed to understand how VSL varies systematically with underlying study attributes, such as estimation method or location of studies. The empirical Bayes approach outlined here provides a useful starting point in developing the variables needed for such studies.

The widely cited pooled estimate of $6.3 million from the EPA 812 study based on Viscusi's assessment of the VSL literature was derived from a simple histogram method. This early approach ignored within and between study variability. Mrozek and Taylor presented an alternative method for deriving a mean VSL estimate for policy purposes based on a best fit regression model using only the hedonic wage studies. . We examine both CV and HW studies

and present a different methodology using all available information to adjust individual VSL estimates based on the within and between study variability. By generating distributions of VSL, the method allows us to test individual hypotheses regarding study attributes. These comparisons have generated a number of hypotheses that should form the foundation for future meta-analyses of VSL combining the CV and HW approaches.

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Table 1. VSL Data Summary

	HW	CV	Total
Number of collected studies	47	29	76
Number of selected studies	31	14	45
Number of estimated VSL	181	51	232
Number of positive VSL with imputed SE	161	35	196
Mean (million $)	12.3	3.8	10.8
(Coefficient of variation)	(1.2)	(1.5)	(1.3)
Number of VSL subsets at 1st stage	43	17	60
Mean (million $)	12.4	3.8	9.8
(Coefficient of variation)	(1.1)	(0.8)	(1.2)
Number of VSL subsets at 2nd stage	43	17	60
Mean (million $)	9.4	2.8	5.4
(Coefficient of variation)	(0.5)	(0.5)	(0.4)

Table 2. Results of Empirical Bayes Estimates and Bootstrap Tests for Distribution Comparisons (2000 dollars)

	Mean (million $)	SD (million $)	Coefficient of variation	Bootstrap Test		
				Mean	Median	Interquartile
Distribution Comparison by Evaluation Method						
Total (60)	5.4	2.4	0.4	P-value (Ho: HW = CV)		
CV (18)	2.8	1.3	0.5	<0.001	<0.001	<0.008
HW (42)	9.4	4.7	0.5			
Distribution Comparison by Study Location (HW only)						
USA (30)	8.5	4.9	0.6	P-value (Ho: US =UK)		
UK (7)	22.6	4.9	0.2	<0.001	<0.001	<0.403
Distribution Comparison by Occupational Risk Data Source (HW only)						
BLS (3)	10.3	4.3	0.4	P-value (Ho: BLS = NIOSH)		
NIOSH (21)	7.2	3.9	0.5	<0.694	<0.798	<0.734
Distribution Comparison by Evaluation Method After Adding Excluded Estimates						
Total	4.7	2.2	0.5	P-value (Ho: HW = CV)		
CV	2.6	1.3	0.5	<0.001	<0.001	<0.009
HW	8.7	4.6	0.5			
5% trimmed estimate						
Total	5.8	2.5	0.4			
Including negative estimates						
Total (67)	4.1	1.7	0.4	P-value (Ho: HW = CV)		
CV (18)	2.8	1.3	0.5	<.001	<.004	<.108
HW (49)	6.6	3.6	0.5			

Figure1. Illustration of Empirical Bayes Pooling

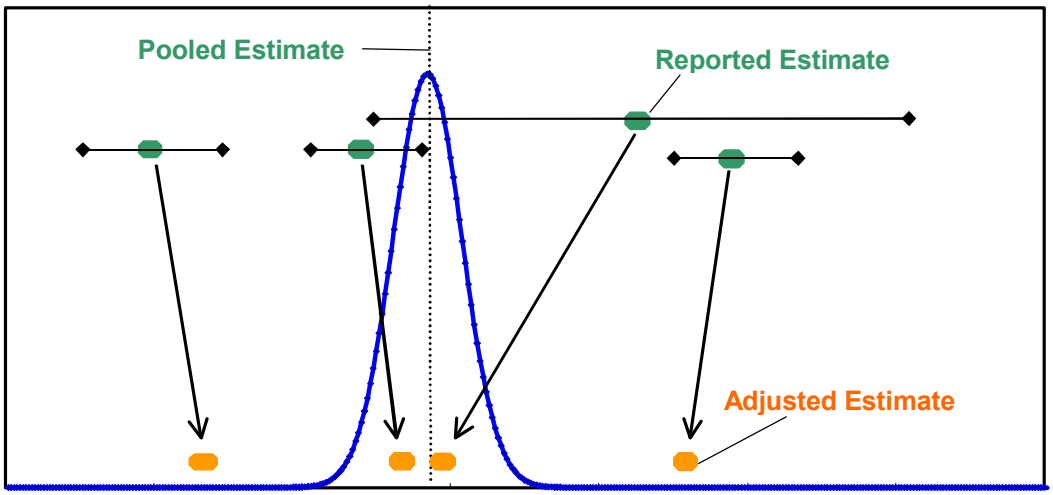


Figure 2. Comparison of Kernel Distribution of Empirical Bayes Adjusted VSL with Distribution of VSL Based on EPA Section 812 Report Estimates

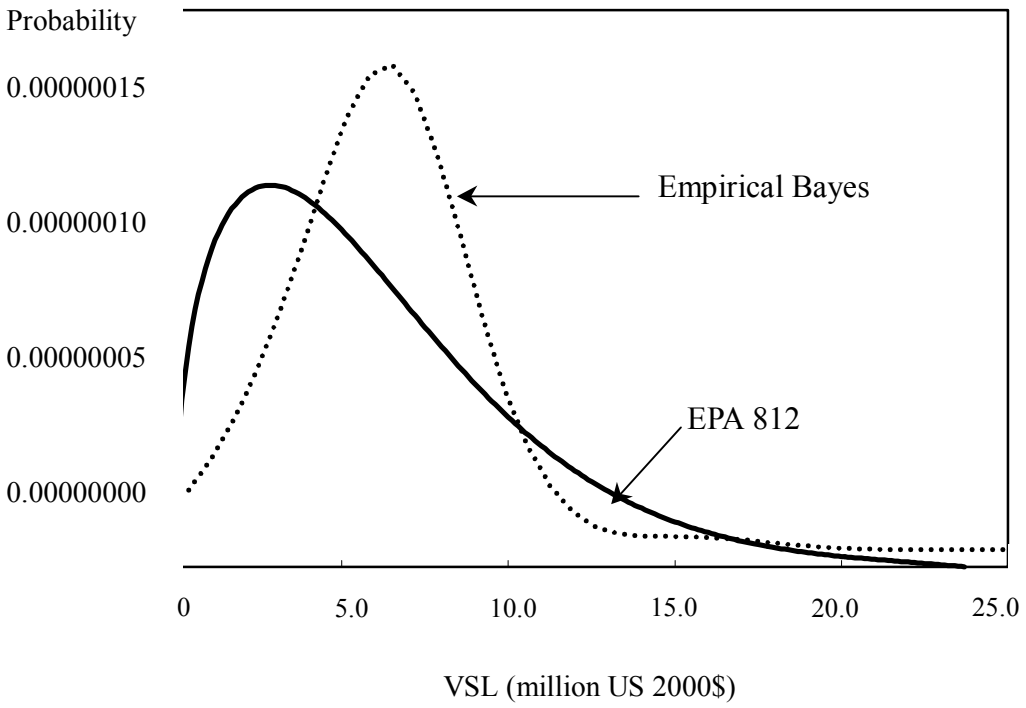
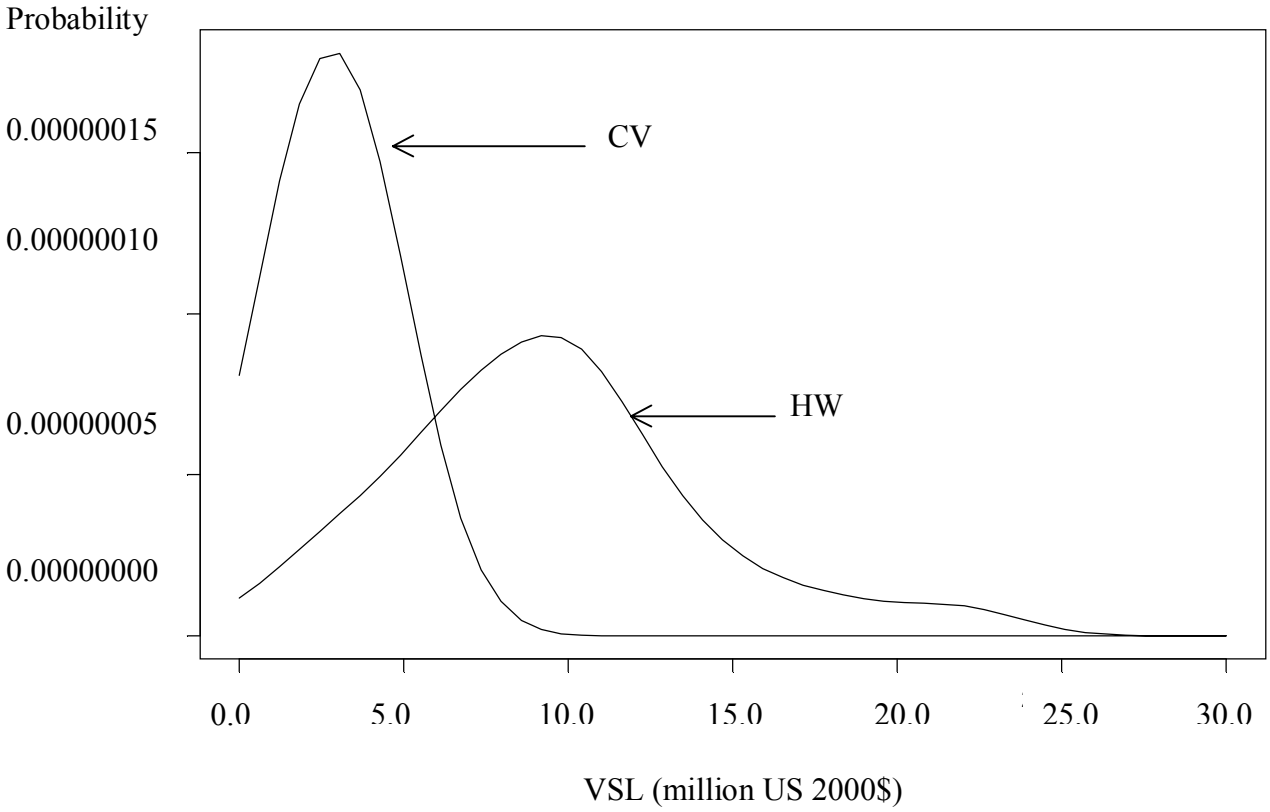


Figure 3. Comparison of Kernel Distribution of Empirical Bayes Adjusted VSL Based on HW and CV Estimates



Notes:

[1]All estimates reported in this paper have been converted to constant 2000 dollars using the Bureau of Labor Statistics Consumer Price Index (CPI). The CPI inflation calculator uses the average Consumer Price Index for a given calendar year. These data represent changes in prices of all goods and services purchased for consumption by urban households. For estimates reported in foreign currency, we first converted to U.S. dollars using data on Purchasing Power Parity from the Organization for Economic Cooperation and Development, and then converted to 2000 U.S. dollars using the CPI.

[2] Most authors do not report standard errors of VSL estimates. We have estimated the standard errors for these and other studies using an approach discussed later in the paper.

[3] We also employed fixed approaches for pooling, but found this resulted in an artifact of providing greater weight to studies whose authors reported multiple estimates.

[4] This is admittedly an arbitrary cutoff. However, we determined that a sample size of 100 did not result in many studies being excluded and smaller samples did not seem to be reasonable.

[5] We exclude one additional study, by Eom (1994), due to concerns about the payment context for the willingness to pay question. In that study, individuals were asked to choose between produce with different levels of price and pesticide risk. The range of potential WTP was limited by the base price of produce. In order to realize an implied VSL within the range considered by Viscusi, individuals would need to have a WTP of around $400 per year. Because WTP in the study was tied to increases in produce prices, which ranged $0.39 to $1.49, it would be very unlikely that individuals would be willing to pay over a 100 times their normal price for produce to obtain the specified risk reduction. Tying WTP to observed prices thus limits the usefulness of this study for benefits transfer.

[6] From http://worldbank.org/data/databytopic/class.htm. High-income OECD member have annual income greater than $9,266 per capita.

[7] One reviewer suggested that some published VSL estimates should be excluded from our analysis because the authors judged these estimates to be invalid. Our review of each study did not reveal authors' arguments excluding VSL estimates except a few instances in which authors questioned the reliability of the BLS and NIOSH occupational risk data. Because it is accepted to use these risk data in hedonic wage studies, we did not view this as a valid reason for dropping those VSL estimates. The summary of each author's review of their VSL estimate is in an appendix available upon request from the authors.

[8] Most studies use the hourly wage or weekly wage. In those cases authors multiply by 2000 (some use 2080) for mean hourly wage, and 50 (some use 52) for mean weekly wage to obtain mean annual wage. We follow each study's estimation approach and if that is not available, we use a multiplier of 2000 for hourly wage and 50 for weekly wage.

[9] The coefficient $d\ lnY/\ d\ p_i$ does not depend on the units in which Y is measured. The requirement for a comparison across is that results are converted in the same units, e.g. per thousand per year.

[10] To assure the quality of re-estimation of VSL, we matched our results with estimates done by the original authors when available. Although the VSL estimates from Kneisner and Leeth (1991), Smith and Gilbert (1984) and V.K. Smith (1976) are included in EPA 812 report, the original manuscripts do not provide VSL estimates, and we could not replicate the estimates reported in EPA 812. Therefore we exclude those studies from our analysis.

[11] A full listing of studies and their associated VSL are available from the authors upon request.

May 12, 2003

APPENDIX I

ANALYTICAL PLAN FOR AIR TOXICS CASE STUDY - BENZENE EMISSIONS REDUCTIONS IN HOUSTON

PURPOSE AND SCOPE OF THE CASE STUDY

The purpose of this document is to refine the analytical plan for a hazardous air pollutant (HAP) benefits assessment to accompany the main criteria pollutant analysis in the second 812 prospective study. Efforts to characterize the benefits of HAP reductions under Title III in prior 812 analyses have been only partially successful. An analysis of NESHAP regulations conducted for the retrospective analysis was criticized by the SAB as substantially overstating benefits, with particular note made of the use of "upper bound" dose-response relationships (i.e., the cancer potency factor used for standard setting). EPA made a second attempt to incorporate air toxics benefits, in the first prospective analysis, but the SAB felt the national air quality and exposure model proposed (ASPEN/HAPEM) would not yield estimates suitable for benefits analysis. In July 2001, however the SAB Council proposed that EPA undertake a case study, and suggested benzene as a good candidate pollutant. This document focuses on the development of a case study of the benefits of benzene emissions reductions attributable to CAAA regulations.

In the original analytical plan, we proposed to estimate only the VOC benefits of HAP controls, as part of the larger criteria pollutant analysis. Building off the results of the then-recent SAB-triggered workshop on air toxics benefits analysis, we concluded that the available tools were not appropriate for a comprehensive benefits analysis. We further proposed to conduct cost-effectiveness calculations (cost per ton HAP reduced).

In response to the original analytical plan, the SAB issued the following comments:

! **Representative HAP analysis.** The SAB advises the EPA to work with the National Air Toxics Assessment to select one representative Hazardous Air Pollutant (HAP) for which to perform a prototype 812 analysis. The SAB recommends benzene because of the wealth of available national ambient concentration data, but notes that toxic metals such as arsenic and cadmium are also options.

! **Benzene as prototype.** The SAB feels that an 812 analysis using the available benzene data would:

- identify limitations and gaps in the data base,
- provide an estimate of the uncertainties in the analyses and perhaps provide a reasonable lower bound on potential health benefits from control, and

- provide a scientific basis for deciding whether there is merit in pursuing a greater ability to assess the benefits of air toxics.

! **AQM and exposure analysis.** The SAB questions whether the sort of national modeling that is being performed for ozone and nitrogen (PM) assessments is appropriate for benzene.

In response to these comments, we are proposing to undertake a metropolitan scale analysis of the benefits of Clean Air Act controls on benzene emissions. The smaller scale will allow us both to perform a more rigorous analytical effort and to build on previous EPA modeling efforts for benzene. We propose a local-scale study of the Houston, Texas area (Harris county, specifically); this approach will allow EPA to utilize existing modeling data developed for an ongoing air toxics study in this area. The analysis will be designed to capture benefits of reductions in benzene resulting from multiple CAA Titles and provisions.

While the focus of the 812 analysis of HAP benefits remains the benzene case study recommended by the SAB, EPA has also been making progress in recent years addressing the SAB Council's earlier concerns about the data and modeling tools available to support national-scale assessments of benefits in previous 812 studies. Therefore, EPA plans to explore the feasibility and appropriateness of conducting a national-scale analysis to supplement the case study approach planned for the current 812 study. If such a national-scale assessment is conducted, advice pertaining to the merits and design of such an assessment will be sought during a future SAB review.

The analytical framework for this analysis will follow the approach for benefits analysis used in the criteria pollutant analysis of the Section 812 study. The framework includes the following steps: Scenario Development, Emissions Estimation, Air Quality Modeling, Exposure Assessment, Health Effects Estimation, and Benefit Valuation. Our plans for these steps are described in detail in the following sections.

SCENARIO DEVELOPMENT

As in the criteria pollutant analysis, the HAP case study relies on detailed descriptions of the pre-CAAA and post-CAAA scenarios. We propose to define reasonable scenarios describing benzene emissions control requirements as currently implemented and as they would be in the absence of the CAAA. The differences in the emissions, impacts, and benefits realized under these two scenarios represent the primary results of the analysis.

We define the scenarios to be consistent with those in the criteria pollutant analysis. That is, the pre-CAAA scenario freezes Federal, State, and local benzene controls applicable to Houston at 1990 levels, and the post-CAAA scenario includes all Federal, State, and local benzene rules enacted in response to the 1990 CAAA. However, due to resource considerations, we are proposing to limit the study period for the HAP case study to 20 years, from 1990 to 2010.

Although this is a case study of a hazardous air pollutant, we do not propose to analyze benefits specific to Title III of the CAAA (the Title that specifically focuses on HAPs), because doing so would ignore significant benefits related to reductions of benzene emissions from mobile and stationary sources. Instead, the difference between the pre- and post-CAAA scenarios for benzene in Houston will reflect the effect of all CAAA regulations that affect benzene emissions.

Pre-CAAA (Baseline) Scenario

This scenario will be consistent with the baseline scenario for the main analysis. It will assume no further controls on benzene emissions beyond what was in place in 1990, prior to issuance of the amended Clean Air Act. Details of the regulations included in the pre-CAAA scenario can be found in Chapter 2 of the 812 Analytical Blueprint.

Post-CAAA (Control) Scenario

This scenario will include all current and currently anticipated regulations that affect benzene emissions resulting from the amended Clean Air Act issued in 1990. We expect the scenario will include the regulations listed in Exhibit I-1.

Exhibit I-1 **Benzene Case Study Post-CAAA Projection Scenario Summary, by Title**	
Title I	Any effects of Title I will be expressed through state implementation plan (SIP) requirements, such as (enhanced) I/M programs, transportation control measures, other VOC controls. These requirements are dependent on the ozone non-attainment status of the case study area(s).
Title II	**Tailpipe standards** **Onroad** Tier 1 Standards (phased in 1994 to 1997) NLEV program –voluntary bridge between Tier 1 and Tier 2 Tier 2 Standards take effect in 2004 Heavy Duty Engine/Diesel Fuel Rule - New emission standards – 2007 model year, new fuel standards 2006 **Nonroad** Federal Phase I and II compression ignition (CI) engine standards, Federal Phase I and II spark ignition (SI) engine standards, Federal locomotive standards, Federal commercial marine vessel standards, Federal recreational marine vessel standards. **Evaporative Emissions** Stage II Vapor Recovery Systems (Section 182) Onboard Refueling Vapor Recovery (Section 202; 1998 model year and on) Fuel Spit-back rule Clean Fuel Vehicle Program **Fuel Regulations** RFG Standards (1995 on) Phase II – (2000 – present) – benzene requirements essentially unchanged Anti-dumping standards – do not specifically regulate benzene content of gasoline Summertime Volatility Requirements for Gasoline (Phase II – 1992 on) Anti-backsliding provisions of Mobile Source Air Toxics rule
Title III	**MACT Standards** We will review the full range of MACT standards to identify those that would be expected to have a significant effect on future-year benzene emissions in the Houston area. We expect that the final list of MACT standards to be analyzed in the study will include: Oil and Natural Gas Production: 7-Year MACT Petroleum Refineries: 4-Year MACT Gasoline Distribution: 4-Year MACT Pulp and Paper Production: 7-year MACT Municipal Landfills: 10-year MACT Natural Gas Transmission and Storage: 10-year MACT Publicly Owned Treatment Works (POTW) Emissions: 7-year MACT Coke Ovens: Pushing, Quenching, & Battery Stacks: 4-year MACT Synthetic Organic Chemical Manufacturing: 2-year MACT

EMISSION ESTIMATION

This section provides a brief overview of our approach to developing emissions inventories for benzene for use in the HAP case studies to be included in the second 812 prospective analysis.

To facilitate this analysis, we seek to build on previous emissions estimation efforts by EPA, while still maintaining consistency with emissions estimation for the main 812 analysis.

Available Emissions Data Sources for Benzene

The primary data source for benzene (and other HAP) emission estimates is the National Toxics Inventory (NTI) which has recently been renamed as the National Emission Inventory for Hazardous Air Pollutants (NEI for HAPs). EPA's Office of Air Quality Planning and Standards (OAQPS) is using the NEI for HAPs to support analyses required by the Clean Air Act and Government Performance and Results Act (GPRA) that depend on a high quality, comprehensive HAP emission inventory. The inventory is a critical component of the entire national air toxics program. A recent example of its use is in the EPA National Air Toxics Assessment (NATA).

The NEI for HAPs is developed every three years (1993, 1996, 1999, etc.) with the draft version 3 of the 1999 NEI for HAPs being the most recently completed version. The final version 3 of the 1999 NEI for HAPs is expected to be completed in July. The NEI for HAPs contains emission estimates for large stationary sources (point), small stationary sources (non-point), and mobile sources. Point sources in the inventory include major and area source categories as defined in Section 112 of the Clean Air Act. Non-point source categories in the inventory include area sources that are not included in the point sources and other stationary source categories. Individual emission estimates are developed for point sources, while aggregate emission estimates at the county level are made for non-point stationary and mobile sources. For all inventory years, the NEI for HAPs also identifies emission sources that are associated with MACT categories.

In addition to the NEI for HAPs data years, the benzene analysis approach also considers recently completed/ongoing HAP studies performed for the Houston-Galveston, Portland (Oregon), and Philadelphia areas.

Recent EPA Efforts to Improve Emissions Projections

EPA's Office of Air and Radiation has participated in three urban scale studies of air toxic emissions and associated concentrations, which are at different stages of completion. The three urban areas are Houston, Portland (Oregon), and Philadelphia. All three studies examined benzene as one of the HAPs evaluated and employed on-road emission estimation methods that involve some improvements to standard methods like allocations of emissions to major roadway segments. Two of the three (Houston and Philadelphia) use the ISCST3 Gaussian dispersion model to estimate ambient benzene concentrations, while the CALPUFF model is used in the Portland study, where terrain effects are more of a concern.

EPA's OAR completed urban scale modeling analyses and evaluations in Houston using 1996 HAP emission estimates, with benzene being one of the four HAPs included in the analysis. The Houston domain for the EPA study included all of the Houston-Galveston-Brazoria ozone nonattainment area counties, which are Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller.

The Portland Air Toxics Assessment is a pilot project funded by EPA in cooperation with the Oregon Department of Environmental Quality. The reference material available for the Portland study describes the on-road vehicle air toxic emission estimation procedures used to calculate hourly air toxic emissions by roadway link and travel analysis zone for the Portland-Vancouver area.

The third EPA urban study of air toxics is currently being performed for the Philadelphia ozone nonattainment area. The counties in the Philadelphia urban study domain include five Pennsylvania counties, five New Jersey counties, and one county in Delaware. The base year for this study is 1996. Study documentation available to date primarily addresses emissions processing steps. The processing steps involve running EMS-HAP programs and a post-processing program designed to split the domain into rural and urban portions, so that the air dispersion model – ISCST3 – is applied separately for urban and rural domain emissions. Benzene is one of the nine HAPs evaluated in the Philadelphia urban study.

While it may be desirable to pursue benzene analyses for all three potential urban areas of interest, this proposed analytic approach focuses on the data and analysis tools available for the Houston area that would be used to perform the needed evaluations for the second prospective. Techniques that might be applied to Portland, Oregon, or Philadelphia in the 812 assessment would be expected to be similar (but not exactly the same).

Necessary Modifications to Ensure Consistency with Main 812 Analysis

The tool that has been developed by EPA-OAQPS for performing HAP emission projections is EMS-HAP. This model has the ability to employ the same EGAS 4.0 growth factors that we propose to use in the criteria pollutant analysis in order to account for likely changes in pollution generating activity in future years affecting HAP sources. For deployment in the 812 analysis, there will need to be adjustments made to start the projections with 1999 base year emissions data and to estimate future year emissions in 2010.

Note that the growth factors in EGAS 4.0 for the Houston area are developed from a Regional Economic Models Inc. (REMI) regional model that distinguishes the Houston area from other urban/non-urban areas in Texas. However, because some source categories' (e.g., fuel combustion) growth factors are based on forecasts from non-REMI sources (e.g., Department of Energy), there will be source categories where EGAS will have the same growth factor for Houston as it does for the entire State.

Proposed Approach by Source Type

This section presents our proposed approach to benzene emissions estimation (including base inventory source, any necessary adjustments, and projection methods) for each of the major source categories we plan to include in the analysis.

Point Source Emissions

EPA has modeling inventories for Houston point sources for 1990 and 1999. The 1990 point source inventory was prepared by the Texas Commission on Environmental Quality (formerly the TNRCC) for Harris County. This data set is considered the best estimate of point source benzene emissions in 1990.

Similarly, the 1999 point source benzene emission estimates in the 1999 National Toxics Inventory, version 3 final, are the recommended data source for estimating recent (1999) emission levels. This version is expected to be completed in July 2003. With the significant reductions in reported air toxic emissions between 1990 and 1999, the suggested 1990 and 1999 point source data bases should provide the best indicator of post-CAAA scenario emission changes in this time period. Post-CAAA scenario benzene emissions for 2000 in Houston can be estimated by either using 1999 values as a surrogate, or performing a one-year projection from 1999 to 2000.

One of the key parts of this analysis will be identifying the point source benzene emission reductions attributable to MACT standards promulgated during the 1990s. To evaluate EPA's progress in reducing air toxic emissions via MACT standards, and to identify sources that may be modeled as part of residual risk assessments, operations within facilities that are subject to MACT standards are identified in the NEI for HAPs by MACT codes. MACT codes are assigned at the process level, or at the site level. For example, the MACT code for municipal waste combustors is assigned at the site level, while the MACT code for petroleum refining catalytic cracking is assigned at the process level. These MACT codes are expected to be used as an indicator of where MACT standard associated emission reductions have occurred (by 1999) or are likely to occur in future years.

One of the most important issues in the 2010 emission projections for the Houston case study is determining the appropriate level-of-detail for evaluating the expected benzene emission reductions to attribute to the CAAA measures post-1999. One way to do this is to survey the MACT-standard affected facilities in order to determine their compliance plans. However, such an effort would likely be resource intensive and time consuming. In addition, it is not clear what authority EPA has to survey the Houston area facilities in order to gather the data needed to accomplish this approach.[1] Another possibility is to work with the state agency to see if they have

[1]Paperwork Reduction Act compliance requires a rigorous application process for any survey effort involving more than nine participants.

this level of information. Again, such an effort would be resource intensive and time consuming and may not yield any of this specific data.

Our proposed approach to developing the post-CAAA scenario benzene point source emission estimates will include estimates of the likely emission reductions by facility, or source category, needed to meet residual risk requirements of Title III, if these data are available within the time frame of our analysis.

- **Option 1:** EMS-HAP contains future year control factors by source category that are designed to be applied to 1996 base year emission estimates to include the effects of MACT standards implemented post-1996. These control factors capture the average VOC HAP emission reduction expected when the new standard is applied to all affected sources in the country. Thus, the estimated nationwide emission reduction associated with a MACT standard might be 45 percent, when the range of emission reductions by individual sources varies from 0 (facilities on which the MACT standard is based) to 90 percent (at a previously uncontrolled facility). Applying the 45 percent control factor in Houston could greatly under-or-over estimate the MACT standard benefits for a source category.

- **Option 2:** The analysis alternative (for developing control factors) that is most like the criteria pollutant approach is to develop an estimate of the VOC/benzene emission control efficiency required by the applicable MACT standard for each affected source category, and to then compare the existing (1999) VOC/benzene control efficiency with that MACT standard control level to determine whether the facility is expected to be adding controls in order to meet the MACT standard requirements. This approach for modeling the future year benzene emission reductions associated with each point source category in the Houston area can only be applied if base year control efficiencies are available for MACT standard category affected units. If they are, then the MACT standard requirement can be compared with the existing control efficiency, and a further emission reduction applied if the existing control efficiency is less than what is required by the MACT standard. EMS-HAP has the capability of accounting for base year control efficiencies in computing the actual expected control efficiency with a future MACT standard. However, the base year efficiency must be in the input inventory.

  It is also our understanding that the Houston ozone SIP has been recently revised to include additional point source VOC emission controls, and that these measures may have some effect on benzene emissions at the affected facilities. Thus, control factors for the chemical and petroleum industry sources in the Houston area would have to be assembled from SIP documents and an analysis of MACT standard effects on these same sources.

- **Option 3:** Another point source analysis alternative is to see whether the EPA MACT standard Background Information Documents contain enough information to characterize the before and after MACT standard performance of the particular (either as a whole or individually) chemical and petroleum industry facilities in the area.

- **Option 4:** The simplest point source analysis alternative is to assume that the 1999 point source file emission estimates capture the majority of the post-CAAA emission benefits, and that benzene emissions will be relatively constant thereafter. This overlooks the benefits of 7- and 10-year MACT standards in the area and the recent Houston-Galveston area SIP requirements that are expected to further reduce point source VOC emissions.

The pre-CAAA scenario 2000 and 2010 point source emission estimates will be generated by applying expected increases in activity levels assuming no additional controls are implemented beyond those that were in place in 1990. Activity changes will be estimated by applying EGAS 4.0 growth factors for the Houston-Galveston area by SIC or SCC code for the 1990 to 2000 and 1990 to 2010 periods.

Given the information available, our primary recommendation is that the HAP Case Study analysis be limited to the benzene emission sources in Harris County, Texas. This allows us to focus our efforts on quantifying the estimated effects of the 1990 CAAAs on point source benzene emitters between 1990 and 1999, and the likely future changes post-1999. From a modeling standpoint, because the transport of benzene emissions from other nearby counties in the urbanized area will not be captured, it will be important to set appropriate background concentration levels to capture this contribution to ambient benzene levels in Harris County.

If the benzene analysis is performed for all eight counties in the Houston-Galveston ozone nonattainment area, then there will be significant additional effort needed to prepare point source benzene emission estimates for 1990. This would involve taking the 1999 point source file for these counties and backcasting these estimates to 1990 conditions. The primary approach that would be used to prepare 1990 benzene emission estimates for these point sources is to use data collected by EPA's Emission Standards Division during the MACT standard-setting process to estimate pre-MACT standard operating conditions and emissions.

Highway Vehicle Emissions (On-Road)

The benzene emission factors used in the 1996 Houston study were from MOBTOX (the predecessor to MOBILE6). MOBTOX-estimated benzene emission factors were estimated using a 19.6 mile per hour average speed and standard Federal Test Procedure cycle hot and cold start percentages. MOBILE6 and MOBTOX fuel parameters are the same.

In the Houston analysis, on-road emissions are modeled in ISCST3 in two ways. The first was to assign on-road emissions to 1 kilometer (km) grid cells. A second method was to allocate on-road emissions to major road segments such as Interstate, U.S., and State highways using GIS software. On-road vehicle emissions not specifically allocated to these roads were interpolated to 1 km grid cells. Therefore, for the 812 analysis, either of these two options could be used to estimate on-road benzene emissions.

- **Option 1:** Use county-level on-road benzene emission estimates and allocate to 1 km grid cells.

- **Option 2:** Where possible, place roadway emissions at actual locations using a GIS system and activity estimates for individual links (vehicle counts).

EPA prefers Option 2 because the dispersion model performance is better in Houston when this option is employed.

It is proposed that the on-road ISCST3 input file for 1996 will be used as the base file for the analysis, with scaling factors applied to these 1996 benzene emission estimates to estimate on-road vehicle benzene emissions for all of the Section 812 analysis years of interest. The scaling factors will account for MOBILE6-estimated emission factors and expected vehicle miles traveled (VMT) changes in each analysis year. It is expected that VMT projections for Harris County will be available from the Houston-Galveston Area Council to support our ability to estimate likely future year VMT changes by year and geographic area within Harris County. The proposed approach is to use available travel demand model projections for the area to prepare estimates of 2010 and 2020 VMT at the 1x1 km grid cell level. Because Houston's attainment year is 2007, it is expected that VMT projections will be available for that year. In addition, the area will have also had to prepare a long-range forecast for transportation conformity purposes. Their current efforts are in preparing a long range forecast to 2030. A year closer to 2020 may have been included in previous transportation conformity analyses. In any event, it is expected that some interpolations will be required to incorporate Harris County-specific VMT forecasts in the 2000 and 2010 HAP analysis. Our objective is to capture the expected changes in traffic patterns across Harris County in future years. Because traffic counts were used to estimate base year VMT by geographic area, and future year VMT estimates are expected to come from the travel demand model, there will have to be some reconciliation of travel demand model-estimated traffic county-estimated VMT, as well.

One of the factors in how we approach revising the on-road benzene emission estimates to incorporate MOBILE6 and to model different scenario years is the complexity associated with separating the different exhaust and evaporative benzene components, and allocating these spatially/temporally. Benzene *all vehicle* MOBILE6 emission factors for average Texas conditions are shown below:

Emission Factor Components	Benzene mg/mile
Exhaust	58.18
Hot Soak	1.54
Diurnal	0.32
Resting Loss	0.58
Running Loss	5.14
Total	66.03

In the Houston analysis, MOBTOX was used to estimate on-road benzene emissions, and we now want to use MOBILE6 to generate on-road emission factors. For calendar year 1999, the *national* on-road benzene emissions are estimated to be 174,720 tons per year using MOBILE6, and were estimated to be 165,700 tons per year using MOBTOX. This is a 5 percent increase with MOBILE6. Because this is an annual estimate for the entire United States, there could be bigger differences in specific areas and seasons. While the Houston analysis will include fuel parameters and other conditions particular to that area, the national level benzene emission differences provide a sense of what the MOBTOX to MOBILE6 adjustment might be.

In case the Philadelphia or Portland areas are to be included in the 812 HAP case study, some information about how their on-road emissions analysis methods differ from those used in Houston is provided below. The information provided in this analytical plan outline for these two areas is limited to on-road vehicle emission estimation methods because this source type was a point of emphasis in both studies.

The information available for Portland, Oregon focuses on on-road vehicle emission estimation methods. Methods applied to estimate current year HAP emissions are more sophisticated than those used for the Houston area inventory. The primary methods improvements compared with those used in Houston include accounting for differences in vehicle speeds and their effect on emission rates, differentiating running and non-running emissions -- with allocations of non-running emissions to trip origins, and using household survey results and the trip assignment model for Portland Metro to allocate travel by hour of the day. Separate MOBILE6 runs were conducted for each combination of area *fleets*, two seasons, four link types, and 14 speed bins. Speed curve equations were generated to allow benzene emissions to be computed for any associated speed. MOBILE6 emission factors were applied at a link level to compute running emissions by hour. Emissions from intra zonal travel, and all non-running emissions, were allocated at trip origins.

If the analysis is extended to the Philadelphia area, the on-road vehicle emission estimation methods used in Philadelphia are consistent with those applied to estimate Portland area emissions.

For the pre-CAAA scenario, we need to estimate what the fuel parameters were likely to have been in the absence of regulation (using 1990 values is one option). The remainder of the MOBILE6 set-up for the pre-CAAA HAP scenario will be consistent with that being performed for the criteria pollutant analysis. These data are available from the procedures used for the NEI.

Non-road Vehicle and Engine Emissions

For the off-road sector, most source categories are included in EPA's NONROAD model, so the latest version of NONROAD will be the recommended model for estimating benzene emissions (where benzene will be estimated as a fraction of VOC emissions). Off-road vehicles/engines source types not included in NONROAD are aircraft, railroad locomotives, and marine vessels.

For the source categories in Houston whose benzene emissions for 1996 were estimated using the NONROAD model, for each analysis year, the most recent NONROAD model will be used to develop a benzene emission factor for each source category. The ratio of the new emission factor for each analysis year to the previously estimated 1996 Houston benzene emission factor will be used to develop a composite non-road benzene emissions scaling factor that will be used to adjust the gridded benzene emissions file (input to ISCST3).

For the 1996 Houston analysis, special processing was performed for aircraft emissions. These emissions are separated from the mobile inventory using Airport Proc. This program separates airport emissions from the mobile inventory and prepares the airport emissions for input into the point source processing programs. Airport Proc allows for modeling airport-related emissions as ISCST3-area sources with known locations and dimensions, rather than as spatially allocated mobile sources. This capability was built into the program because airport locations are known. EMS-HAP has been revised (though not yet documented) to include this capability for airport-related emissions from the area source inventory (e.g., aircraft refueling) and generalize it to include other traditionally nonroad or non-point sources in which specific locational data could be supplied.

For categories not included in NONROAD, we propose to apply growth and control factors (VOC factors) developed for the criteria pollutant analysis to develop consistent emission projections for benzene. Some adjustments may be necessary to aircraft emissions so that they can be processed separately for input to point source processing programs.

To configure the NONROAD model to remove the effects of the CAAA for the pre-CAAA scenarios, we expect to develop a specialty input file for NONROAD. This input file will be used to simulate emission rates if uncontrolled 1990 emission rates persist. All non-road engine emission standards are attributable to the CAAA, so we need uncontrolled 1990 emission factors to apply to the expected activity in each projection year.

Area Source Emissions (Non-Point)

For non-point (area) sources, the criteria pollutant analysis is designed to use the 1990 and 1999 National Emission Inventory emission estimates for most source categories to reflect the emission changes for the post-CAAA scenario. The exception to this is for source categories with significant emission estimation method changes in this period. The same basic approach is proposed for benzene. We need to determine which source categories have had the most significant methods changes. Fire emission estimates were mentioned in a recent conference call as one candidate for separate treatment.

Within non-point, one of the most prominent benzene sources is service station emissions. An important component of these emissions is vehicle refueling (because of the personal exposure). MOBILE6 is the preferred tool for producing emission factors for refueling because it can account for the combined effects of any Stage 2 controls plus the onboard vehicle refueling controls that have appeared on new gasoline-fueled vehicles since the mid-1990s. It is our understanding that the Houston ozone nonattainment area counties implemented Stage 2 controls in 1993. Therefore, the post-CAAA scenario benzene emission estimates for Houston will include these Stage 2 associated emission reductions (at a 95 percent control efficiency). The 1990 and pre-CAAA benzene emission estimates will be at pre-control (uncontrolled) levels. While service station emissions are typically represented in the non-point source data base, there may be service stations included in the point source data base for the area which will have to be reconciled with the non-point source estimates.

For the pre-CAAA scenario analysis, it will be necessary to identify the area source categories that emit benzene that have been affected by 1990 CAAA provisions. This may be a combination of Title I - Nonattainment provisions designed to reduce ozone precursors and Title III requirements. One way to investigate this is to identify where control factors have been applied in estimating benzene non-point source emissions in the 1999 NEI for HAPS.

> ***We need to determine which benzene-emitting area source categories have had significant methods changes between when the 1990 and 1999 area source emission estimates were produced. For these categories, 1990 benzene emissions will be re-calculated using methods consistent with those used for 1999 estimates. Because the Texas CEQ submitted its own 1990 area source emission estimates based on some specialized surveys performed for Harris County, and the 1999 draft benzene emission estimates include some non-point source submittals from the State, some effort will have to be spent ensuring that regulation-affected benzene emissions can be pinpointed.***

AIR QUALITY MODELING

The choice of an air quality model for use in a specific geographic area depends on several factors, including the complexities of weather and terrain in the area; the level of detail available in the emissions inventory, and the schedule and resources of the project. While many air quality models could be used to assess ambient concentrations of a HAP in an urban area, we have selected the Industrial Source Complex - Short Term (ISCST3) model for this analysis. ISCST3 is a steady-state Gaussian plume model used to assess pollutant impacts from multiple point, area, and mobile sources.

An air dispersion model used to estimate air toxic pollutant concentrations in an urban area should meet certain criteria. Ideally, the model should:

- be readily available;
- represent state-of-the-art modeling practice;
- be applicable to urban areas and irregular terrain;
- be capable of handling point, area and mobile sources;
- be capable of accounting for dry and wet deposition of pollutants;
- be capable of treating atmospheric chemical transformations - pollutant chemistry;
- be capable of accounting for pollutant emissions that vary by season and hour-of-day;
- be able to group source types for assessing impact;
- be capable of providing annual average concentration estimates (as well as shorter time averages);
- be computationally efficient; and
- demonstrate good performance when compared with observed concentrations. (U.S. EPA, 2002)

The ISCST-3 Gaussian plume model is widely used for estimating the impacts of non-reactive pollutants such as benzene because of its good performance against field measurements, and because it is computationally efficient relative to other types of models, such as grid and puff models. The lack of complex terrain in the Houston area also makes ISCST3 a good choice for this analysis; cities with less level terrain may benefit from more complex models such as CALPUFF. Other features of the ISCST3 dispersion model that make it useful for modeling air toxics in an urban environment also include:

- modeling of multiple point, area, and mobile sources;
- incorporation of building downwash effects;
- availability of an urban dispersion option;
- flexibility in specifying receptor locations and grouping of source impacts;
- algorithms for assessing the effects of elevated and/or complex terrain;
- modeling of the effects of deposition of gaseous and particulate emissions;
- an option to vary emissions by season and hour-of-day; and

- an option to treat atmospheric transformations by exponential decay (U.S. EPA, 2002).

Additional details about the ISCST3 dispersion model can be found in the ISC3 model user's guide (U.S. EPA, 1995).

We plan to run ISCST3 for the base year, 1990, and each of the target years for both the pre- and post-CAAA scenarios to calculate annual average benzene concentrations at the population weighted centroid of each census tract in Harris County. We plan to use a similar modeling approach to that used by EPA in its previous analysis of benzene concentrations in the Houston area in 1996 (U.S. EPA, 2002)

Alternatively, if the source locations used in the 1996 emissions year modeling analysis for Houston closely correspond with the 1990 and 1999 source locations, a source-receptor approach to air quality modeling may be possible. If the source locations match, the most efficient way to provide model-estimated benzene concentrations for the pre- and post-CAAA scenarios is to estimate source-receptor coefficients (grids versus 701 receptors) and to use the grid-level changes in benzene emissions and the source-receptor coefficients to estimate benzene concentrations at each of the receptors for each scenario. The choice between ISCST3 runs and a source-receptor approach depends on how computationally efficient the ISCST3 model is. If the model set-up and computational time is minimal, then making additional model simulations would be preferred to developing the source-receptor coefficients.

We plan to evaluate the validity of modeled annual average benzene concentrations against monitoring data for benzene in the Houston area. Air toxics monitoring data will be obtained from EPA's Aerometric Information Retrieval System (AIRS) web site at http://www.epa.gov/airs. Agreement between modeled and observed values within a factor of two will be deemed acceptable for use in the modeling effort.

HUMAN HEALTH EFFECTS ESTIMATION

This section presents our proposed approach to estimating avoided adverse health effects in humans resulting from reductions in exposures to benzene in ambient air and in various microenvironments. We begin by describing how we translate the ambient benzene concentrations output from the air quality model into estimates of benzene exposures to individuals as they carry out their daily activities. We then explain how we calculate numbers of cases of avoided cancer cases due to changes in exposure levels, using dose-response data for benzene.

Exposure Estimation

We plan to estimate time-weighted average exposure concentrations to benzene for the general populations in the study area of interest, based on the output of the air quality models. In

addition, we plan to assess risk reductions to two specific high-exposure subpopulations: individuals living in homes with attached garages who spend most of their day at home, and service station workers. We plan to generate these estimates using the same exposure model used for the general population.

The options for exposure modeling for the HAP case study include 1) the latest version of Hazardous Air Pollutant Exposure Model (HAPEM), HAPEM5, and 2) the new Air Pollutants Exposure Model (APEX).

HAPEM was developed for use in the 1996 National Air Toxics Assessment (NATA) which attempted to characterize exposures and risks from high-priority urban air toxics for population groups nationwide, using available EPA toxicity data. The HAPEM model inputs the ambient air concentrations from an air quality model, and uses microenvironment (ME) factors (factors relating the ambient outdoor concentration with the concentration for a specific indoor or vehicular microenvironment) to adjust these concentrations to reflect the conditions in each of 37 microenvironments, including gasoline service stations. Using these factors and exposure pattern data derived from EPA's Consolidated Human Activity Database (CHAD) to assess time spent in each ME for specific population cohorts, the model yielded an estimate of "exposure concentration" for each HAP to which members of the cohort were exposed.

The Science Advisory Board review of NATA criticized the HAPEM version used in the analysis (HAPEM4) for inadequately representing the distribution of exposures (U.S. EPA,EPA-SAB-EC-ADV-02-001, 2001a).[2] In particular, the SAB objected to the use of point estimates for the ME factors. The new version under development, HAPEM5, has several improvements designed to respond to the SAB criticisms. The 37 ME factors can now be input as distributions rather than point estimates, in order to better capture the full distribution of exposures. HAPEM5 can also incorporate spatial variability in air quality estimates within a census tract. Comparison of HAPEM5 with HAPEM4 indicates that mean exposure concentration estimates changed little, but variability was greatly increased in HAPEM5. HAPEM has been used in Houston previously for EPA's recent assessment of local-scale urban air toxics. However the resolution of this model for assessing temporal variability in concentrations of air pollutants is limited, because it is based on average seasonal concentrations. Completion of HAPEM5 is expected in summer 2003.

An alternative exposure modeling option is the newer Air Pollution Exposure (APEX) model. This model has been designed for smaller scale modeling and is based on OAQPS' probabilistic national exposure model for carbon monoxide (pNEM/CO). APEX is part of the inhalation component of EPA's Total Risk Integrated Methodology (TRIM), a time-series multimedia modeling system. APEX can incorporate hourly emission rates and simulate hourly inhalation exposures for all individuals in the sample population, rather than simply using the seasonal average concentration values as HAPEM does. This feature allows for assessment of acute

[2]The SAB comments on HAPEM are included for review as supplementary material for the SAB Council.

as well as chronic exposures, and correlation of exposures with specific activities. APEX also can use a mass balance approach to deriving estimates of concentrations in microenvironments. These features would be advantageous in a local-scale case study such as is planned for Houston, where locations of roads and service stations are likely to impact exposure patterns significantly. However, for this case study APEX would be paired with the less-detailed ISCST3 output, which would not fully utilize the capacity of the model to assess temporal changes in air quality. APEX uses the same data describing human activity patterns (CHAD) as the HAPEM model.

APEX also does not have the capacity to allocate pollution to "source bins" such as point or mobile sources, as HAPEM does, and it has not yet undergone external peer review (release of a beta version is planned for the near future).

At this time, we are proposing to use the HAPEM5 model, once finalized, to evaluate exposures to benzene in the Houston area. We believe it represents a reasonable approach, especially since the revisions to HAPEM5 address the key concerns raised during the SAB review of the NATA study. While the APEX model is promising and may provide the ability for more detailed analysis of exposures in the future, the benefits to the current proposed case study are not expected to be large enough to justify using a model that has undergone less review than HAPEM.

Addressing High-Exposure Subpopulations

To provide a more complete illustration of the effects of reducing benzene exposures to populations in the Houston area, we propose to do supplemental calculations of risk reductions to two high-end exposure groups - service station workers and individuals spending significant amounts of time in homes with attached garages. Both subpopulations spend large portions of their day in microenvironments expected to have above-average concentrations of benzene. Studies of the indoor air concetrations of benzene by EPA and others have found that benzene concentrations in indoor air of homes with attached garages can be two to five times higher than outdoor benzene concentrations. Exposures to service station workers are expected to be high, especially during refueling of vehicles (assuming a full-service station).

We propose to perform these supplemental calculations using the HAPEM5 exposure model. HAPEM includes microenvironmental factors for evaluating exposures at service stations (both indoors and outdoors) and in a residence with an attached garage. We will estimate the size of each of the subpopulations exposed in these environments and their age distribution, and will develop an activity profile for each group to reflect time spent at work or at home each day. We will estimate risk reductions to these groups using the same approach we are proposing for the general Houston population.

Key Benzene Health Endpoints

Our proposed method of assessing benefits from reduction in population exposures to benzene is to estimate the monetary value of the cases of adverse health outcomes avoided and to provide qualitative discussion for non-quantifiable effects likely to occur at ambient concentrations. Effects may be non-quantifiable due to a limited database associating them with benzene exposure or because they are likely to have a threshold concentration above ambient environmental levels.

Cancer

From a dose-response perspective, benzene is a very well-studied chemical with a substantial database of epidemiological data associating it with leukemia. The Integrated Risk Information System (IRIS) entry for benzene identifies the cohort studies of benzene-exposed Pliofilm workers in Ohio (Rinsky et al., 1981, 1987) as the best available data for dose-response evaluation. Due to a lack of historical exposure data, those studies had to rely on assumptions about exposure levels, which have been extensively re-evaluated by other investigators (Crump and Allen, 1984; Paustenbach et al., 1992). IRIS presents a range of unit risk estimates for benzene-induced leukemia (2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$ benzene in air). The ends of the range are derived from estimates reported in Crump (1994) and reflect two alternative approaches to estimating benzene exposures to Pliofilm workers. We note that these maximum likelihood risk estimates do not represent upper bound potency estimates, as is the case with most toxicological data for air toxics; as a result, they are better suited for use in an 812-type analysis where an assessment of typical, not high end, benefits is the goal. We propose to use data from Crump's study (1994) to develop quantitative estimates of avoided cases of leukemia due to implementation of the Clean Air Act Amendments of 1990.

In addition to leukemia, benzene exposure has been associated with other cancers in epidemiological studies, particularly non-Hodgkin's lymphoma (Hayes et al., 1997). However, the data on this endpoint are inconsistent and do not yet support a quantitative evaluation of this endpoint.

Non-Cancer

Benzene has also been associated with a number of non-cancer health effects; however, many of these appear unlikely to occur at levels expected to be found in ambient air (less than 10 parts per billion, based on EPA's NATA study). Benzene exposure at high concentrations has been associated with various hematological abnormalities, including aplastic anemia.

EPA has recently developed a reference concentration (RfC) of 0.03 mg/m^3, based on benzene's hematological effects.[3] The RfC is based on a cross-sectional study by Rothman et al. (1996) of 44 workers in Shanghai, China, who were occupationally exposed to benzene via inhalation. The critical effect on which the RfC is based is "decreased lymphocyte count." The IRIS profile notes that such an effect is a biomarker of exposure, but that the effect itself is of uncertain clinical significance to the average population. The significance of the effect depends both on the magnitude of the decrease in lymphocytes and an individual's baseline lymphocyte level. For example, the effect of reduced lymphocytes might be more significant for individuals whose immune systems were compromised (e.g., those suffering from HIV/AIDS).

At this time, we are not proposing an effort to develop a fully quantitative estimate of non-cancer hematological effects based on the dose-response data underlying the proposed new RfC for benzene. We considered extrapolating the dose-response function based on the data supporting the RfC, in order to estimate "cases" of reduced lymphocyte counts expected at environmental exposure levels. However, the data set supporting the proposed RfC is limited (2 data points) and would not support an extrapolation beyond the benchmark concentration (8.2 mg/m^3) down to the low exposures expected in the environment. We propose therefore, to assess this endpoint by reporting the difference in the number of individuals experiencing benzene concentrations above the RfC under the pre-CAAA and post-CAAA scenarios. While we recognize that exposure above the RfC does not necessarily imply the presence of an adverse effect in a given individual, this estimate nonetheless provides some measure of progress towards reducing the likelihood of adverse hematological effects.

Results from other studies suggest a possible association between benzene and respiratory effects, including reduced lung function, chronic respiratory symptoms, and asthma. However, these studies assessed benzene as a component of volatile organic compounds (VOCs) or engine exhaust and thus could not isolate any effect attributable specifically to benzene (Ware et al., 1993; Laitinen et al., 1994).

Approach to Estimating Avoided Cancer Cases

The goal of this approach is to calculate the expected number of fatal and non-fatal cases of benzene-induced leukemia avoided as a result of the implementation of the 1990 Clean Air Act regulations affecting benzene emissions in the Houston area. We will estimate benefits both on an annual basis for each target year (i.e., 2000 and 2010) and cumulatively across the entire 20-year study period. The approach we are proposing to estimate these benefits is based on the model used to estimate risks due to radon exposure in the National Research Council's BEIR IV report (1988). The approach entails a life table analysis that calculates the probability of contracting (or dying

[3]A reference concentration (RfC) is an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily inhalation exposure of the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime.

from) leukemia for a given age cohort in a given time period, conditional on the probability of surviving to that period.

The life table approach allows us to estimate benefits to age-specific cohorts, taking into account age-specific mortality rates, both all-cause and leukemia-specific. This approach also allows us to explicitly integrate into our model an exposure lag parameter, L, that assigns a weight of zero to an individual's last L years of exposure. This approach allows us to estimate a delay in the realization of benefits, but it is not necessarily the same as the "cessation lag" effect previously cited by the SAB (EPA-SAB-EC-01-008, 2001b). The "cessation lag" refers to the estimate of how fast cancer risks in a population will decline to a new steady-state level following a reduction in exposure. The lag, L, represents the period before any benefits begin to be observed. However, given the limited data available on cessation lag, this approach may provide a reasonable first approximation of the effect of latency on benefits (see below.)

We intend to calculate a partial lifetime risk of dying from leukemia, focusing on the study period. We will estimate this risk for both the pre-CAAA and post-CAAA exposure scenarios. The equation we will use for calculating the partial lifetime probability of dying from leukemia (R_0) is:

$$R_0 = \sum_{i=1}^{20} h_i / h_i^* * S_{i-1} * (1 - q_i)$$

where:

R_0 = partial lifetime risk of Leukemia incidence in the study period
h_i = Leukemia mortality rate in the study period i
h_i^* = all-cause mortality rate in the study period i
S_{i-1} = the probability of surviving through period i-1
q_i = the probability of surviving in period i
$(1-q_i)$ = the probability of dying in period i

Data on all-cause mortality rates will be obtained from the United States Department of Health and Human Services' National Center for Health Statistics for years 1990 through 2000 (if available). The estimate of the baseline leukemia mortality rate will be obtained from the National Cancer Institute's SEER database for all available years in the study period. We propose to use mortality data from the latest available year to estimate risks in the latter part of the study period. We will attempt to use Houston-specific or Texas-specific data where available.

The partial lifetime probabilities of Leukemia under the pre-CAAA and post-CAAA exposure scenarios will be estimated for different age subcohorts, assessing risk at five-year intervals using the output data from the exposure model. The cases of Leukemia in each scenario will be estimated by multiplying the probabilities associated with each subcohort by the 2000 census

population for that subcohort, and then summing the results for each target year across subcohorts. (We will also sum results across the entire 20-year study period to generate an estimate of cumulative risk). We estimate the number of leukemia cases avoided as the difference in the number of leukemia cases in the pre- and post-CAAA scenarios.

Survival rates for Leukemia have improved since the time of the Pliofilm cohort, suggesting that a increased percentage of leukemia incidence in 1990-2010 will be non-fatal. Non-fatal leukemia cases represent a separate health endpoint in our benefits analysis. Thus, we plan to estimate benefits using both Leukemia incidence rates and Leukemia mortality rates. The difference between these results will represent the estimate of avoided non-fatal cases of Leukemia.

We will estimate the change in the leukemia mortality rate due to changes in exposure in the pre- and post-CAAA scenarios using a proportional hazards model based on the cumulative exposure multiplicative risk model used by Crump (1994):

$$\Delta h_i = h_i * \beta * \Delta C$$

where:

Δh = the change in the leukemia mortality rate in study period i
h_i = the baseline leukemia mortality rate in study period i
β = an estimate of benzene's carcinogenic potency (risk per ppm-year)
ΔC = the change in cumulative benzene exposure (ppm-years)

The estimate for the beta coefficient will be the maximum likelihood value reported by Crump (1994) for the cumulative exposure linear multiplicative risk model incorporating a five-year exposure lag. (We plan to use a low-end and a high-end beta estimate, based on different assumptions about the exposure of the Pliofilm workers, to generate a range of benefit estimates; see below.) Crump also estimated coefficients for this model assuming a three and zero year lag; however he reported that the five-year lag assumption combined with the multiplicative risk model produced the best fit to the data. The true latency period for benzene-induced leukemia (and hence the corresponding cessation lag period for the full benefits of exposure reduction to be realized) is uncertain, however, and alternative assumptions about the lag structure could also be reasonable.

The estimates of the change in benzene exposure for the target years 2000 and 2010 will be derived from the exposure model output for each age cohort. We will need to interpolate estimates of exposure concentrations for years in between the target years. Our initial proposal is to perform a linear interpolation of concentration changes between the target years.

Some assumptions inherent in these calculations are that Crump's exposure-response modeling results for the epidemiology study (Pliofilm cohort) can be applied to the general population and that the relative risk model obtained applies to all age groups. The applicability to

the general population is a source of uncertainty, but the same assumption was also inherent in the cancer potency estimates already adopted by EPA. The assumption of applicability to all age groups is generally a reasonable one and is commonly used (this assumption is also apparently integral to Crump's analysis). To the extent that the cause(s) and pathogenesis of some childhood leukemias may be different from those of adult leukemias, the inclusion of the childhood leukemia rates may overestimate benefits to the younger subcohorts. However, these younger subcohorts may be more sensitive to benzene exposure, or benzene exposure may contribute similarly to the development of childhood leukemias; thus, it seems reasonable and prudent to include them.

Cessation Lag

EPA's Science Advisory Board has defined "cessation lag" as the period it takes for risk to decline to a steady state level following a reduction in exposure.[4] For most, if not all, health effects associated with air toxics, there will be little or no data estimating the length of this period. Therefore, in order to develop a reasonable temporal stream of benefits, we must rely on available data that attempt to characterize the disease *latency* (the time between a critical exposure and the development of symptomatic disease or death).

Crump (1994) evaluated benzene risk using several models based on data from the Pliofilm cohort. His cumulative exposure models employ a "lag", L, that assign a weight of zero to the last L years of an individual's exposure. This model assumes that exposures during the most recent L years do not affect the mortality rate. Crump tested lags of 0, 3, and 5 years and found that a lag of 5 years produced a significantly better fit to the data than lags of 0 or 3 years. These findings would suggest that the *latency* period for benzene-induced leukemia is *at least* five years, but could be more. It also implies that zero benefits would accrue in the first five years following an exposure change.

Also, a recent paper by Silver et al. (2002) that evaluated the effect of follow-up time on risk estimates in the Pliofilm cohort found that the relative risk of leukemia peaks in the first few years following cessation of benzene exposure and that exposures five to ten years prior to the cutoff of exposure have maximal impact on risk. Together with Crump's findings, this suggests that a new steady state risk level may not be reached before at least five years and possibly 10 years following an exposure reduction.

We are proposing using an exposure lag of 5 years in the HAP case study when estimating the time stream of benefits due to reductions in benzene exposure, as a first approximation to the "cessation lag". However, we acknowledge that the database regarding the latency of benzene-induced leukemia, on which we must base our framework, is quite limited and uncertainty in the mode-of-action of benzene carcinogenesis makes it difficult to assess the biological plausibility of the values reported in these studies. As a result, we propose to evaluate the effect of alternative lag

[4] U.S. EPA, *Arsenic Rule Benefits Analysis: An SAB Review*. August 9, 2001. EPA-SAB-RSAC-01-005.

structures (e.g., zero years, ten years, or five-years with a "phasing-in" of benefits) on benefits as part of a sensitivity analysis.

ECONOMIC VALUATION OF EFFECTS

This section describes our approach to assigning economic value to the estimated benefits of reductions in ambient benzene concentrations. The scope of the valuation methodology is determined by the prior steps in the case study, which necessarily limits monetization to those health effects for which concentration-response estimates are available. This is not meant to imply that the ecological and non-quantified health benefits of benzene reductions have no value, only that within the framework of this case study we are unable to estimate that value.

Overview of Approach

We plan to apply valuation methods that are consistent with those employed to value the benefits of the Second Prospective analysis of criteria pollutants. For example, the valuation of fatal cancers will rely primarily on the base value of statistical life (VSL) estimates used for PM mortality valuation. In the benzene exposure case, however, there is the additional consideration of a potential "cancer premium" that many analysts believe to be an aspect of the health risk context that is important for valuation. In addition, the valuation of non-fatal cancer cases is not reflected in the criteria pollutant analysis. Finally, there is the consideration of non-cancer health effects associated with benzene. Although no quantification of non-cancer effects is planned for the case study, we plan to provide some economic context for these real benefits of benzene control programs by providing, where possible, cost-of-illness estimates and a summary of potentially relevant willingness-to-pay values for the critical effect of concern (decreased lymphocyte count).

For non-fatal cancer case valuation we propose to follow recent SAB advice on this topic given during a consultation in 2001 regarding a possible arsenic rule-making by EPA's Office of Water (EPA/SAB 2001b). Those recommendations have not been implemented by EPA to date, in part because the arsenic drinking water rule was finalized based on a prior analysis, but we believe the recommendations are relevant here, with some adjustment as outlined below.

Valuation of Cancer Endpoints

Fatal Cancers

Fatal cancers will be valued on a per-case basis using the VSL estimate developed from meta-analysis of estimates in the relevant economic literature. The approach to developing this VSL estimate is described in depth in Chapter 8 of the Analytical Blueprint, Economic Valuation. The estimate developed from the meta-analysis described there reflects valuation of immediate, non-

cancer risks. As a result, this value needs to be adjusted to reflect the timing of the manifestation of the risk (addressed in a separate section below), and the potential for a "cancer premium."

The potential for a cancer premium was explicitly acknowledged in the previously cited SAB report, and derives from the observation that cancer victims may suffer greater fear or dread than the victims of the causes of death involved in VSL studies that underlie the meta-analysis used here (see page 17 of their report). If health individuals perceive that a death from cancer is worse than a death from another cause, then it is plausible to conclude that they would be willing to pay more to avoid that type of death. The SAB concluded that there was little reliable information on how large the premium might be, however.

The SAB did nonetheless endorse "the addition of estimates of the medical costs of treatment and/or amelioration for fatal cancers to the VSL as a lower bound on the true value of avoiding fatal cancers." In our case, these estimates would relate to the treatment costs for a fatal case of leukemia. EPA is aware of no careful, comprehensive estimates of the cost of illness for leukemia treatment, and leukemia is not one of the cancers currently covered by EPA's Cost of Illness Handbook, but costs for other, potentially similar cancers may be appropriate for this purpose.[5] Resource limitations preclude the development of a new primary cost-of-illness estimate to support this study, but EPA plans to conduct a review of the health economics literature to ensure that the best available estimates are used.

Non-fatal Cancers

Estimates of the value of avoiding non-fatal cancers are sparse in the economic literature. The SAB arsenic panel, commenting on a valuation strategy for non-fatal bladder cancer, recommended the use of two estimates that could be interpreted as the "two extreme estimates available in the literature" as bounds in an uncertainty analysis. The two estimates are for the value of avoiding chronic bronchitis obtained by Viscusi, Magat, and Huber (1991), and the value of avoiding nonfatal lymphoma obtained by Magat, Viscusi, and Huber (1996). Both estimates are willingness to pay estimates, but both are derived from mall intercept studies that raise concerns about the representativeness of the sample. Chronic bronchitis is a serious chronic condition that the EPA Office of Drinking Water has interpreted to be similar in severity to nonfatal cancer.

We plan to follow the SAB's advice for valuation of nonfatal cancers, but to use a chronic bronchitis value consistent with that used in the Second Prospective criteria pollutant analysis, which incorporates downward adjustments in severity of the chronic bronchitis case that are consistent with the type of case usually associated with air pollution exposure.

[5] See **http://www.epa.gov/oppt/coi/** for access to EPA's Cost of Illness Handbook.

Consideration of Cessation Lag

As discussed in prior sections of this chapter, reduction in exposure to benzene leads to reduction in cancer cases after a period of cessation lag. In economic terms, it is plausible to assume that individuals would prefer avoidance of immediate health effects relative to avoidance of health effects with a delay, suggesting that their willingness to pay to avoid delayed health effects is affected. Because the underlying VSL estimates are largely for immediately manifest risks of death, the VSL estimate needs to be adjusted to account for the effect of the cessation lag on willingness to pay.

We plan to make this adjustment by discounting the VSL estimate by the period of cessation lag using two alternative discount rates consistent with those applied in the Second Prospective as a whole (i.e., a primary estimate using a discount rate of 3 percent, and an alternative estimate using a discount rate of 7 percent).

UNCERTAINTY ANALYSIS

This section discusses of proposed efforts to characterize uncertainty and variability in the benefits estimates for the benzene analysis.

Emissions and Air Quality Modeling

The uncertainties associated with these two elements of the analytical chain are complex, and we currently anticipate that resource limitations will preclude a probabilistic, quantitative treatment of the effect of these uncertainties on the benefit results. Therefore, at this time, we propose to address uncertainties in these elements qualitatively, by identifying the key uncertainties, assessing their relative magnitude (e.g., major versus minor) and their likely impact on our results.

Exposure

The HAPEM model incorporates variability and uncertainty distributions into its exposure modeling algorithm, facilitating the characterization of variability and uncertainty in exposure. Among the stochastic elements in the HAPEM model are variability in demographic characteristics, activity patterns across demographic groups (e.g., time spent in different microenvironments), and variability in work location. The version of HAPEM currently being developed (HAPEM5) also incorporates variability and uncertainty in microenvironment factors that relate concentrations in a microenvironment to ambient levels, and spatial variability in ambient HAP concentrations within census tracts. The output of the HAPEM model will provide distributions of exposure concentrations for different demographic groups that can serve as inputs to a probabilistic Monte Carlo analysis of the benefits of reductions in benzene exposure.

Dose Response

The major sources of uncertainty in this part of the analysis center on uncertainty surrounding the true value of the beta coefficient describing the carcinogenic potency of benzene and the true shape of the concentration response function at the lower concentrations expected to be found in ambient air.

Uncertainty in the Concentration-Response (C-R) Coefficient (Beta)

Much of the uncertainty surrounding the carcinogenic potency estimates for benzene arises from uncertainty in reconstructing the exposures of the Pliofilm workers. To reflect this uncertainty, we propose to calculate the primary benefit estimate of the reduced risk of benzene-induced leukemia as a range of values. This lower end of this range will be calculated using the beta value that is associated with the lower Paustenbach exposure estimates in a multiplicative cumulative exposure risk model with L = 5 years (1.1E-02); the upper end risk reduction will be calculated using the beta value that is associated with the higher Allen exposure estimates and a multiplicative cumulative exposure risk model with L = 5 years (1.7E-02). Because IRIS does not assign probabilities to the potency estimates calculated using alternative exposure assumptions, we do not assign probabilities to the alternative benefit values calculated using those beta values. Thus, the range should not be interpreted as a statistical confidence interval; the primary benefit estimate is expected to fall within the reported range of values, however.

We will also estimate an uncertainty distribution around each of the beta values used to calculate primary benefits. This distribution will capture the uncertainty in the measurement of the beta value, separate from uncertainty in the exposure reconstruction. For each beta value, we will use the the reported estimate in the study as the best estimate of the mean of the distribution of C-R coefficients. We will then characterize the uncertainty surrounding the estimate of the mean C-R coefficient as a normal distribution, with a standard deviation derived from the standard error of the reported beta value. These distributions can then be used as inputs into a Monte Carlo analysis of benefits that would generate a distribution of benefits results for each of the two ends of the benefits range.

Uncertainty in the Dose/Response Model

The mode of action for benzene-induced leukemia is complex, and despite significant advances in our understanding of the process, much remains uncertain. As a result, the true shape of the dose response function can not be known with certainty. EPA has concluded that there is insufficient evidence at present to reject a linear dose-response curve for benzene, and thus recommends use of the low-dose linear model.

However, there is some evidence suggestive of a non-linear dose response at low doses, and risk estimates would be significantly affected if a non-linear model were to be adopted. Ideally, EPA would address this model uncertainty in the proposed benzene case study using a sensitivity

analysis that illustrates the effect on benefits of assuming one or more alternative dose-response shapes (e.g., a supralinear and a sublinear model). Identifying suitable alternative functions from the many non-linear possibilities appears to be a quite difficult task, however, due to the lack of observed data in the low-dose range and the remaining uncertainties surrounding the benzene mode of action. Therefore, we are not proposing to recommend a quantitative evaluation of dose-response model uncertainty but will instead include a qualitative discussion of its possible impact on benefits.

Valuation

Uncertainty analysis for the valuation component will largely depend on analytic choices made in the criteria pollutant analysis. Uncertainty in the base VSL estimate used for fatal cancer will be characterized based on the Kochi et al. (2003) results presented in Appendix H of this document. The project team continues to explore options for characterizing uncertainty in the medical cost of treatment component of fatal cancer valuation. One option is to rely on estimates of measurement error and/or variability in cost of illness as it is currently estimated based a national survey method. The approach for non-fatal cancer valuation that we propose implies uncertainty characterized by a uniform distribution of values within the bounds of the "two extreme estimates available in the literature" for chronic bronchitis.

In the cases of fatal and non-fatal cancers, these characterizations of uncertainty are appropriate for inclusion in a probabilistic framework. Uncertainty in valuation of the effect of a cessation lag, however, is more appropriately addressed by a sensitivity test. We propose to evaluate the effect of using a seven percent discount rate rather than the three percent rate we plan to use for the primary analysis.

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May 12, 2003

APPENDIX J

IMPLEMENTING QALY'S IN THE ANALYSIS OF AIR POLLUTION REGULATIONS

QALY Paper for Environmental and Resource Economics -- Draft May, 2002

Implementing QALYs in the Analysis of Air Pollution Regulations

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Quantifying and valuing improvements in health resulting from environmental regulation is a difficult and often thankless job. While economists and some policymakers see the necessity for such valuations in order to determine whether policies are an efficient use of societies resources, many other disciplines find cost-benefit analysis, and particularly the assignment of a dollar value to reductions in the risk of mortality, to be an "inherently flawed" process (Heinzerling and Ackerman, 2002). Even within the community of practitioners of cost-benefit analysis, there is broad disagreement over the appropriate methods by which improvements in public health should be quantified and valued.

One of the more recent controversies regards whether reductions in mortality risk should be reported and valued in terms of statistical lives saved or in terms of life years saved. A further complication in the debate is whether to apply quality adjustments to life years lost. Under this approach, individuals with preexisting health conditions would have a lower number of quality adjusted life years (QALY) lost relative to healthy individuals for the same loss in life expectancy. However, the QALY approach has some appealing characteristics, for example, it provides an alternative framework to cost-benefit analysis for aggregating quantitative measures of health impacts. As such, it provides an alternative method that can account for morbidity effects as well as losses in life expectancy, without requiring the assignment of dollar values to calculate total benefits. Whether this aggregation is appropriate for evaluating environmental regulations has still to be determined.

In recent analyses of air pollution regulations (U.S. EPA, 1999, 2000), the U.S. EPA has applied a standard damage-function approach to quantifying and monetizing health benefits of

reducing air pollution. This approach has been used elsewhere in numerous applications (Kunzli et al., 2000; Levy et al., 1999). This approach quantifies reductions in individual health outcomes, such as premature mortality and chronic bronchitis, and assigns dollar values to those outcomes to obtain aggregate measures of monetized health benefits.

This paper examines the implications of an alternative approach, the quality-adjusted life years (QALY) method, which converts all health impacts (both mortality and morbidity) into changes in quality adjusted life years. Once the conversion to QALY has been accomplished, QALY can be aggregated across health outcomes and combined with costs to provide cost-utility ratios. Alternatively, a monetary value can be assigned to each QALY gained to provide an estimate of aggregate monetized benefits which can be compared with costs to calculate net benefits.

Within this paper, I provide an overview of the key issues involved in implementing a QALY based approach for evaluating the health impacts of air pollution regulations and illustrate these issues with an example based on the recent Heavy Duty Engine/Diesel Fuel regulations. Section 2 presents a review of the current benefit-cost framework and the motivations for exploring an alternative QALY based framework. Section 3 compares the assumptions embedded in willingness-to-pay based values with those embedded in QALY values. Section 4 outlines several different methods that may be used to integrate QALYs into a cost-benefit framework. Section 5 provides the results and discussion of the illustrative application of the QALY approach to the Heavy Duty Engine/Diesel Fuel regulations. Section 6 concludes with some thoughts on future research needs and policy considerations.

Cost-Benefit Methods and the Rationale for a QALY Based Analysis

EPA is required under Executive Order 12866 to evaluate the costs and benefits of major regulations, defined as those expected to have a cost of at least $100 million dollars (Clinton, 1993). The current interpretation of this directive is to provide estimates of economic benefits based on aggregations of individual willingness-to-pay (WTP), which reflects individual preferences for health and environmental improvements. EPA's current approach uses WTP applied to incidence of disease and premature death to calculate health-related benefits of air pollution reductions. Length of life lost and quality of life are not treated independently of WTP, although age-specific WTP for mortality risk reductions is considered in a sensitivity analysis (U.S. EPA, 2000).

Based on the current cost-benefit framework, the most important quantifiable health benefits associated with reduced air pollution are reduced risk of death and reduced risk of chronic illness. Monetized health benefits are dominated by the value of PM-related premature mortality benefits. The absolute size of mortality benefits is driven by two factors, the relatively strong concentration-response function, which leads to a large number of premature deaths predicted to be avoided per microgram of ambient $PM_{2.5}$ reduced, and the value of a statistical life, estimated to be about $6.3 million (2000$). The relative size of mortality benefits, i.e. the share of total health benefits accounted for by mortality, is driven by both the large absolute magnitude of mortality benefits and by the relatively low values placed on non-mortality effects.

In recent reports, the Office of Management and Budget, which reviews all regulations for compliance with E.O. 12866, has argued that "there are strong arguments that 'life-years' is a

better measure than 'lives' of the effectiveness of regulatory alternatives" and that in cases where there are reductions in non-fatal risks, i.e. risks of disease, "OMB is considering the use of new effectiveness measures that combine information on mortality and morbidity." (U.S. Office of Management and Budget, 2001, 2002) Two such measures mentioned by OMB are QALYs and disability adjusted life years (DALY). OMB recommends such measures because 1) they allow for aggregation of mortality and morbidity without application of dollar values, 2) they provide more emphasis on morbidity impacts, and 3) QALYs have been widely adopted in the public health economics literature (U.S. Office of Management and Budget, 2002).

The EPA Science Advisory Board has also suggested that "EPA consider reporting some results in terms of implied cost-effectiveness (e.g., dollars per life-year)." They suggest that "EPA consider calculating the cost-effectiveness of the CAA and certain of its provisions for comparison with other interventions that improve health. In other areas of public health, cost-effectiveness is frequently characterized as cost per QALY gained." But they also note that "alternative measures, such as the value of a statistical life-year (VSLY) or the value of a QALY, are not consistent with the standard theory of individual WTP for mortality risk reduction" (U.S. EPA Science Advisory Board, 2001).

The recommendations of OMB and SAB are consistent with the recommendations by the National Academy of Sciences panel on cost-effectiveness. The NAS panel recommended the use of QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity (Gold et al., 1996). The OMB, SAB and NAS panel recommendations motivate the following discussion of implementation issues for QALYs in assessing the benefits

of air pollution reductions. However, the following discussion is predicated on the assumptions embedded in the QALY analytical framework. As noted in the QALY literature, QALYs are consistent with von Neumann-Morgenstern utility theory only if one imposes several restrictive assumptions, including independence between longevity and quality of life in the utility function, risk neutrality with respect to years of life, and constant proportionality in tradeoffs between quality and quantity of life (Pliskin, Shepart, and Weinstein, 1980; Bleichrodt, Wakker, and Johannesson, 1997) To the extent that these assumptions do not represent actual preferences, the QALY approach will not provide results that are consistent with a cost-benefit analysis based on the Kaldor-Hicks criterion. Even if the assumptions are reasonably consistent with reality, because QALYs represent an average valuation of health states rather than the sum of societal WTP, there are no guarantees that the option with the highest QALY per dollar of cost will satisfy the Kaldor-Hicks criterion, i.e. generate a potential Pareto improvement (Garber and Phelps, 1997).

Cost-benefit analysis based on WTP is not without potentially troubling underlying structures as well, incorporating ability to pay (and thus the potential for equity concerns) and the notion of consumer sovereignty. Table 1 compares the two approaches across a number of parameters. For the most part, WTP allows parameters to be determined empirically, while the QALY approach imposes conditions a priori. Noting these differences, the remainder of the paper takes an agnostic view of the two methods and investigates additional issues that arise in applying the QALY method to air pollution regulations.

QALY Implementation Issues

In designing a QALY-based analysis of the benefits of reducing air pollution, a number of important issues need to be addressed. These include (in no particular order of importance): treatment of non-health benefits, treatment of acute symptoms, assessment of baseline life expectancy and quality of life weights, assessment of loss in quality adjusted life years from mortality and morbidity due to air pollution, and integration of QALYs into cost benefit analysis, i.e. assignment of values to QALYs. There are potentially other issues, however, I will focus on this set as the most likely to substantially affect the evaluation of the QALY method.

Reductions in air pollution may result in a broad set of health and environmental benefits, including improved visibility in national parks, increased agricultural and forestry yields, reduced acid damage to buildings, and a host of other impacts. QALYs address only health impacts, and the SAB notes that "EPA should be careful to acknowledge that the costs per QALY or life-year would be overstated to the extent that there are other benefits of the pollution reduction." To address this issue, OMB suggests that agencies "develop a suitable measure of the effectiveness of disparate programs directed toward enhancing other [non-health] aspects of the nation's welfare" and in the construction of their league table in the 2002 Federal Budget, chose to "subtract the value of these benefits from the aggregate cost estimate to yield a net cost estimate." I will follow this same "net cost" approach in the illustrative exercise.

Health effects from exposure to particulate air pollution encompass a wide array of chronic and acute conditions in addition to premature mortality (U.S. EPA, 1996). While chronic conditions and premature mortality generally account for the majority of monetized benefits,

acute symptoms can impact a broad population or sensitive populations, e.g. asthma attacks in asthmatic children. Bala and Zarkin (2000) suggest that QALY are not appropriate for valuing acute symptoms, due to problems with both the measurement of utility for acute health states, and application of QALY in a linear fashion to very short duration health states. Johnson and Lievense (2000) suggest using conjoint analysis to get healthy-utility time equivalences which can be compared across acute effects, but it is not clear how these can be combined with QALY for chronic effects and loss of life expectancy. There is also a class of effects which EPA has traditionally treated as acute, such as hospital admissions, which may also result in a loss of quality of life for a period of time following the effect. For example, life after asthma hospitalization has been estimated with a utility weight of 0.93 (Bell et al., 2001; Kerridge, Glasziou, and Hillman. 1995).

How should these effects be combined with QALY for chronic and mortality effects? One method would be to convert the acute effects to QALY, however, as noted above, there are problems with the linearity assumption, i.e. if a year with asthma symptoms is equivalent to 0.7 year without asthma symptoms, then one day without asthma symptoms is equivalent to 0.0019 QALY gained. This is troubling from both a conceptual basis and a presentation basis. An alternative approach is simply to treat acute health effects like non-health benefits and subtract the dollar value (based on WTP or cost-of-illness) from compliance costs in the cost-effectiveness analysis. However, this takes away one of the key comparative advantages of using QALY, the ability to aggregate morbidity and mortality effects without resorting to monetization. With that limitation in mind, I follow the latter approach in the illustrative exercise.

For air pollution regulations that result in gains in life expectancy (reduction in premature death), a critical variable in QALY analysis is the baseline life expectancy and health condition of the affected population. There is evidence that, at least for some of the mortality risks associated with short term exposure to elevated levels of air pollution, the susceptible population is comprised of individuals with chronic diseases (Goldberg et al., 2001). However, recent cohort analyses have found increased risk of all-cause mortality, as well as increased risks of cardiopulmonary and lung cancer mortality (Krewski et al., 2000; Pope et al., 2002). To the extent that the life expectancy of populations potentially affected by air pollution differs from that of the general population, QALY estimates of the benefits of air pollution reductions will be biased if general population life expectancies are used. However, there are some important issues to consider when evaluating the appropriate baseline health condition and life expectancy.

First, there is little information on life expectancy with many chronic diseases, and, QALY weights are available for some, but not all chronic health conditions. One of the more comprehensive collections of QALY weights can be found in the Cost Utility Analysis Database at the Harvard Center for Risk Analysis (Bell et al., 2001). This database lists QALY weights for many of the chronic diseases that may be preexisting risk factors for susceptibility to air pollution, including lung cancer, diabetes, congestive heart failure, cardiac disability, hypertension, and chronic obstructive pulmonary disease (COPD).

For many epidemiology studies, including most of the studies linking mortality with long-term exposure to air pollution, the distribution of causes of death within the populations is unknown except at the very broadest scale (i.e. all cardiopulmonary causes). And, for most time

series analyses of mortality, age at death is also not known. Unless we know the distributions of age at death, causes of death from air pollution and the underlying health condition of those dying from specific causes, it is difficult to assign life expectancy and baseline quality of life.

An additional important issue in determining baseline life expectancy and health conditions is whether we have properly accounted for morbidity preceding premature mortality. There are a number of epidemiological and toxicological studies linking exposure to air pollution with chronic diseases, such as chronic bronchitis and atherosclerosis (Abbey et al., 1995; Schwartz, 1993; Suwa et al., 2002). If these same individuals with chronic disease caused by exposure to air pollution are then at increased risk of premature death from air pollution, there is an important dimension of "double-jeopardy" involved in determining the correct baseline for assessing QALY lost to air pollution (see Singer et al. (1995) for a broader discussion of the double jeopardy argument).

Analyses estimating mortality from acute exposures that ignore the effects of long-term exposure on morbidity may understate the health impacts of reducing air pollution. As shown in Figure 1, individuals exposed to chronically elevated levels of air pollution may realize an increased risk of death and chronic disease throughout life. If at some age they contract heart (or some other chronic) disease due to the exposure to air pollution, they will from that point forward have both reduced life expectancy and reduced quality of life. The benefit to that individual from reducing lifetime exposure to air pollution would be the increase in life expectancy plus the increase in quality of life over the full period of increased life expectancy. Now, because the individual has contracted a chronic disease, he or she is also more susceptible

to short-term episodes of high pollution which can lead to immediate death (as picked up in the daily time series studies). If the QALY loss is determined based on the underlying chronic condition and life expectancy without regards to the fact that the person would never have been in that state without long term exposure to elevated air pollution, then the person is placed in double-jeopardy. In other words, air pollution has placed more people in the susceptible pool, but then we penalize those people in evaluating policies by treating their subsequent deaths from acute exposure as less valuable, adding insult to injury, and potentially downplaying the importance of life expectancy losses due to air pollution. If the risk of chronic disease and risk of death are considered together, then there is no conceptual problem with measuring QALYs, but this has not been the case in recent applications of QALY to air pollution (Carrothers, Evans, and Graham, 2002). The use of QALYs thus highlights the need for a better understanding of the relationship between chronic disease and long-term exposure and suggests that analyses need to consider morbidity and mortality jointly, rather than treating each as a separate endpoint (this is an issue for the current cost-benefit approach as well).

Once one has arrived at estimates of QALY gained (or lost) due to an air pollution reduction, the question arises at to how and whether to integrate these estimates into the cost-benefit framework in which other, non-health benefits are considered. The EPA SAB suggests that QALYs "are not estimates that conform with, or should be combined with, VSL estimates" (U.S. EPA Science Advisory Board, 2001). OMB is not quite as strong in their, statements, suggesting that there are several options, including: 1) don't place a dollar value on QALYs, just use them in cost-utility analysis; 2) apply a reference value from the health economics literature –

current literature suggests from $100,000 to $250,000 per QALY; or 3) apply a single value derived from the value of a statistical life (VSL), e.g. starting from a base VSL of $6.1 million, the discounted value of a life year (assuming 35 years of remaining life expectancy and a 3 percent discount rate) is $284,000. A recent QALY based analysis of mortality impacts of air pollution applies a value of $300,000 per QALY, constructed by applying an adjustment to the standard VSL-derived VSLY to account for differences in the contexts of air pollution risk and the risks on which the standard VSL is based (Carrothers, Evans, and Graham, 2002).

A fundamental problem with converting VSL into VSLY is the implicit assumption embedded in the VSLY approach that there is a linear relationship between the VSL and age. This assumption is not consistent with the current evidence on the age-VSL relationship. One potential alternative that may be more consistent with recent stated preference literature (Jones-Lee, 1989, 1993; Krupnick et al., 2000) is to use age-specific VSL to calculate age-specific value of life-years. The EPA Science Advisory Board in general agrees, noting that "inferring the value of a statistical life year...requires assumptions about the discount rate and about the time path of expected utility of consumption. The Committee agrees ....that the theoretically appropriate method is to calculate WTP for individuals whose ages correspond to those of the affected population, and that it is preferable to base these calculations on empirical estimates of WTP by age (U.S. EPA Science Advisory Board, 2000)." The VSL literature does not support additional adjustments to VSL or VSLY for health related quality of life (based on Krupnick et al, 2000). This is supported by the EPA SAB, which noted that "there are no published studies that show that persons with physical limitations or chronic illnesses are willing to pay less to

increase their longevity than persons without these limitations. People with physical limitations appear to adjust to their conditions, and their WTP to reduce fatal risks is therefore not affected (U.S. EPA Science Advisory Board, 2000)."

Table 2 lists the derived value of statistical life year for different ages based on the Jones-Lee (1989, 1993) stated preference studies. Note that the Jones-Lee 1989 paper estimated a very steep quadratic relationship between age and WTP. The Jones-Lee 1993 estimated a much flatter relationship. Because of this, for the Jones-Lee 1989 estimates, during the younger years, the VSL is decreasing more slowly than the number of remaining life years (thus VSLY increases) but during older years, the VSL is decreasing more rapidly than the number of remaining life years, leading to reductions in VSLY. For the 1993 study, VSL is always decreasing more slowly than the number of remaining life years, so you see a steadily increasing VSLY.

Setup for Illustrative Exercises: EPA's Heavy Duty Engine/Diesel Fuel Regulations

To illustrate the issues raised above and to highlight some of the implications of those issues, I develop two illustrative exercises based on the benefits analysis conducted in support of the Heavy Duty Engine/Diesel Fuel (HDE) regulations promulgated by EPA in 2000. Both exercises are based on the same data and methods for generating QALYs. The first exercise illustrates the use of cost-effectiveness or cost-utility analysis with QALYs. The second exercise extends the use of QALY by demonstrating how QALYs might be integrated into benefit-cost analyses.

The HDE regulations are estimated to result in a population weighted reduction in $PM_{2.5}$

of 0.65 μg/m^3 in 2030, when the fleet of heavy duty vehicles is expected to be fully turned over. The benefits analysis was therefore based on projected populations in the year 2030. Although we typically adjust WTP to reflect growth in real income in the future, no adjustment is assumed in this exercise for simplification. In addition, the focus of this exercise is only on mortality and chronic bronchitis risk associated with fine particulate matter. Other health and environmental benefits total $3.8 billion (U.S. EPA, 2000). The estimated costs of the rule are $4.2 billion/year, representing the annualized costs of compliance over the period of implementation.

In this exercise, I develop estimates of the QALY gained from reductions in incidence of premature mortality and chronic bronchitis associated with reductions in ambient $PM_{2.5}$. For gains in life years resulting from reduced exposure to $PM_{2.5}$, QALYs are calculated as:

$QALY\,GAINED = \sum_i \Delta D_i \times w_i \times DLE_i$, where ΔD_i is the number of premature deaths avoided in age interval i, w_i is the average QALY weight for age interval i, and DLE_i is the average discounted life expectancy for age interval i, calculated as $DLE_i = \sum_{t=1}^{LE_i} \frac{1}{(1+r)^t}$, where r is the discount rate and LE_i is the average life expectancy in age interval i. For gains in quality of life resulting from reduced incidences of PM-induced chronic bronchitis, QALYs are calculated as $QALY\,GAINED = \sum_i \Delta CB_i \times DLE_i \times \left(w_i - w_i^{CB}\right)$, where ΔCB_i is the

number of incidences of chronic bronchitis avoided in age interval i and w_i^{CB} is the QALY weight associated with chronic bronchitis. Following the literature, I discount QALYs over the period of life expectancy using a 3 percent discount rate (Gold et al., 1996). Using these QALY, I then develop examples of both cost-utility ratios and monetary estimates of benefits, by applying three different VSLY approaches. In addition to the age-dependent VSLY outlined in Table 2, I examine the impact of using QALY values from the health effects literature and VSLY derived from an age-independent VSL of $6.1 million.

The source of the concentration-response function for premature mortality is the reanalysis of the American Cancer Society cohort study of mortality and long-term exposure to fine particles, applied to adults aged 30 and over (Krewski et al., 2000). This study implies a relative risk of 1.003 for the HDE reduction in PM2.5 of 0.65 $\mu g/m^3$. Another recent QALY-based analysis (Carrothers, Evans, and Graham, 2002) suggests that mortality can be divided into acute exposure and long-term exposure risk, however, the HDE analysis focused on the risks from long-term exposure. This is consistent with recommendations from the EPA SAB and recent literature (Kunzli et al., 2001). Although there is no specific scientific evidence of a lag between reduction in PM and reductions in premature mortality, current scientific literature on adverse health effects associated with smoking and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure would not occur in the same year as the exposure reduction. This literature implies that lags of a few years are plausible. Consistent with advice from the SAB, I have assumed a five-year distributed lag

structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years (U.S. EPA Science Advisory Board, 1999).

Life expectancy at different ages was obtained from the Centers for Disease Control abridged life tables for 1999 (U.S. CDC, 2001). No information is provided in the Krewski et al. (2000) analysis to determine the distribution of underlying health status within the study population. As such, I had to make an assumption regarding the appropriate baseline quality of life for the affected population. Because the general population is likely to be on average at somewhat less than perfect health, I followed recent literature and assumed a QALY weight of w_i=0.95 for life years lost to air pollution (Carrothers, Evans, and Graham, 2002; Gold et al., 1996).

The concentration-response function for chronic bronchitis is taken from a cohort analysis of Seventh Day Adventist non-smokers, applied to adults aged 27 and older (Abbey et al., 1995). This study implies a relative risk of 1.0086 for the HDE reduction in PM2.5 of 0.65 $\mu g/m^3$. Prevalence rates were obtained from the Centers for Disease Control (Adams, Hendershot, and Marano, 1999, Table 57). There are no nationally representative estimates of the incidence of new cases of chronic bronchitis. Instead, we used an incidence estimate from Abbey, 1993, of 3.78 cases per thousand population, adjusted for age using the age distribution of the prevalence rates. I was not able to identify any literature estimating the life expectancy of individuals with chronic bronchitis. As such, I assumed that individuals with chronic bronchitis had the same age-specific life expectancy as the general population, thus chronic bronchitis is assumed to

result in no loss in life years. Based on estimates reported in de Hollander et al. (1999), years of life with chronic bronchitis are assumed to have a QALY weight of w_i^{CB}=0.69. Years without chronic bronchitis are assumed to have the same weight as the general population, i.e. w_i=0.95. In the WTP based analysis, avoided incidences of chronic bronchitis are valued at $331,000 (1999$). This value is derived from the severe chronic bronchitis/cost of living tradeoff values reported in Viscusi, Magat and Huber (1991), adjusted to average severity chronic bronchitis using the elasticity of WTP with respect to severity of illness reported in Krupnick and Cropper (1992). For more details, see the technical support document for the HDE analysis (Abt Associates, 2000).

Results of Illustrative Exercise

Table 3 provides the results of the QALY analysis of mortality risk reductions. Based on the life table analysis, the average length of life lost by an individual dying due to causes related to long-term exposure to PM2.5 is around 15 years. The total discounted QALY gained from the HDE reduction in PM2.5 in 2030 for the population over 30 is 83,771.

Table 4 provides similar results for chronic bronchitis. Based on the life table analysis, the total discounted QALY gained from reductions in chronic bronchitis resulting from the HDE regulation for the population over 27 is 33,844. It is worth noting that most of this benefit (87 percent) is due to reductions in chronic bronchitis occurring in populations under 65 (i.e. the non-elderly population). This is due to the relatively long period of life that is lived with increased quality of life without chronic bronchitis.

The relationship between QALY gained and age is shown in Figure 2. Because the baseline mortality rate is increasing in age at a much faster rate than the prevalence rate for chronic bronchitis, the share of QALY gained accounted for mortality is proportional to age. At the oldest age interval, avoiding incidences of chronic bronchitis leads to only a few QALY gained, due to the lower number of years lived with chronic bronchitis. QALY gained from avoided premature mortality is low in the youngest age intervals because of the low overall mortality rates in these intervals, although the number of QALY per incidence is high. In later years, even though the QALY gained per incidence avoided is low, the number of cases is very high due to higher baseline mortality rates.

Placing these results in the context of a cost-utility analysis, based on the costs of the HDE rule of $4.2 billion, total cost per discounted QALY ignoring all other benefits, is $35,700. The HDE rule also resulted in $3.8 billion in other health and environmental benefits, or net costs of $0.4 billion. Net cost per discounted QALY is then $3,400. Even ignoring other benefits, the cost per QALY for the HDE rule compares favorably with many other health interventions reported in the Harvard Cost Utility Analysis database, and is well below the median cost per life-year saved for live-saving interventions of $48,000 (1993$) as reported by Tengs et al. (1995). With other benefits considered, the cost per QALY is very low relative to others in the literature.

There are several important assumptions I have made due to a lack of sufficient data. One key assumption is that chronic bronchitis does not result in reduced life expectancy. If this is not the case, then individuals will gain not only the lost quality of life, but the increased life

expectancy in improved health. Another important assumption is the baseline utility weight for the population affected by long-term exposure to air pollution. I assumed that the average population affected by air pollution would have a utility weight of 0.95. However, some literature suggests that baseline quality of life is age dependent. For example, if we assume that individuals over 84 have a base utility weight of 0.81 (Tsevat et al, 1998), this reduces the QALY gained by 6 percent for reductions in premature mortality. Finally, the assumption of a 3 percent discount rate has a relatively large impact on the resulting QALY estimates. Assuming a zero percent discount rate would increase the QALY gained by 42 percent, while assuming a 7 percent discount rate would decrease QALY gained by 25 percent. Note that discount rates will have less of an impact on the overall cost-utility comparison if costs and QALY are discounted at the same rate, as both the numerator and denominator will be affected by any change in discount rate.

Based on the standard WTP method, the estimated 8,025 avoided incidences of premature mortality are valued at $6.1 million per statistical life, discounted over the 5 year cessation lag at 3 percent. This yields a total value for reduced mortality risk of $46.5 billion. Using age-dependent VSL as defined in Table 5, the total values for reduced mortality risk are $25.0 and $42.2 billion when using the Jones-Lee 1989 and 1993 adjustments, respectively. The 6,543 cases of chronic bronchitis are valued at $2.2 billion. The combined benefits of chronic bronchitis, mortality, and all other monetized benefits are thus $54.1 billion using the standard VSL, $32.6 billion using the Jones-Lee 1989 age-dependent VSL, and $49.8 billion using the Jones-Lee 1993 age-dependent VSL. When compared with costs of $4.2 billion, there are substantial net benefits regardless of which VSL method is employed.

Integration of QALY into the cost-benefit framework requires assigning a value per QALY to QALY gained from reductions in chronic bronchitis and premature mortality. Note that a QALY gained is considered the same, regardless of whether it arises from improvements in quality of life (from reduced chronic bronchitis) or improvements in quantity of life (gains in life expectancy from reduced premature death). Because of this, chronic bronchitis is not valued using a different valuation estimate. All QALY gained will be assigned the value, regardless of source. As indicated above, for this analysis, I examine five different values per QALY: two based on the medical cost-effectiveness literature, one based on the standard $6.1 million VSL, and two based on the Jones-Lee age-dependent VSL. The results of this analysis are presented in Table 6. The most striking result of this table is that when the QALY approach is used, the value of chronic bronchitis reductions is drastically increased relative to the value of reductions in mortality risk. All of the QALY based estimates of total benefits are lower than total benefits under the standard WTP based approach with age-independent VSL. However, when age-dependent VSL are used, the QALY approach actually results in larger total benefits because the value of QALY from chronic bronchitis reductions offsets the reduction in value from reduced mortality risk.

To further emphasize this finding, consider that in the standard WTP methodology, mortality risk reduction accounts for over 95 percent of combined benefits. In the direct QALY method, increase in life expectancy accounts for only 71 percent of the total QALY benefit. In the integrated assessment, this carries over, so that the value of mortality risk reduction now accounts for between 71 and 80 percent of total benefits, depending on which method is used to

derive the value of a QALY. Even when age-dependent VSL are applied in the traditional WTP based analysis, mortality benefits still account for over 90 percent of combined benefits. Clearly, the QALY method provides greater emphasis on chronic diseases which reduce quality of life relative to reductions in mortality risk which have greater impacts on older populations with lower life expectancy.

An important qualification to this finding is the sensitivity of this result to the assumed dollar value of $331,000 per case applied to chronic bronchitis. I have applied values for mortality risk reductions and chronic bronchitis risk reductions that were derived from separate sources. However, Viscusi, Magat, and Huber suggest that it may be appropriate to use their risk-risk data to derive an implicit value of chronic bronchitis which is more consistent with the assumed VSL. Using this implicit value method, the value of an incidence of severe chronic bronchitis is 0.32*$6.12, or $1.95 million. The corresponding value for an average case of chronic bronchitis would be around $958,000 per case, or around three times the assumed value. This would bring the total value of chronic bronchitis in the cost-benefit analysis to $6.3 billion, or 12 percent of total benefits, which is much more in line with the QALY based results. However, the implicit value method is very similar in concept to the QALY method, i.e. both derive value per incidence by scaling VSL, so the result is not surprising.

Conclusions

As I have demonstrated in this paper, it is a relatively straightforward process to develop estimates of QALY gained from air pollution regulations for mortality and chronic disease. It is

also numerically straightforward to monetize QALYs using a number of different methods. However, as I have discussed earlier in the paper, it is not necessarily appropriate to employ QALYs, monetized or not, in cost-effectiveness analysis, and may be especially inappropriate in benefit-cost analysis of environmental regulations. Depending on the method used to value QALYs, an integrated cost-benefit/QALY approach can result in either lower or higher monetized benefits than the traditional WTP approach. If one accepts the validity of applying dollar values to QALYs, then the additional value assigned to chronic disease in some cases more than offsets the loss in the value of a statistical life for older populations. As the value of a life year increases for older populations (the VSL falls at a less than proportional rate with age), the value of reduced mortality risk approaches the value using the VSL approach. The QALY valuation approach most consistent with the stated preference WTP literature is based on age-specific WTP. And in fact, these two methods (age-dependent VSL and age dependent value of a QALY) provide very similar values for reductions in mortality risk.

In considering the appropriateness of using QALYs in regulatory cost-benefit analysis, it is important to recognize that the QALY method requires additional data and assumptions about life expectancy, baseline health states for affected populations, life years impacted by chronic disease, and utility weights for chronic diseases which may not be appropriate for environmental policy analyses focused on maximizing net benefits. Derivation of dollar values for QALYs remains a controversial issue and adds additional uncertainty to a QALY based cost-benefit analysis. In addition, the QALY approach forces attention to the question "Does air pollution just cause death in already ill people, or does it cause the disease that leads to death?" Ignoring

this question can lead to "double jeopardy" for those with chronic illnesses caused or exacerbated to any significant degree by long-term exposures to air pollution.

From an ethical standpoint, the QALY approach may be less desirable to decision makers because it explicitly places a lower value on reductions in mortality risk accruing to older populations with lower quality of life. On the other hand, the QALY approach enhances the perceived importance of chronic disease relative to premature mortality, especially when the mortality impact is on older populations, and so can be argued to give more equitable consideration to individuals who might suffer with chronic disease for a long period of life who might otherwise be undervalued if appropriate WTP values are not available. Under certain assumptions, the QALY approach can even give larger total dollar benefits than the current method because of the enhanced value of chronic disease reductions. This may or may not hold for other environmental scenarios, depending on the suite of health impacts considered. Finally, the issue of how to aggregate acute health effects for which QALYs do not seem well suited with QALY estimates for chronic diseases and premature death may prevent QALYs from being useful for policy analysis when there are a broad range of acute and chronic health outcomes from a regulation.

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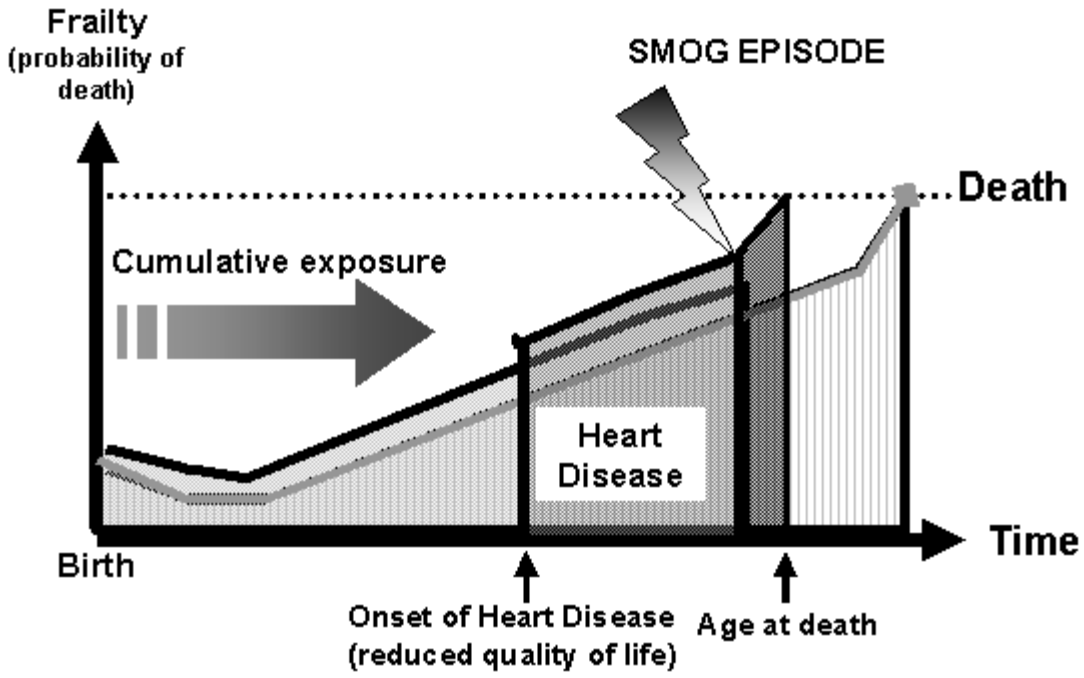

Figure 1. Relationship Between Long-term Exposure, Short Term Exposure, Chronic Disease and Death

(Source: Adapted from Kunzli, 2001).

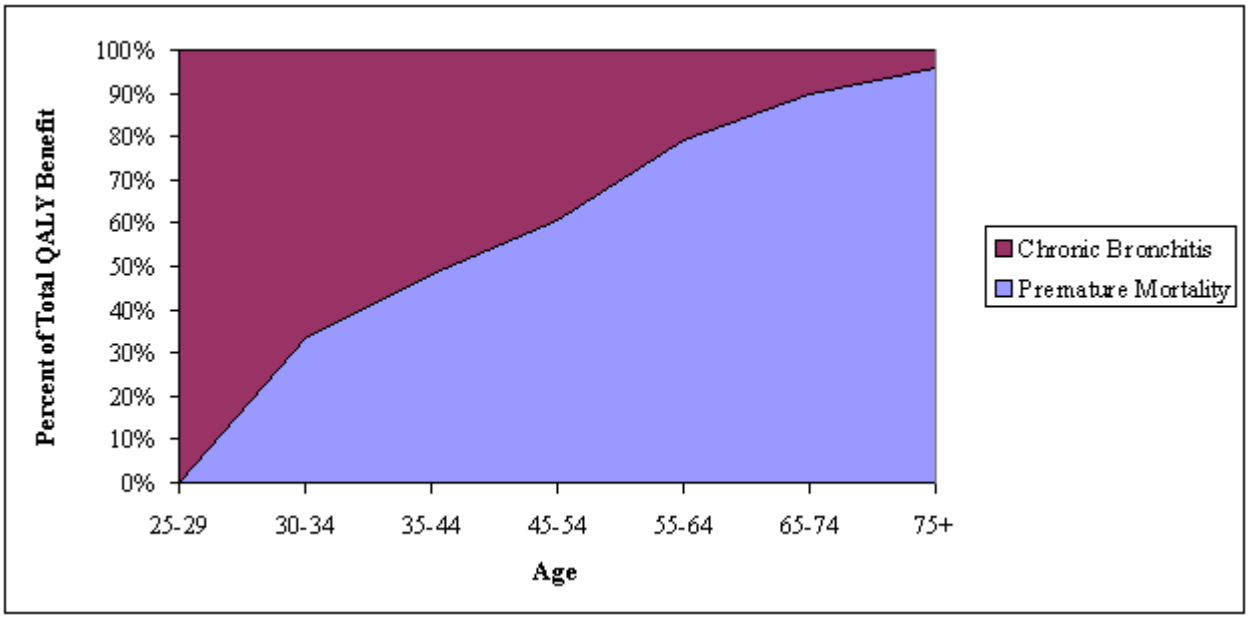

Figure 2. Age Structure of QALY Benefits

Table 1. Comparison of QALY and WTP approaches

Parameter	QALY	WTP
Risk Aversion	Risk neutral	Empirically determined
Relation of duration and quality	Independent	Empirically determined
Proportionality of duration/quality tradeoff	Constant	Variable
Treatment of time/age in utility function	Utility linear in time	Empirically determined
Preferences	Community	Individual
Source of preference data	Stated	Revealed and stated
Treatment of Income and Prices	Not explicitly considered	Constrains choices

Table 2. Derivation of Age-dependent Value of Statistical Life Year

Jones-Lee Age Group	Life expectancy	Discounted Life Expectancy (3% rate)	Adjusted VSL (J-L 1989)	Adjusted VSL (J-L 1993)	Implied Value of Life Year in Average Health for Age Group (J-L 1989)	Implied Value of Life Year in Average Health for Age Group (J-L 1993)
20-29	52	27.0	$4.00	$5.45	$152,869	$208,284
30-39	45	25.3	$5.45	$6.00	$222,149	$244,613
40-59	34	21.8	$6.12	$6.12	$289,610	$289,610
60-69	20	15.3	$5.26	$5.94	$353,770	$399,019
70-79	14	11.6	$3.86	$5.63	$341,322	$498,439
80-84	7	6.4	$1.71	$5.20	$275,044	$834,954
85+	5	4.6	$0.43	$5.02	$93,543	$1,095,791

Table 3. QALY Gained from Reductions in PM Mortality Risk

Age Interval	Reduction in PM-related Deaths in Interval (2030 Population)	Proportion of PM Deaths in Interval	Life Expectancy	Discounted Life Expectancy	Undiscounted Life years gained in interval	Discounted Life Years gained in interval
30-34	98	0.01	47.5	25.9	4,676	2,550
35-44	315	0.04	42.8	24.6	13,451	7,749
45-54	451	0.06	33.5	21.6	15,112	9,733
55-64	901	0.11	24.8	17.8	22,323	16,060
65-74	1,882	0.23	17.0	13.5	31,964	25,504
75-84	2,409	0.30	10.4	9.1	25,060	21,894
85+	1,969	0.25	5.0	4.7	9,847	9,290

Average length of life lost	15.26	11.56

	Undiscounted	Discounted
Total Gain in QALY (QALY weight = 0.95)	116,310	88,141
Discounted over 5-year distributed cessation lag:	110,544	83,771

Table 4. QALY Gained from Reductions in PM Chronic Bronchitis Risk

Age	Total 2030 Population (million)	CB Prevalence Rate (per 1000)	CB Incidence Rate (per 1000)	Reduction in CB Incidence	Discounted Life Expectancy (QALY weight = 0.95)	Discounted Quality Adjusted Life Expectancy with CB (QALY weight = 0.69)	Discounted QALY Gained per Incidence Reduced	Total Discounted QALY gained in age group
25-29	30.3	45.40	3.14	776	25.1	18.2	6.9	5,328
30-34	30.3	45.40	3.14	776	24.0	17.4	6.6	5,100
35-44	52.6	45.40	3.14	1,347	22.8	16.6	6.2	8,406
45-54	35.2	59.10	4.09	1,155	19.9	14.4	5.4	6,287
55-64	29.4	59.10	4.09	965	16.4	11.9	4.5	4,318
65-74	25.2	60.70	4.20	850	12.4	9.0	3.4	2,878
75+	18.2	67.30	4.65	675	8.3	6.0	2.3	1,526

	Undiscounted	Discounted
Total Gain in QALY (QALY weight = 0.69)	56,951	33,844

Table 5. Age-Dependent VSL Analysis of Reductions in Premature Mortality

Jones-Lee Age Group	Jones-Lee (1989) Ratios	Jones-Lee (1993) Ratios	J-L 1989 Adjusted VSL (million $)	J-L 1993 Adjusted VSL (million $)	# of Lives Prolonged	Non-Age-Specific VSL Benefits (billion $)	Jones-Lee (1989) Mortality Benefits (billion $)	Jones-Lee (1993) Mortality Benefits (billion $)
30-39	0.89	0.98	$5.45	$6.00	256	$1.5	$1.3	$1.5
40-59	1.00	1.00	$6.12	$6.12	1,059	$6.1	$6.1	$6.1
60-69	0.86	0.97	$5.26	$5.94	1,392	$8.1	$6.9	$7.8
70-79	0.63	0.92	$3.86	$5.63	2,146	$12.4	$7.8	$11.4
80-84	0.28	0.85	$1.71	$5.20	1,205	$7.0	$2.0	$5.9
85+	0.07	0.82	$0.43	$5.02	1,969	$11.4	$0.8	$9.4
Total Mortality Benefits						**$46.5**	**$25.0**	**$42.2**

Table 6. Comparison of Total Monetized Benefits Across Life Year Valuation Methods

	billion (1999$)		
Valuation Approach	Chronic Bronchitis	Premature Mortality	Total
Health Cost-Effectiveness Literature ($100,000 - $250,000 per QALY)	$3.4 - $8.5	$8.4 - $20.9	$11.8 - $29.4
Standard $6.1 million VSL Basis			
Statistical Lives Saved	$2.2	$46.5	$48.7
QALY	$9.6	$23.8	$33.4
Age-Adjusted VSL Basis			
Statistical Lives Saved, J-L 1989	$2.2	$25.0	$27.2
QALY, J-L 1989	$8.5	$24.4	$32.9
Statistical Lives Saved, J-L 1993	$2.2	$42.2	$44.3
QALY, J-L 1993	$10.9	$42.7	$53.6