



United States  
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# User's Manual for the Co-Benefits Risk Assessment Health Impacts Screening and Mapping Tool (COBRA)

Version: 4.0



**Developed for  
State and Local Energy and Environment Program**



**State and Local  
Energy and Environment Program**

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# INSTALLATION INSTRUCTIONS

## System Requirements

Users must have at least 6 GB free hard drive space, 6+ GB of RAM, i5 processor or AMD equivalent, and Windows 7 or newer. As a general recommendation, COBRA will perform better with a faster processor and a faster hard drive. A SSD drive is preferred. COBRA performance is strongly impacted by hard drive performance. Antivirus programs and full disk encryption programs may negatively impact performance. Typical COBRA run time is 5 to 15 minutes.

## Installation

COBRA can be downloaded directly to your computer or installed from a CD sent to you in the mail. If you are downloading the COBRA model, note that the installer file is large and the amount of time required to complete the download will depend on your connection speed. Find the program 'setup.exe' in the location where the installer file was saved. If you are installing the COBRA model from a CD, exit all programs and insert the Installation disk into your CD-ROM drive. The installation program may start automatically; if not, go to **Start... Run...** and then find the program 'setup.exe' in your computer's CD-ROM drive.

During installation, follow the prompts on your screen. COBRA is a large program, and depending on the configuration of your computer, it will take five minutes up to an hour to complete the installation.

## Launching the Model

To launch the model, go to **Start... Programs... COBRA**. To allow COBRA to run efficiently, turn off any antivirus programs.

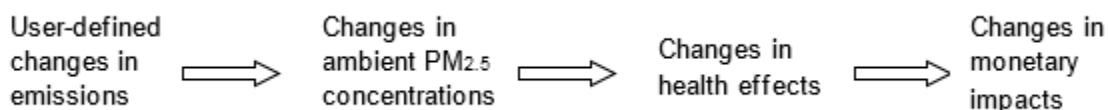
## Technical Assistance

For more information, please contact Emma Zinsmeister at 202-343-9043 or [zinsmeister.emma@epa.gov](mailto:zinsmeister.emma@epa.gov).

# CHAPTER 1. Introduction

## What is COBRA?

COBRA is a screening tool that provides preliminary estimates of the impact of air pollution emission changes on ambient particulate matter (PM) air pollution concentrations, translates this into health effect impacts, and then monetizes these impacts,<sup>1</sup> as illustrated below.



The model does not require expertise in air quality modeling, health effects assessment, or economic valuation. Built into COBRA are emissions inventories, a simplified air quality model, health impact equations, and economic valuations ready for use, based on assumptions that EPA currently uses as reasonable best estimates. COBRA also enables advanced users to import their own datasets of emissions inventories, population, incidence, health impact functions, and valuation functions. Analyses can be performed at the state or county level and across the 14 major emissions categories (these categories are called “tiers”) included in the National Emissions Inventory.<sup>2</sup> COBRA presents results in tabular as well as geographic form, and enables policy analysts to obtain a first-order approximation of the benefits of different mitigation scenarios under consideration. However, *COBRA is only a screening tool*. More sophisticated, albeit time- and resource-intensive, modeling approaches are currently available to obtain a more refined picture of the health and economic impacts of changes in emissions.

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<sup>1</sup> In calculating health impacts, COBRA generates mean estimates of health impacts. This is in contrast to a risk assessment, which typically builds in a margin of safety by presenting 95<sup>th</sup> percentile estimates.

<sup>2</sup> The emissions inventory in COBRA includes fourteen broad tier 1 categories (e.g., on-road motor vehicles); within each of these larger categories there are tier 2 (e.g., diesels), and tier 3 (e.g., heavy duty diesels) categories. The fourteen tier 1 categories include: Chemical & Allied Product Manufacturing, Fuel Combustion: Electric Utilities, Fuel Combustion: Industrial, Fuel Combustion: Other, Highway Vehicles, Metals Processing, Miscellaneous, Natural Resources, Off-Highway, Other Industrial Processes, Petroleum & Related Industries, Solvent Utilization, Storage & Transport, and Waste Disposal & Recycling.

## How is COBRA used?

COBRA can be used to quickly identify important emission sources and compare the impacts of different types of control options. Using the mapping capabilities in COBRA, users can identify the locations and types of emissions sources that contribute to local air quality problems. When considering different policy options, COBRA can help identify those options that are likely to maximize health benefits, or that could be expected to achieve health risk reductions in the most cost-effective manner. Once state and local officials narrow the set of most promising policy options through COBRA, they can then conduct analyses with more sophisticated air quality models to finalize their policy choices.

The model contains detailed emissions estimates for the years 2016, 2023, and 2028. These baseline emissions estimates account for federal and state regulations as of May 2018.<sup>3</sup> The projected EGU emissions comply with the Cross-State Air Pollution Rule Update (CSAPR Update) finalized December 27, 2016, the Mercury and Air Toxics Rule (MATS), and the Standards of Performance for Greenhouse Gas Emissions from New, Modified, and Reconstructed Stationary Sources.<sup>4</sup>

Using COBRA, you can create your own new scenarios by specifying increases or reductions to the baseline emissions estimates for the analysis year. Baseline data is preloaded for analysis years 2016, 2023, and 2028, and advanced users can analyze other years by importing custom datasets. Emissions changes can be entered at the county, state, or national level.

COBRA then generates changes in PM<sub>2.5</sub> concentrations between the baseline scenario (the “business-as-usual” estimates for the analysis year) and the control scenario (the

### Who can use COBRA?

- **State and local officials** who would like to quickly identify important emission sources and compare the impacts of different control options;
- **Analysts** looking to improve their understanding of the air quality improvements and health benefits associated with clean energy policies under consideration;
- **Environmental agencies** trying to inexpensively screen through many options to identify those that maximize the health benefits and to quantify the economic value of health improvements;
- **Energy officials** looking to estimate and promote the air quality, health, and associated economic co-benefits of their energy efficiency or renewable energy policies; and
- **Transportation planners** interested in understanding the air quality and health impacts of fuel switching or reductions in vehicle miles traveled.

<sup>3</sup> More details about the development of the 2016, 2023, and 2028 baseline emissions case are available in the supporting information for the 2016v1 Emissions Modeling Platform, available here: <https://www.epa.gov/air-emissions-modeling/2016v1-platform>

<sup>4</sup> For more information on the electricity sector modeling used to develop the emissions baselines used in COBRA, see the Documentation for EPA’s Power Sector Modeling Platform v6 Using the Integrated Planning Model, available here: <https://www.epa.gov/airmarkets/documentation-epas-power-sector-modeling-platform-v6>

analysis year modified by the user's emissions changes). A source-receptor matrix translates the air pollution emissions changes into changes in ambient PM<sub>2.5</sub> (for more information about the emissions inventory and the source-receptor matrix, see Appendix A). Using a range of health impact functions, COBRA then translates the ambient PM<sub>2.5</sub> changes into changes in the incidence of human health effects (see Appendices B through E). Finally, the model places a dollar value on these health effects (for more information, see Appendix F).<sup>5</sup> COBRA estimates the change in air pollution-related health impacts, and estimates the economic value of these impacts, using an approach that is generally consistent with EPA Regulatory Impact Analyses, including the analysis of the National Ambient Air Quality Standards for particulate matter (U.S. EPA, 2012). These analyses reflect the current state of the science regarding the relationship between particulate matter and adverse human health.

Outcomes can be modeled nationwide or for smaller geographic areas. Results include changes in ambient PM<sub>2.5</sub> concentrations, and changes in the number of cases of a variety of health endpoints that have been associated with PM<sub>2.5</sub>. These health endpoints include:

- Adult and infant mortality;
- Non-fatal heart attacks;
- Respiratory-related and cardiovascular-related hospitalizations;
- Acute bronchitis;
- Upper and lower respiratory symptoms;
- Asthma-related emergency room visits;
- Asthma exacerbations;
- Minor restricted activity days (i.e., days on which activity is reduced, but not severely restricted); and
- Work days lost due to illness.

Users can view the results in tabular or map form as well as export the data for use in their own communications.

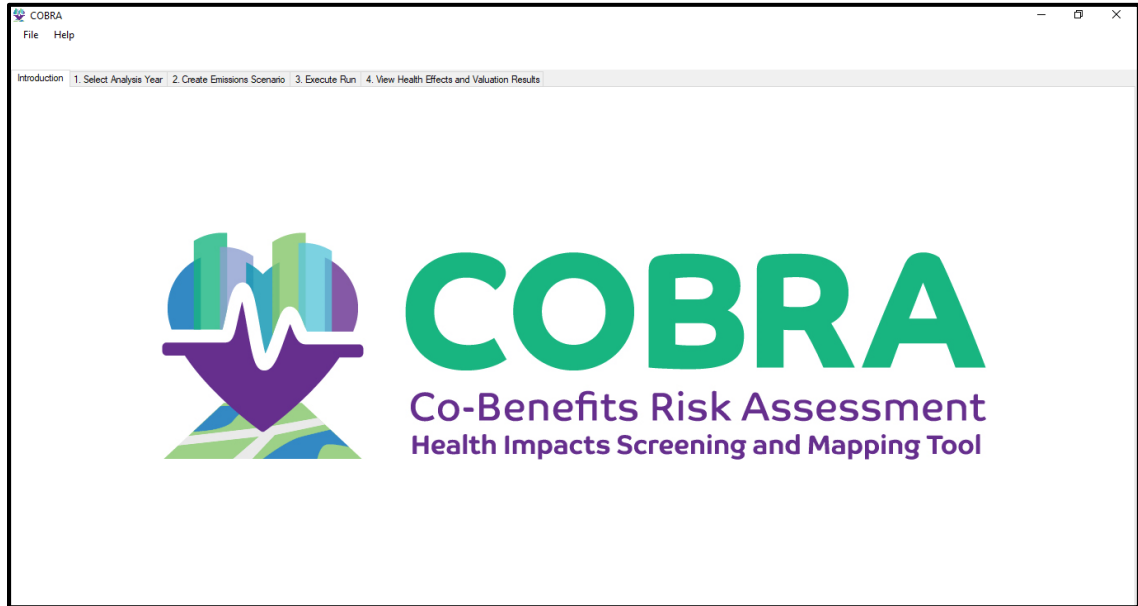
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<sup>5</sup> There is a large literature regarding the health impacts of air pollution and approaches to value these impacts. COBRA uses a subset that EPA deems most credible. More sophisticated users interested in using additional approaches may want to use EPA's Environmental Benefits Mapping and Analysis Program (BenMAP), which is available at: <https://www.epa.gov/benmap>.

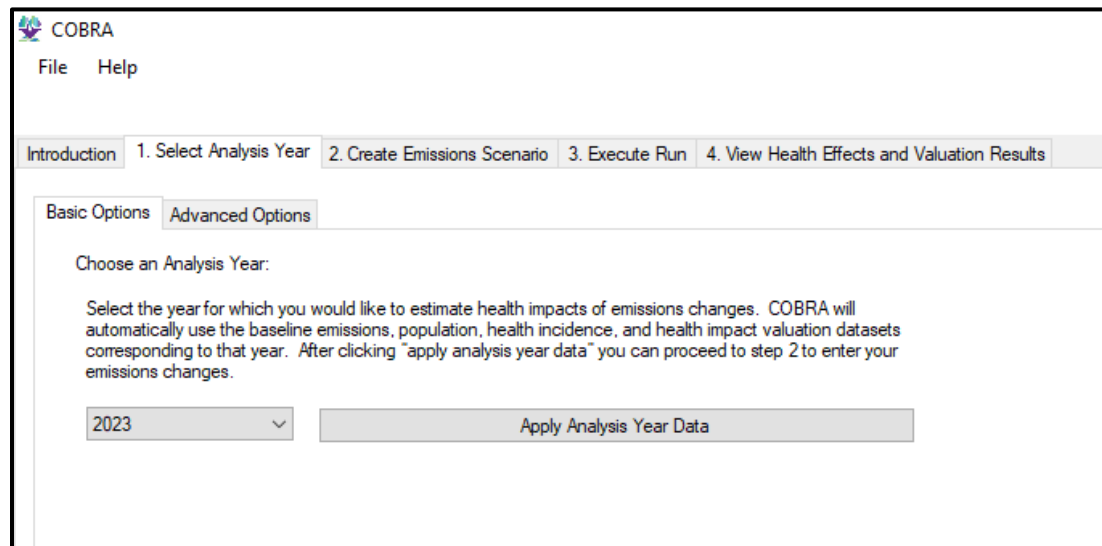


## Overview of Model

The COBRA screening model is a stand-alone Windows application that contains all of the data needed for the analysis of alternative emissions scenarios; the user is only required to enter changes in emissions. Upon launching the model, you will see the **Introduction** screen.

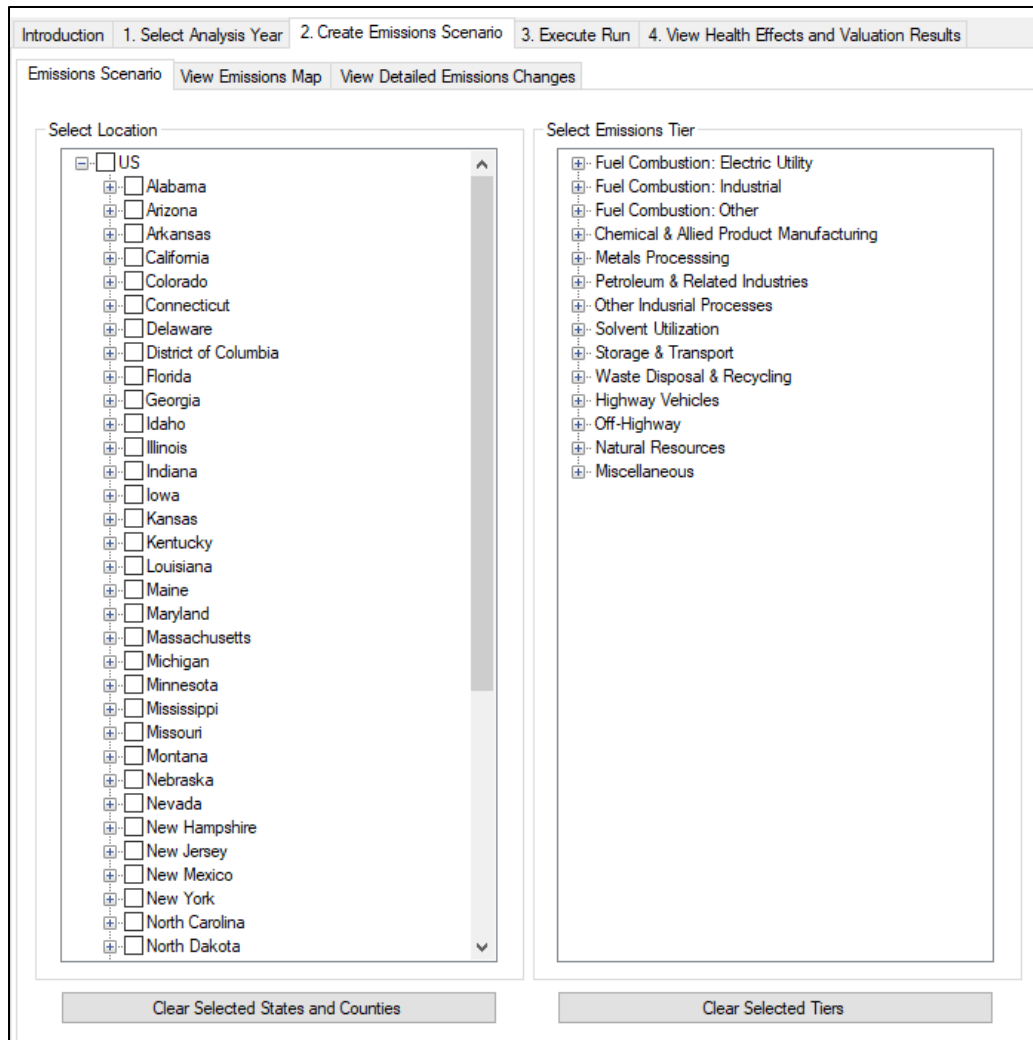


Once you are ready to run a comparison, go to the **1. Select Analysis Year** screen and select the analysis year of interest. Advanced users can load a customized baseline emissions inventory at this stage, which is discussed in more detail in Chapter 3.



Then, go to the **2. Create Emissions Scenario** screen. Use the ‘Select Location’ panel on the top left of the **Emissions Scenario** tab to indicate the geographic level at which you wish to make your emissions changes. ‘US’ means that any emissions changes will be applied to all sources in that category throughout the entire U.S. Alternatively, you can enter emissions changes for selected states or selected counties.

Next, select the category of emissions that a policy or action is expected to affect in the ‘Select Emissions Tier’ panel. For example, to assess the impacts of a renewable energy or energy efficiency policy that is expected to affect utility-related emissions, you would select ‘Fuel Combustion: Electric Utility’ as the first tier. COBRA provides three levels of emissions sources (tiers) in a directory tree structure. If you know the specific fuel source within the utility category that would be reduced or displaced (e.g. coal or natural gas), you can select the appropriate second tier. If you do not know the specific fuel source within the utility category that would be reduced or displaced, you can enter emissions changes at the tier 1 level. For a policy that involves fuel switching or reductions in transportation through vehicle miles traveled, for example, you would select ‘Highway Vehicles’ as the first tier. If you knew the specific fuel (e.g. diesel) that would be reduced, you would select it in the second tier.



Once your geography is determined, you can enter your emissions changes for the nation, a single state, groups of states, a single county, or groups of counties, depending on your previous selection.

**Exhibit 1. Basic Tools and Data Sources for Determining Emission Reductions**

Online Tool	Description
EPA’s Emissions & Generation Resource Integrated Database (eGRID) ( <a href="https://www.epa.gov/energy/emissions-generation-resource-integrated-database-egrid">https://www.epa.gov/energy/emissions-generation-resource-integrated-database-egrid</a> )	Provides data on the environmental characteristics of electric generation by power plants in the United States.
EPA’s AVOIDed Emissions and geneRation Tool (AVERT) ( <a href="https://www.epa.gov/statelocalenergy/avoided-emissions-and-generation-tool-avert">https://www.epa.gov/statelocalenergy/avoided-emissions-and-generation-tool-avert</a> )	Estimates displaced emissions (at the county, state, and regional levels) at electric power plants due to renewable energy or energy efficiency policies and programs.
EPA’s Motor Vehicle Emission Simulator (MOVES) ( <a href="https://www.epa.gov/moves">https://www.epa.gov/moves</a> )	Estimates emissions from mobile sources, including emissions from cars, trucks, and motorcycles.
National Emissions Inventory ( <a href="https://www.epa.gov/air-emissions-inventories/national-emissions-inventory-nei">https://www.epa.gov/air-emissions-inventories/national-emissions-inventory-nei</a> )	Allows users to view emissions by sector (for 60 emissions inventory sectors) for specific pollutants at varying levels of geographic aggregation.
Power Profiler ( <a href="https://www.epa.gov/energy/power-profiler">https://www.epa.gov/energy/power-profiler</a> )	Allows users to view the emissions that can be attributed to electricity use in homes or businesses.

*Note:* For more details on these basic tools and on other methods, see: (1) Chapter 4 of EPA’s “Quantifying the Multiple Benefits of Energy Efficiency and Renewable Energy: A Guide for State and Local Governments” report (U.S. EPA, 2018), available at <https://www.epa.gov/statelocalenergy/quantifying-multiple-benefits-energy-efficiency-and-renewable-energy-guide-state>; or (2) Appendix I of EPA’s “Roadmap for Incorporating Energy Efficiency/Renewable Energy Policies and Programs into State and Tribal Implementation Plans” (U.S. EPA, 2012c), available at <https://www.epa.gov/energy-efficiency-and-renewable-energy-sips-and-tips>.

Once you have determined the appropriate tier category and location, enter the emission changes for one or more of the five included pollutants in the ‘2. Create Emissions Scenario’ panel at the top right of the COBRA screen. You can enter emissions changes in tons or percentages. Absolute emission reductions in tons can be estimated using a variety of methods. See Exhibit 1 above for a description of a few basic methods. Percentage reductions can be used to assess the benefits of a goal that results in reductions in activity levels or emissions from a particular source (or group of sources), such as a renewable portfolio standard, transportation policies requiring reductions in vehicle miles traveled, and energy efficiency programs.

For example, EPA’s Emissions & Generation Resource Integrated Database (eGRID) website provides electric generation data and corresponding emissions rates for the

United States.<sup>6</sup> On the eGRID website, click on ‘**eGRID2018 Summary Tables (PDF)**.’<sup>7</sup> Using the Western Electricity Coordinating Council (WECC) Southwest Region (which includes Arizona and New Mexico) as an example, you can obtain the following information:

- **Electric Generation:** The “Subregion Resource Mix (eGRID 2018)” table on page 3 summarizes electric generation data by region. The net generation for the WECC Southwest Region in 2018 was approximately 165,353,383 megawatt hours (MWh).
- **Emissions Rates:** The “Subregion Output Emission Rates (eGRID 2018)” table on page 2 provides the WECC Southwest Region’s non-baseload output emissions rate for SO<sub>2</sub>: 0.3 lbs. per MWh.<sup>8</sup>

If a policy is expected to reduce electric generation by 2% in the WECC Southwest Region, you can calculate the reduction in MWh:  $2\% \times 166,327,576 \text{ MWh} = 3,307,068 \text{ MWh}$ . You can then calculate the emission reductions as:

$$\text{Emission Reduction} = 3,326,552 \text{ MWh} \times 0.3 \text{ lbs. SO}_2 \text{ per MWh} = 992,120 \text{ lbs. SO}_2.$$

This reduction is equal to about 496 tons ( $992,120 \text{ lbs.} \div 2000 \text{ lbs. per ton}$ ) of SO<sub>2</sub>.<sup>9</sup>

After you have calculated emissions changes, you can enter these changes for as many tier categories as you wish, and you can enter different sets of changes for each state or county (or groups of states and/or counties, if you choose to select more than one). After each change in emissions for a state, county, or group of states or counties, click **Apply Changes**.

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<sup>6</sup> While eGRID provides a basic approach for estimating changes in emissions from energy efficiency and renewable energy using annual data, another useful resource available for estimating changes in emissions is EPA’s **Avoided Emissions and Generation Tool (AVERT)**. AVERT enables users to estimate the regional, state, and county-level emission impacts of different energy efficiency and/or renewable energy programs based on temporal energy savings and hourly generation profiles. For more information or to download AVERT, see: <https://www.epa.gov/statelocalenergy/avoided-emissions-and-generation-tool-avert>.

<sup>7</sup> Note that because the latest edition of eGRID was developed using 2018 data, it would be most appropriate to choose the 2016 baseline for the analysis in COBRA.

<sup>8</sup> Non-baseload emissions come from power plants that dispatch to the grid when demand for power exceeds baseload needs (U.S. EPA, 2020).

<sup>9</sup> For more information on the benefits of reductions in SO<sub>2</sub> emissions, see EPA’s “Quantifying the Multiple Benefits of Energy Efficiency and Renewable Energy: A Resource for State and Local Governments” report (U.S. EPA, 2018), available at: <https://www.epa.gov/statelocalenergy/quantifying-multiple-benefits-energy-efficiency-and-renewable-energy-guide-state>.

**Modify Emissions**

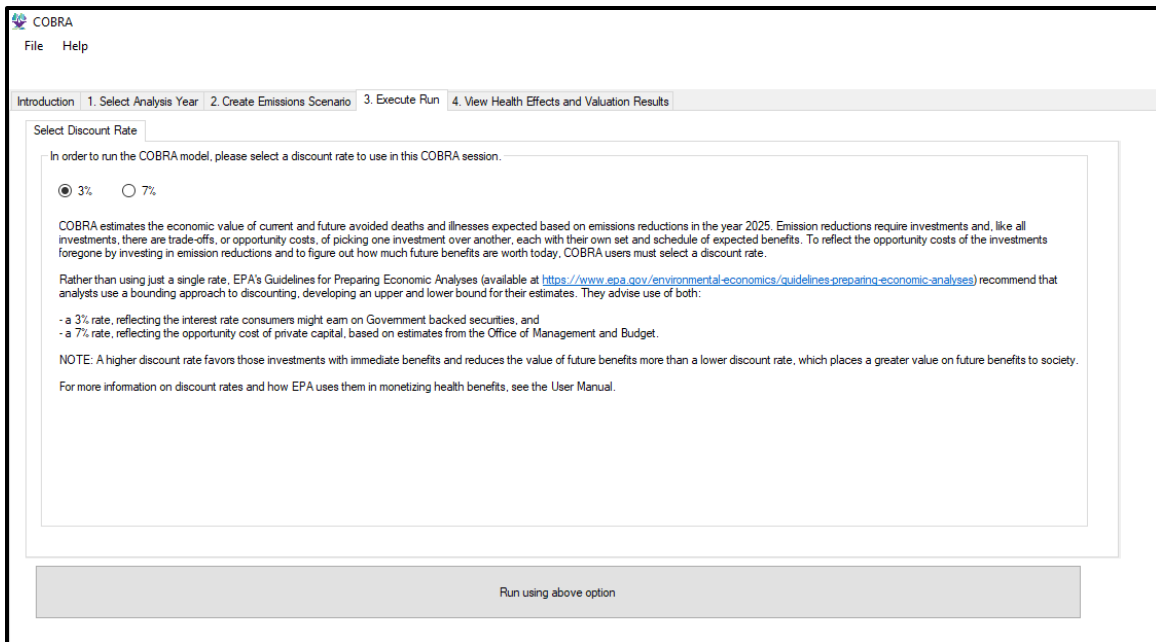
**Emissions Scenario / Baseline**

PM 2.5	<input checked="" type="radio"/> reduce by <input style="width: 80px;" type="text" value="25.00"/> <input type="radio"/> increase by	<input checked="" type="radio"/> pct <input type="radio"/> tons	6,508.58 [tons] / 8,678.10 [tons]
SO2	<input checked="" type="radio"/> reduce by <input style="width: 80px;" type="text" value="25.00"/> <input type="radio"/> increase by	<input checked="" type="radio"/> pct <input type="radio"/> tons	7,234.32 [tons] / 9,645.76 [tons]
NOx	<input checked="" type="radio"/> reduce by <input style="width: 80px;" type="text" value="25.00"/> <input type="radio"/> increase by	<input checked="" type="radio"/> pct <input type="radio"/> tons	21,729.93 [tons] / 28,973.24 [tons]
NH3	<input checked="" type="radio"/> reduce by <input style="width: 80px;" type="text" value="25.00"/> <input type="radio"/> increase by	<input checked="" type="radio"/> pct <input type="radio"/> tons	2,217.58 [tons] / 2,956.78 [tons]
VOC	<input checked="" type="radio"/> reduce by <input style="width: 80px;" type="text" value="25.00"/> <input type="radio"/> increase by	<input checked="" type="radio"/> pct <input type="radio"/> tons	1,488.53 [tons] / 1,984.71 [tons]

To review your scenario, click **View Detailed Emissions Changes**.

The data grid below will show a detailed record of the emissions changes occurring under the current scenario.														
RPS	State	County	TIER 1	TIER 2	TIER 3	TYPE	MODIFIED	Base NO2	Base SO2	Base NH3	Base PM 2.5	Base VOC	Control NO2	Control SO2
42011	Pennsylvania	Berks	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	9.5676	0.0051456	0.0274968	0.00041808	2.92656	7.1757	0.0038592
42017	Pennsylvania	Bucks	Fuel Combustion: Electric...	Gas	Natural	MEDIUM	<input checked="" type="checkbox"/>	0.4889828	0.045024	0.2412	1.484184	0.4134168	0.3667446	0.033768
42029	Pennsylvania	Chester	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	1.83312	0.0101304	0.0540288	0.1316952	0.0927816	1.37484	0.0075978
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	184.68270501	0	7.54521435	65.13290357	12.96833713	138.5120287...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	247.92400974	0	11.67653782	72.86463209	20.07420561	185.9430073...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	242.32358071	0	11.57096164	72.18726268	19.68759031	181.7426855...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	182.83780898	0	7.44744728	104.50389793	12.80030007	137.1283567...	0
42043	Pennsylvania	Dauphin	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	55.6305288	0.234768	1.1619408	1.7421072	13.0953304	41.7328896	0.176074
42045	Pennsylvania	Delaware	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	30.2477664	0.1505088	0.8175072	1.5816208	1.9405344	22.4658248	0.1128814
42045	Pennsylvania	Delaware	Fuel Combustion: Electric...	Gas	Natural	MEDIUM	<input checked="" type="checkbox"/>	36.06874299	0	1.7265092	16.56981598	2.96493122	27.05153724...	0
42063	Pennsylvania	Indiana	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	5.1	0.025	0	0.3	0.1	3.825	0.01875
42069	Pennsylvania	Lackawanna	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	33.64728699	0.06292021	0.4786762	10.33628192	42.30395598	25.33546524...	0.0487191575
42069	Pennsylvania	Lackawanna	Fuel Combustion: Electric...	Gas	Process	LOW	<input checked="" type="checkbox"/>	7.11264498	6.37493143	0.19152684	4.66293202	16.92122806	5.334833735	4.7811985725
42071	Pennsylvania	Lancaster	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	0.220296	0	0.004884255	0.070752	0	1.66232	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	63.21001401	0	2.51760044	10.0876373	4.32172573	47.40795109...	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	43.79603242	0	1.73209076	6.94544552	2.97874968	32.847024315	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	36.00767491	0	1.68448555	6.7506578	2.89520955	27.0057561...	0
42079	Pennsylvania	Luzerne	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	0	0	7.375	0	0	0	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	5.95185	0.04874	0.17938	0.77734	0.32785	4.46833875	0.036555
42095	Pennsylvania	Northampton	Fuel Combustion: Electric...	Gas	Process	LOW	<input checked="" type="checkbox"/>	54.2526502	34.19367821	0.20799838	14.20915265	17.84509687	40.68947765	25.64525865...
42095	Pennsylvania	Northampton	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	1411.6922768	0	44.1153837	94.37208941	75.83331576	1058.7692076	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	1400.7110922	0	43.77221169	93.63799369	75.23350599	1050.533319...	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric...	Gas	Natural	HIGH	<input checked="" type="checkbox"/>	0	0	32.14625	0	0	0	0
42097	Pennsylvania	Northumberland	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	0.75576	0.0045024	0.03216	0.06432	0.04824	0.56662	0.0033768
42101	Pennsylvania	Philadelphia	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	4.7688652	0.8060904	0.9641568	0.5738952	1.6520592	3.5162724	0.6045678
42105	Pennsylvania	Potter	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	2.8932	0.03	0	0.3296196	0.1065	2.1699	0.0225
42111	Pennsylvania	Somerset	Fuel Combustion: Electric...	Gas	Natural	LOW	<input checked="" type="checkbox"/>	2.2280448	0.0744504	0	0.5184192	0.2172408	1.6711036	0.058878
42111	Pennsylvania	Somerset	Fuel Combustion: Electric...	Gas	Process	LOW	<input checked="" type="checkbox"/>	2.4390144	1.5793776	0	0.4024824	0.2095224	1.8292608	1.1845332

Then, go to the **3. Execute Run** screen to select settings for running your scenario. This tab asks you to choose a discount rate (described in more detail in the Chapter 2 Tutorial). Choose a 3% or 7% discount rate and click **Run using above options** to run the comparison between the scenario you have just created and the baseline scenario.



The model may take 5 to 15 minutes to run, depending on your computer. When the model is done running, you can examine the results in the **4. View Health Effects and Valuation Results** tab. Regardless of the geographic level at which you made your emissions changes, you can examine the results for every county in every state in the country. For each county, COBRA calculates three types of results: the change in ambient PM<sub>2.5</sub> concentration; the change in health effects associated with that change; and the dollar value associated with the change in health effects.

These results can be viewed in a table (in the **Table** tab) or geographically on a map of the United States (in the **Map** tab).

Introduction	1. Select Analysis Year	2. Create Emissions Scenario	3. Execute Run	4. View Health Effects and Valuation Results						
FIPS	State	County	Base PM 2.5	Control PM 2.5	Delta PM 2.5	\$ Total Health Benefits (low estimate)	\$ Total Health Benefits (high estimate)	Mortality (low estimate)	\$ Mortality (low estimate)	Mortality
Contains: Pennsylvania			Total: 245,785,482.65			Total: 553,903,597.88		Total: 22,4342		Total: 242,383,186.06
42001	Pennsylvania	Adams	7.701	7.662	0.0399	2,969,781.27	6,720,499.73	0.2735	2,654,981.43	
42003	Pennsylvania	Allegheny	8.455	8.44	0.025	20,544,170.44	46,297,534.64	1.8783	20,293,163.88	
42005	Pennsylvania	Armstrong	8.542	8.488	0.0536	2,919,234.59	6,586,695.94	0.2673	2,887,775.62	
42007	Pennsylvania	Beaver	7.566	7.549	0.0163	2,256,916.42	5,088,723.32	0.2066	2,232,404.28	
42009	Pennsylvania	Bedford	7.358	7.343	0.0154	574,548.95	1,297,198.06	0.0526	568,434.13	
42011	Pennsylvania	Berks	8.931	8.888	0.0446	11,254,780.31	25,355,547.45	1.0251	11,075,055.76	
42013	Pennsylvania	Blair	7.549	7.532	0.0173	1,739,592.58	3,920,842.85	0.1592	1,720,451.42	
42015	Pennsylvania	Bradford	6.703	6.67	0.0338	1,542,610.36	3,473,030.83	0.141	1,523,754.78	
42017	Pennsylvania	Bucks	8.155	8.116	0.0395	18,848,284.69	38,020,331.22	1.5402	16,640,163.2	
42019	Pennsylvania	Butler	8.144	8.135	0.0087	1,245,720.71	2,806,831.09	0.114	1,231,275.49	
42021	Pennsylvania	Cambria	7.458	7.438	0.02	2,211,530.01	4,993,319.16	0.2027	2,189,535	
42023	Pennsylvania	Cameron	6.822	6.805	0.0169	65,010.96	146,611.09	0.006	64,407.48	
42025	Pennsylvania	Carbon	7.781	7.742	0.0388	2,163,042.62	4,880,616.51	0.1982	2,141,764.04	
42027	Pennsylvania	Centre	7.27	7.254	0.016	1,030,705.48	2,316,642.27	0.0933	1,007,787.14	
42029	Pennsylvania	Chester	8.494	8.471	0.0227	6,562,779.87	14,778,712.6	0.5981	6,461,769.92	
42031	Pennsylvania	Clarion	7.831	7.812	0.0186	494,450.56	1,117,297.32	0.0452	488,696.06	
42033	Pennsylvania	Clearfield	7.327	7.3	0.0269	1,658,951.07	3,744,108.37	0.1518	1,640,242.4	
42035	Pennsylvania	Clinton	7.016	6.997	0.0194	473,027.88	1,066,979.56	0.0432	466,739.56	
42037	Pennsylvania	Columbia	7.715	7.688	0.0274	1,228,174.45	2,769,181.19	0.1122	1,212,271.79	
42039	Pennsylvania	Crawford	7.237	7.231	0.0066	493,807.8	977,849.34	0.0397	428,677.75	
42041	Pennsylvania	Cumberland	8.312	8.292	0.0204	3,402,239.97	7,662,901.93	0.3109	3,358,883.71	
42043	Pennsylvania	Dauphin	8.528	8.488	0.0407	6,438,573.31	14,496,895.6	0.5883	6,323,521.34	
42045	Pennsylvania	Delaware	8.405	8.373	0.0322	11,014,454.62	24,812,407.3	1.0015	10,820,137.6	
42047	Pennsylvania	Elk	7.007	6.987	0.0199	499,828.37	1,113,039.96	0.0453	489,102.93	
42049	Pennsylvania	Erie	6.901	6.895	0.0064	1,233,405.24	2,780,101.84	0.1127	1,217,283.13	
42051	Pennsylvania	Fayette	7.17	7.157	0.0128	1,487,885.15	3,305,604.5	0.1344	1,452,180.36	
42053	Pennsylvania	Forest	7.247	7.236	0.0112	65,180.76	147,273.07	0.006	64,542.81	
42055	Pennsylvania	Franklin	7.468	7.459	0.0173	1,748,886.69	3,935,771.38	0.1596	1,732,188.54	
42057	Pennsylvania	Fulton	7.405	7.392	0.0135	138,648.93	312,465.85	0.0127	136,966.99	
42059	Pennsylvania	Greene	5.757	5.751	0.0063	147,281.98	332,605.04	0.0135	145,661.39	

\$ Nonfatal Heart Attacks (low estimate)	Nonfatal Heart Attacks (high estimate)	\$ Nonfatal Heart Attacks (high estimate)
Equals: Total: 280,951.39	Equals: Total: 19,8014	Equals: Total: 2,609,601.82
2,364.69	0.1667	21,962.38
20,245.58	1.4271	188,071.33
3,173.62	0.2236	29,472.43
2,312.22	0.163	21,481.3
658.76	0.0464	6,120.15
13,808.42	0.9731	128,246.69
1,732.23	0.1221	16,092.84
1,801.38	0.127	16,732.38
21,020.29	1.4814	195,238.01
1,406.55	0.0992	13,068.43
2,262.5	0.1595	21,018.63
75.81	0.0053	704.26
2,293.23	0.1616	21,299.83
1,528.47	0.1077	14,200.04
9,149.62	0.645	84,997.42
553.65	0.039	5,143.46
1,845.57	0.1301	17,144.1
537.17	0.0379	4,990.32
1,408.59	0.0993	13,084.81
466.34	0.0329	4,332.89
4,105.03	0.2894	38,135.54
8,045.91	0.587	74,730.05
12,601.36	0.8882	117,051.52
554	0.0391	5,146.68
1,317.07	0.0929	12,237.37
1,438.71	0.1014	13,366.59
74.15	0.0052	688.94
2,178.57	0.1536	20,239.53
169.42	0.0119	1,574
159.67	0.0113	1,483.57

The results table includes a row with blue text that summarizes the total impact of the scenario. To view the impact of the scenario on a specific state, filter the “state” column for the state of interest. For example, results in the table above show estimated impacts of the scenario for Pennsylvania. According to these results, the emissions reductions in 2023 would result in the following health effects in Pennsylvania:



- 22.4 to 50.7 avoided premature adult deaths (over the next 20 years), valued at an estimated \$242 and \$548 million;
- 2.1 to 19.8 avoided nonfatal heart attacks (in 2023), valued at an estimated \$280,951, to \$2,609,602 and
- 12.9 avoided asthma emergency room visits (in 2023), valued at an estimated \$8,093.

Note that health effects are not necessarily whole numbers. This is because COBRA calculates small statistical risk reductions which are then aggregated over the population. For example, if 150,000 people experience a 0.001% reduction in mortality risk, this would be reported as 1.5 “statistical lives saved.” This statistical life and its associated monetary value represents the sum of many small risk reductions and does not correspond to the loss or value of an individual life. Users may want to round these values to the nearest whole before presenting the results.

Result sets can be exported for use in outside programs and presentations. To save your results in a table form, click on **Export to CSV** or **Export to Excel** (depending on your preferred format) in the **Table** tab. To save your results in map form, navigate to the **Map** tab, right click on the data in the ‘Legend’ panel, then click **Data...Export Data**.

## Caveats and Limitations

There are limitations to the COBRA screening model that make it inappropriate for certain types of analyses:

- *Determination of attainment.* Modeling the attainment of National Ambient Air Quality Standards (NAAQS) requires more sophisticated air quality modeling than that currently built into COBRA.
- *Estimating dynamic market effects.* COBRA does not account for changes in emissions that can result from changes in electricity market responses to policy. For example, emissions in some states and regions are “capped” by laws or regulations and emission allowances can then be traded across entities within a capped region. In these regions, a reduction in emissions in one location *may* result in an increase (rebound) in emissions in another area subject to the cap. COBRA does not automatically capture these types of potential effects in electricity market dispatch, so care should be exercised when interpreting COBRA results to analyze the net impacts of a change in policy. COBRA is more suited to an attributable risk assessment, which addresses the magnitude of an emission source and the impact of controlling its emissions. That information can be used to develop policies targeted to the appropriate sources.

Because COBRA is intended primarily as a screening tool, it uses a relatively simple air quality model, which introduces additional uncertainty. While comparative work to test the performance of COBRA's air quality model is ongoing, it is not yet fully validated.

As with more complex air pollution benefits models, there is substantial uncertainty surrounding the values of key inputs to COBRA – in the air quality model, emissions inventory, health impact functions, and economic values – and users should exercise caution when interpreting the results of analyses.

Some of the uncertainty in COBRA reflects variability (for example, a health impact function that is appropriate for one location may not be appropriate for another location if the function actually varies across locations). Much of the uncertainty, however, reflects the insufficient level of knowledge about the true values of model inputs.

The appendices discuss these issues and provide sources for additional information. However, developing a quantified confidence interval for the results is beyond the scope of this model. As an alternative, users should consider using sensitivity analyses to determine how their conclusions might change with differences in the location and amount of emissions. When more detailed analyses are required, users should be cognizant of the model's limitations, and consider using more sophisticated modeling approaches.

**Chapter 2** of this User's Manual provides a quick tutorial for the new user. Baseline data are examined, and a simple new scenario is defined and run, and the results are displayed in tables and maps. Subsequent chapters provide more detailed information on each step, and describe additional options you can use for more complex analyses.

**Chapter 3** describes the process of selecting and exploring baseline data.

**Chapter 4** provides details on different ways to define your new scenario, and run the comparison between it (the control scenario) and the baseline scenario.

**Chapter 5** describes the different ways to view and save your results.

**Chapter 6** explains how to use COBRA's mapping functionality.

A **Glossary** is provided at the end of the manual.

### **Additional Information**

The Appendices to this manual provide additional information on the methods and assumptions used in the model.

- **Appendix A: Description of Source-Receptor Matrix and Emissions Data.** Describes the source-receptor matrix embedded within the model that translates the air pollution emissions changes into changes in ambient particulate matter.

- **Appendix B: Derivation of Health Impact Functions.** Explains the derivation of the types of health impact functions used in COBRA.
- **Appendix C: COBRA Health Impact Functions.** Provides an overview of all the functions used to convert changes in ambient PM<sub>2.5</sub> into health effects.
- **Appendix D: Baseline Incidence Rates for Adverse Health Effects.** Lists the baseline incidence rates for each of the types of adverse health effects.
- **Appendix E: Population Forecasts.** Describes the forecasting procedure.
- **Appendix F: Economic Value of Health Effects.** Lists the equations and sources of the values used to monetize the health effects.
- **Appendix G: Instructions for Batch Functionality.** Explains how to run multiple COBRA scenarios in batch mode.
- **Appendix H: References.** Provides all of the sources referenced in the Appendices or used in the model.

## CHAPTER 2. Quick-Start Tutorial

This tutorial will give you a quick introduction to how COBRA works, and how to work through the steps of a simple analysis. As described in the text box below, COBRA v3.3 also includes more advanced options for users who want to change underlying assumptions (e.g. population, emissions baseline). Subsequent chapters describe how to use the more advanced features.

COBRA allows you to estimate the impact of a change in air pollution resulting from a new policy or other type of change. In this example, you will consider changes in one state (we have arbitrarily selected Pennsylvania) that result in a decrease in emissions from electricity generating plants. If a statewide plan to switch 25 percent of electricity generation to renewable sources were put into effect, what would be the difference in ambient particulate matter levels and health effects, compared to business-as-usual? This tutorial will show you how to use COBRA to examine this type of scenario through the following steps:

**Step 1.** Select the analysis year and view the baseline emissions data.

**Step 2.** Select the geography and tier category for emissions changes and define the emissions changes

**Step 3.** Select a discount rate and run the comparison.

**Step 4.** View and save your results.

To open COBRA, click **Start... All Programs... COBRA... COBRA** The model will open and COBRA will display the main screen. You will see five tabs at the very top: **Introduction, 1. Select Analysis Year, 2. Create Emissions Scenario, 3.**

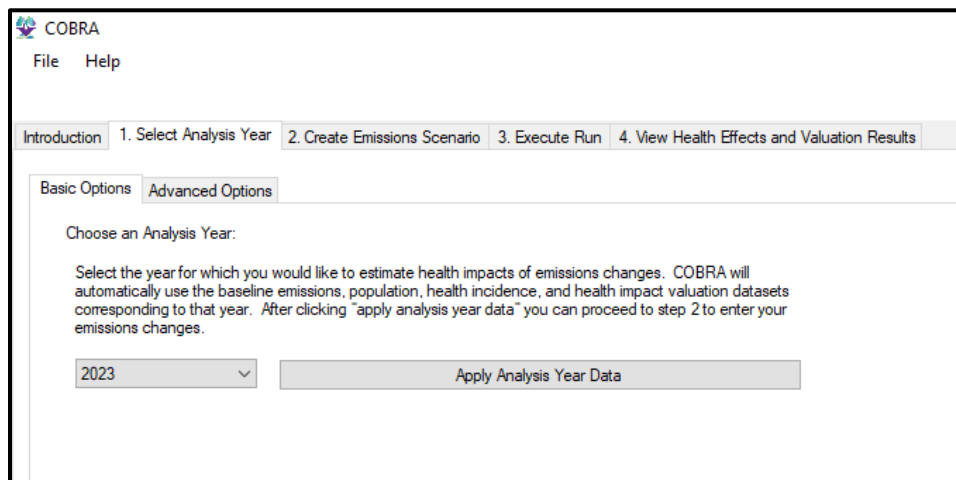
Compared to previous versions, COBRA (3.0) offers additional functionality and flexibility for advanced users, while still being easy to operate for basic analyses. The table below compares COBRA's advanced options to the simpler, quick-start options. More advanced options will be discussed in subsequent chapters.

Quick-Start Options	Advanced Options
Preloaded 2016, 2023, and 2028 emissions baseline	Custom emissions baseline
Preloaded 2016, 2023, and 2028 incidence data	Custom incidence data
Preloaded 2016, 2023, and 2028 population data	Custom population data
Preloaded health effect functions	Custom health effect functions
Preloaded 2016, 2023, and 2028 valuation functions	Custom valuation functions
Enter emissions scenario in user interface	Upload custom emissions scenario
Run COBRA separately for each emissions scenario	Run multiple emissions scenarios in batch mode
View default map output in COBRA	Customize map appearance and export for further

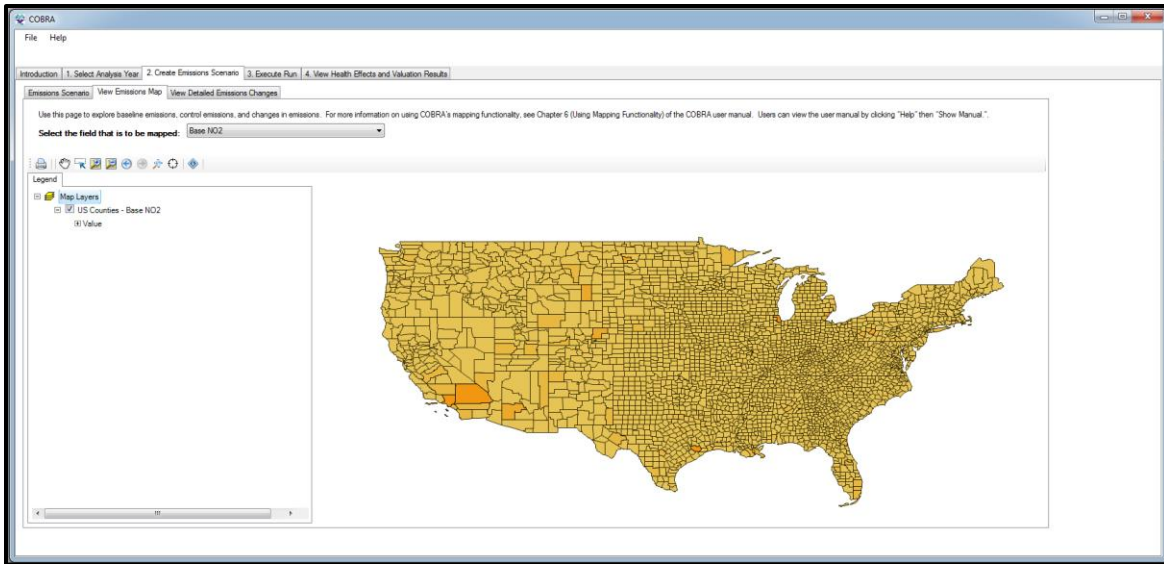
**Execute Run** and **4. View Health Effects and Valuation Results**. Below, we provide detailed descriptions of the steps needed to run a scenario.

### Step 1. Select the baseline data.

Click **1. Select Analysis Year** and you will see two tabs: **Basic Options** and **Advanced Options**. To do a basic screening analysis for analysis year 2023, select “2023” in the drop down menu and click **Apply selected analysis year** in the **Basic Options** tab.

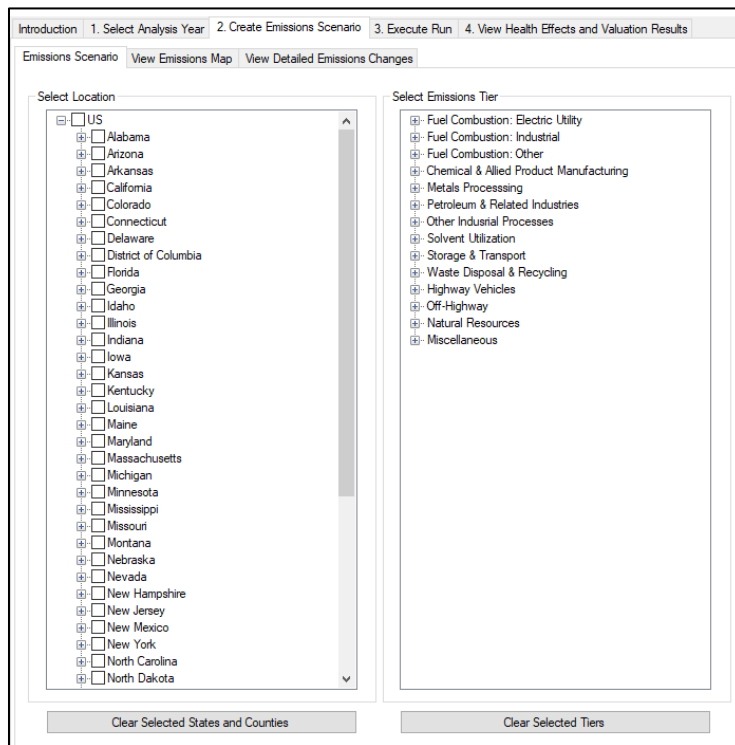


To explore the preloaded baseline emissions data, go to the **2. Create Emissions Scenario** tab. On the top of this screen you will see three tabs: **Emissions Scenario**, **View Emissions Map**, and **View Detailed Emissions Changes**. Click on the **View Emissions Map** tab. At the top of this screen, you will see a drop-down menu labeled ‘Select the field that is to be mapped.’ The first five options listed (Base NH<sub>3</sub>, Base NO<sub>2</sub>, Base PM<sub>2.5</sub>, Base SO<sub>2</sub>, and Base VOC) correspond to the baseline emissions of ammonia (NH<sub>3</sub>), nitrogen oxides (NO<sub>x</sub>), fine particulate matter (PM<sub>2.5</sub>), sulfur dioxide (SO<sub>2</sub>), and volatile organic compounds (VOCs), respectively, from all sectors. Select any of these emissions to view a map of county-level baseline emissions. See Chapter 6 for more details on how to use COBRA’s mapping functionality.



## Step 2. Define the emissions changes and select the geography for emissions changes.

Now that you have seen the baseline emissions, you can define the new (control) scenario to compare to the baseline scenario. Click on the **2. Create Emissions Scenario** tab. On the top of this screen you will see three tabs: **Emissions Scenario**, **View Emissions Map**, and **View Detailed Emissions Changes**. Click on the **Emissions Scenario** tab.



In the top left panel you can select the geographic area to which you want to apply the emissions changes. Note that once your comparison is run, you will be able to see results (changes in ambient PM<sub>2.5</sub> levels and health effects) for the entire country (although only a subset of states usually experience the PM<sub>2.5</sub> reductions and health benefits of a defined scenario). Since this example only analyzes changes statewide in Pennsylvania, check the box next to ‘Pennsylvania’ in the list.

If you wanted to vary the emissions changes across counties, or only make changes in some counties, you could click the ‘+’ button next to Pennsylvania and enter different emissions changes for each county. For instance, if you know the counties in which sources that are likely to be affected (such as power plants) are located, you can enter emissions changes in those counties only. However, in this example you are looking at a statewide change.

The next step is to define the categories of emissions affected. The middle panel contains a directory tree with all the tier 1, 2, and 3 source categories (see Appendix A for a list of source categories and their emissions). You can define emissions changes at any level, but each level always includes all the levels indented underneath it. In this example, you want to change all of the source categories that are electrical utilities, so click ‘Fuel Combustion: Electric Utilities.’

The panel on the right-hand side of the screen lists each pollutant included in the model. Since this scenario reduces all baseline emissions by 25 percent, type ‘25’ in the box next to each pollutant. The default selections are ‘reduce by’ and ‘percent’; leave them as they are. Chapter 4 describes how to create more sophisticated emissions scenarios, such as scenarios that cover multiple geographies or involve different emissions changes for different pollutants. Click **Apply Changes** to save your changes. The changes apply to all the locations selected.

If you wanted to enter additional emission changes in another location, you would click **Clear Selected States and Countries**, then repeat the process of selecting a new geographic location tier and entering the emission change for that location. You must click **Apply Changes** after entering emissions changes for each location. If you make a mistake or want to start over, click the **Reset to Baseline** button, which will erase all entries. In this example, we are running the analysis for a single scenario affecting only one state, so we will proceed without applying any additional emissions changes.

**NOTE:** Users of EPA’s AVERT model can upload the emissions changes quantified in AVERT into COBRA so that the user does not have to enter all the changes manually. Chapter 4 describes the process for uploading AVERT outputs as COBRA inputs.

You can review your emissions scenario in table form by clicking the **View Detailed Emissions Changes** tab or in map form by clicking the **View Emissions Map** tab.

COBRA

File Help

Introduction 1. Select Analysis Year 2. Create Emissions Scenario 3. Execute Run 4. View Health Effects and Valuation Results

Emissions Scenario View Emissions Map View Detailed Emissions Changes

The data grid below will show a detailed record of the emissions changes occurring under the current scenario.

FIPS	State	County	TIER 1	TIER 2	TIER 3	TYPE	MODIFIED	Base NO2	Base SO2	Base NH3	Base PM 2.5	Base VOC	Control NO2	Control SO2
42011	Pennsylvania	Berks	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	9.5676	0.0051456	0.0274968	0.00041808	2.92656	7.1757	0.0038592
42017	Pennsylvania	Bucks	Fuel Combustion: Electric... Gas	Natural	MEDIUM		<input checked="" type="checkbox"/>	0.4889928	0.045024	0.2412	1.484184	0.4134168	0.3667446	0.033768
42029	Pennsylvania	Chester	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	1.83312	0.0101304	0.0540288	0.1316992	0.0927816	1.37484	0.0075978
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	184.68270501	0	7.54521435	65.13290357	12.06833713	138.5120287...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	247.92400974	0	11.67953782	72.88463209	20.07420561	185.9430073...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	242.32358071	0	11.57096164	72.18726268	19.88759031	181.7428655...	0
42033	Pennsylvania	Clearfield	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	182.83780888	0	7.44744728	104.50389793	12.80000007	137.1283567...	0
42043	Pennsylvania	Dauphin	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	55.6305288	0.234758	1.1619408	1.7421072	13.0962304	41.7228966	0.176076
42045	Pennsylvania	Delaware	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	30.2477664	0.1505088	0.8175072	1.5816288	1.9405344	22.8655248	0.1128816
42045	Pennsylvania	Delaware	Fuel Combustion: Electric... Gas	Natural	MEDIUM		<input checked="" type="checkbox"/>	36.06874299	0	1.72505092	16.5696198	2.9649122	27.05155724...	0
42063	Pennsylvania	Indiana	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	5.1	0.025	0	0.3	0.1	3.825	0.01875
42069	Pennsylvania	Lackawanna	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	33.44728699	0.06229221	0.47854763	10.33828192	42.30385598	25.23546524...	0.0487191575
42069	Pennsylvania	Lackawanna	Fuel Combustion: Electric... Gas	Process	LOW		<input checked="" type="checkbox"/>	7.11264498	6.37493143	0.19152464	4.66293202	16.9212806	5.334483735	4.7811985725
42071	Pennsylvania	Lancaster	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	0.230296	0	0	0.004888455	0.070752	0.165222	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	63.21001401	0	2.51760044	10.0878273	4.32712573	47.4075105...	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	43.79603242	0	1.73209076	6.94544552	2.97874968	32.84702315	0
42073	Pennsylvania	Lawrence	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	36.00767491	0	1.68448555	6.7506578	2.8952055	27.0057561...	0
42079	Pennsylvania	Lucerne	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	0	0	7.375	0	0	0	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	5.955185	0.04874	0.17938	0.77724	0.32785	4.46638875	0.036555
42095	Pennsylvania	Northampton	Fuel Combustion: Electric... Gas	Process	LOW		<input checked="" type="checkbox"/>	54.2526302	34.19387821	0.20198838	14.20915265	17.84509687	40.68947265	25.64525865...
42095	Pennsylvania	Northampton	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	1411.6922768	0	44.1153837	94.3720841	75.82311576	1058.7692076	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	1400.7110922	0	43.77222169	93.63799369	75.23305999	1050.533319...	0
42095	Pennsylvania	Northampton	Fuel Combustion: Electric... Gas	Natural	HIGH		<input checked="" type="checkbox"/>	0	0	32.14625	0	0	0	0
42097	Pennsylvania	Northumberland	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	0.75576	0.0045024	0.03216	0.06432	0.04824	0.56682	0.0033768
42101	Pennsylvania	Philadelphia	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	4.7683632	0.8060904	0.9641568	0.5738952	1.6520592	3.57663724	0.6045678
42105	Pennsylvania	Potter	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	2.8932	0.03	0	0.3296196	0.1065	2.1699	0.0225
42111	Pennsylvania	Somerset	Fuel Combustion: Electric... Gas	Natural	LOW		<input checked="" type="checkbox"/>	2.2280448	0.0744504	0	0.5184192	0.2172408	1.6710936	0.0558378
42111	Pennsylvania	Somerset	Fuel Combustion: Electric... Gas	Process	LOW		<input checked="" type="checkbox"/>	2.4390144	1.5793776	0	0.4024824	0.2095224	1.8292808	1.1845332

### Step 3. Run the scenario.

Next, go to the **3. Execute Run** tab. This tab asks you to choose a discount rate for the COBRA session. The discount rate you select is used to express future economic values in present terms. Not all health effects and associated economic values occur in the year of analysis (as explained in Step 5 below). Therefore, COBRA accounts for this ‘time value of money’ preference (i.e., a general preference for receiving benefits now rather than later) by discounting benefits received later. Based on EPA’s Guidelines for Preparing Economic Analyses (U.S. EPA, 2010a), it is recommended that COBRA users calculate monetized health benefits using both 3% and 7% discount rates and then evaluate whether the overall outcome of the analysis is affected by the choice of discount rate. For more details on discount rates, see Appendix F.

In this scenario, you will use a 3% discount rate. Click **Run using above options**.

**NOTE:** COBRA may take 5 to 15 minutes to run, and COBRA may be “not responding” as it runs. The run time depends on your computer.

### Step 4. View and save the results.

#### View the results

Once your run is complete, COBRA will automatically navigate to the last tab, **4. View Health Effects and Valuation Results**. You will see a screen with two tabs at the top: **Table** and **Map**.



Click on the first tab, **Table**. This tab shows the changes in air quality and reductions in health effects associated with your scenario. The air quality variables shown are ‘Base PM 2.5’, ‘Control PM 2.5,’ and ‘Delta PM 2.5.’ Delta PM2.5 is the difference in ambient fine particulate matter (PM<sub>2.5</sub>) between the baseline scenario and your new scenario (the control scenario) in the year of your analysis (in this example, 2023), in units of micrograms per cubic meter (µg/m<sup>3</sup>). Note that positive values indicate a reduction in PM<sub>2.5</sub> in the control scenario.

To the right, this table also shows the reductions in health effects associated with changes in air quality and the dollar values associated with those reductions. Note that positive values indicate a decrease in impacts (that is, fewer cases of illness/premature mortality or avoided economic loss).

The default view shows the whole country, but since the majority of the air quality changes are expected to be in Pennsylvania, filter the state by clicking in the box below ‘State’ and typing ‘Pennsylvania’.

FIPS	State	County	Base PM 2.5	Control PM 2.5	Delta PM 2.5	\$ Total Health Benefits (low estimate)	\$ Total Health Benefits (high estimate)	Mortality (low estimate)	\$ Mortality (low estimate)	Mortality	
Contains: Pennsylvania			Equals:			Total: 245,785,482.65		Total: 553,903,597.86		Total: 22,4342	Total: 242,383,188.66
42001	Pennsylvania	Adams	7.701	7.662	0.0399	2,989,781.27	6,720,499.73	0.2735	2,954,981.43		
42003	Pennsylvania	Allegheny	8.465	8.44	0.025	20,544,170.44	46,297,534.64	1.8783	20,293,163.88		
42005	Pennsylvania	Armstrong	8.542	8.488	0.0536	2,919,234.59	6,586,995.94	0.2673	2,887,775.62		
42007	Pennsylvania	Beaver	7.566	7.549	0.0163	2,256,916.42	5,088,723.52	0.2066	2,232,404.28		
42009	Pennsylvania	Bedford	7.358	7.343	0.0154	574,548.95	1,297,198.66	0.0526	569,434.13		
42011	Pennsylvania	Berks	8.931	8.886	0.0446	11,254,780.31	25,355,647.45	1.0251	11,075,055.76		
42013	Pennsylvania	Blair	7.549	7.532	0.0173	1,739,592.58	3,920,842.85	0.1592	1,720,451.42		
42015	Pennsylvania	Bradford	6.703	6.67	0.0338	1,542,610.36	3,473,030.83	0.141	1,523,754.78		
42017	Pennsylvania	Bucks	8.155	8.116	0.0395	16,848,284.69	38,020,331.22	1.5402	16,640,153.2		
42019	Pennsylvania	Butler	8.144	8.135	0.0087	1,245,720.71	2,806,831.09	0.114	1,231,275.49		
42021	Pennsylvania	Cambria	7.458	7.438	0.02	2,211,530.01	4,993,319.16	0.2027	2,189,635		
42023	Pennsylvania	Cameron	6.822	6.805	0.0169	65,010.96	146,611.09	0.006	64,407.48		
42025	Pennsylvania	Carbon	7.781	7.742	0.0388	2,163,042.62	4,880,616.51	0.1982	2,141,764.04		
42027	Pennsylvania	Centre	7.27	7.254	0.016	1,030,705.48	2,316,642.27	0.0993	1,007,787.14		
42029	Pennsylvania	Chester	8.494	8.471	0.0227	6,562,779.87	14,778,712.6	0.5981	6,491,760.92		
42031	Pennsylvania	Clarion	7.831	7.812	0.0186	494,450.56	1,117,297.32	0.0452	488,696.06		
42033	Pennsylvania	Clearfield	7.327	7.3	0.0269	1,658,951.07	3,744,108.37	0.1518	1,640,242.4		
42035	Pennsylvania	Clinton	7.016	6.997	0.0194	473,027.88	1,066,979.96	0.0432	466,739.96		
42037	Pennsylvania	Columbia	7.715	7.688	0.0274	1,328,174.45	2,769,181.19	0.1122	1,212,271.79		
42039	Pennsylvania	Crawford	7.237	7.231	0.0066	433,807.8	977,849.34	0.0397	426,677.75		
42041	Pennsylvania	Cumberland	8.312	8.292	0.0204	3,493,239.97	7,562,901.93	0.3109	3,358,683.71		
42043	Pennsylvania	Dauphin	8.528	8.488	0.0407	6,438,573.31	14,496,995.6	0.5883	6,323,521.34		
42045	Pennsylvania	Delaware	8.405	8.371	0.0332	11,014,454.62	24,812,407.3	1.0015	10,820,137.6		
42047	Pennsylvania	Elk	7.007	6.997	0.0109	493,828.37	1,113,039.94	0.0453	489,103.93		
42049	Pennsylvania	Erie	6.901	6.895	0.0064	1,233,405.24	2,780,101.84	0.1127	1,217,203.13		
42051	Pennsylvania	Fayette	7.17	7.157	0.0123	1,467,885.15	3,305,604.5	0.1344	1,452,193.36		
42053	Pennsylvania	Forest	7.247	7.236	0.0112	65,180.76	147,273.67	0.006	64,542.81		
42055	Pennsylvania	Franklin	7.486	7.469	0.0173	1,742,884.69	3,935,771.38	0.1596	1,724,188.54		
42057	Pennsylvania	Fulton	7.405	7.392	0.0133	138,848.93	312,455.85	0.0137	136,956.99		
42059	Pennsylvania	Greene	5.757	5.751	0.0063	147,281.98	332,605.04	0.0135	145,661.39		

Here, you see that the emissions scenario results in an estimated 22.4 to 50.7 adult premature deaths avoided and 12.9 avoided emergency room visits for asthma. You can also look at a specific county in Pennsylvania. To do this, filter the county level by clicking in the box next to ‘County’ and typing ‘Montgomery.’

COBRA											
File Help											
Introduction 1. Select Analysis Year 2. Create Emissions Scenario 3. Execute Run 4. View Health Effects and Valuation Results											
FIPS	State	County	Base PM 2.5	Control PM 2.5	Delta PM 2.5	\$ Total Health Benefits (low estimate)	\$ Total Health Benefits (high estimate)	Mortality (low estimate)	\$ Mortality (low estimate)	Mortality	
Contains: Penn			Contains: Montgomery			Total: 13,796,409.05		Total: 31,093,047.58		Total: 1,2586	Total: 13,598,654.66
42091	Pennsylvania	Montgomery	8.453	8.425	0.028	13,796,409.05	31,093,047.58	1,2586	13,598,654.66	Total	
						Total: 13,796,409.05	Total: 31,093,047.58	Total: 1,2586	Total: 13,598,654.66	Total	

You will see that in Montgomery County, the estimated ambient  $PM_{2.5}$  concentration in the control scenario is  $8.425 \mu\text{g}/\text{m}^3$ , compared to the estimated baseline concentration of  $8.453 \mu\text{g}/\text{m}^3$ . The difference between the two estimated concentrations (Delta  $PM_{2.5}$ ) is  $0.028 \mu\text{g}/\text{m}^3$ , which is the estimated change in air quality due to the 25% reduction in emissions from fuel combustion electricity generating plants in the whole state (the change in concentration is due to decreases in emissions from plants within the county and in other counties). Note that positive changes indicate a lower concentration in the control scenario. If the value in ‘Delta  $PM_{2.5}$ ’ were negative, it would indicate an increase in concentration. In this county, the emissions reduction scenario results in an estimated 1.3 to 2.8 adult premature deaths avoided (over the next 20 years), an estimated 0.1 to 1.2 avoided non-fatal heart attacks, and an estimated 0.9 avoided emergency room visit for asthma (in 2023).

The table includes low and high estimates for the changes in the number of cases and the corresponding economic values for adult mortality and non-fatal heart attacks. The low and high estimates are derived using two sets of assumptions about the sensitivity of adult mortality and non-fatal heart attacks to changes in ambient  $PM_{2.5}$  levels. Specifically, the high estimates are based on studies that estimated a larger effect of changes in ambient  $PM_{2.5}$  levels on the incidence of these health effects. For further details on the calculation of low and high estimates, see the description of the health effects table in Chapter 5 and the detailed assumptions in Appendix C.

The three health effects below demonstrate the interpretation of the change in health effects and their economic values for Montgomery County, Pennsylvania: respiratory hospital admissions, adult mortality, and non-fatal heart attacks.

- Emergency Room Visits for Asthma.** In COBRA, most health effects and their economic values are expected to occur in the year of analysis. For instance, this scenario results in less than one avoided emergency room visits for asthma in Montgomery County. This fraction of an avoided case and its economic value (approximately \$534) would occur in 2023.
- Adult Mortality.** In contrast to respiratory hospital admissions, all avoided cases of adult mortality are not expected to occur in the year of analysis. Therefore, COBRA uses the 3% discount rate you selected in Step 4 to calculate the value of all avoided cases of adult mortality in present terms (in Montgomery County, a

low estimate of 1.3 avoided cases of adult mortality are valued at a total of approximately \$14 million).

- Non-fatal Heart Attacks.** Another special case is non-fatal heart attacks. All avoided cases of non-fatal heart attacks are expected to occur in the year of analysis, but the costs associated with this health effect would occur over multiple years. Thus, while this scenario results in a range of 0.1 to 1.2 cases of non-fatal heart attacks in 2023, all economic benefits associated with this change (\$17,620 to \$163,685) would not accrue in that same year.

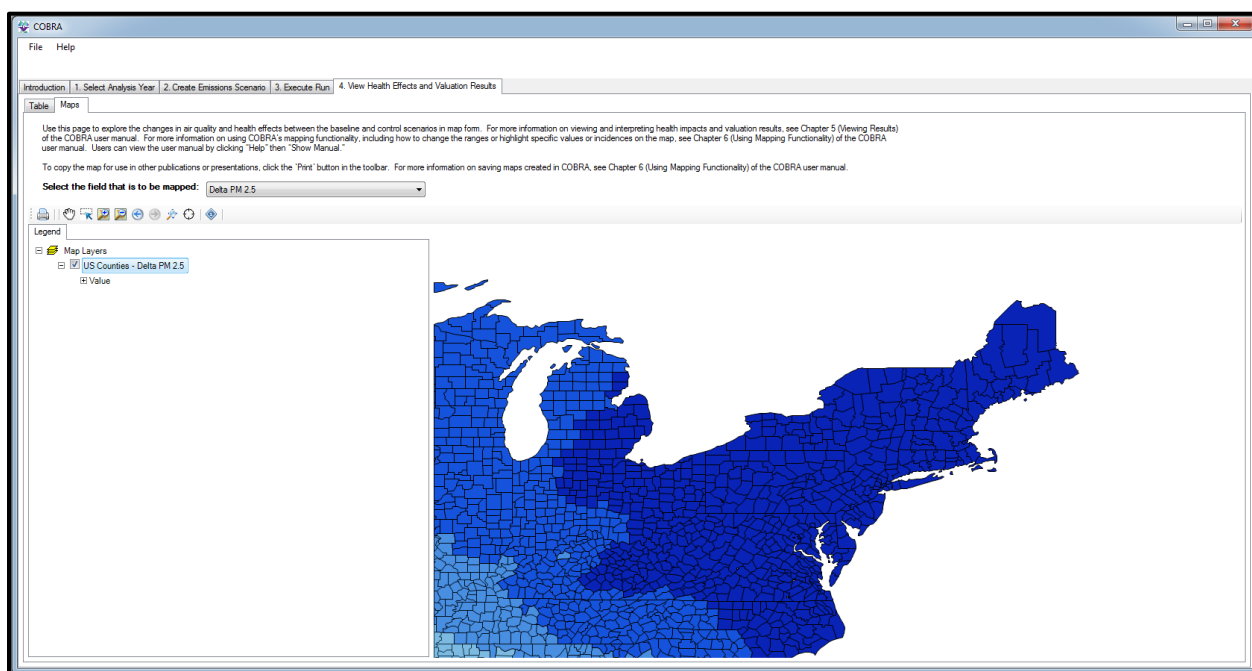
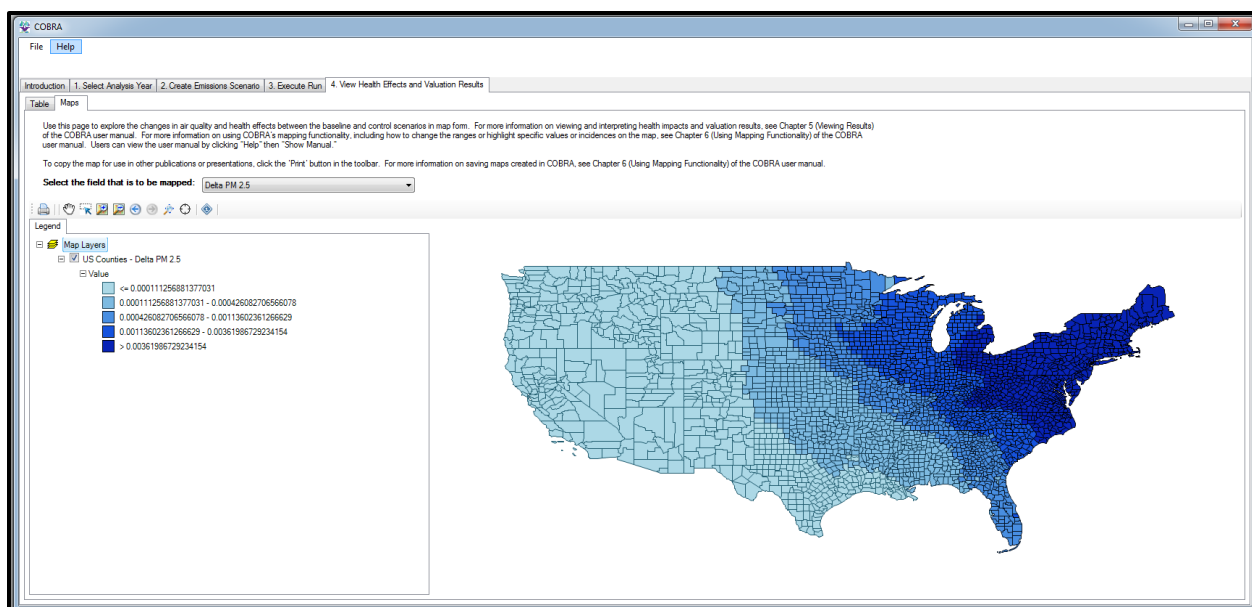
The table below relates the timing of the expected health effects and economic costs or benefits to the specific types of incidences in COBRA.

Type of Health Incidence	All health effects occur in the year of analysis?	All economic costs or benefits occur in same year of analysis?
Adult mortality	No	No, the value is discounted to present terms
Non-fatal heart attacks	Yes	No, the value is discounted to present terms
All other health impacts	Yes	Yes

In addition, remember that although emissions were changed only in Pennsylvania, COBRA calculates changes in PM<sub>2.5</sub> for the whole country. If you would like to examine the results for any of states bordering Pennsylvania, simply filter for different states.

As described earlier, it is also important to remember COBRA does not capture dynamic effects of electricity markets – these markets determine electricity dispatch by EGUs and associated emissions levels. For example, if you assume an emission reduction among power plants in one area, it is possible that electricity generation and emissions may increase in another location unless there is an underlying change in electricity demand. Because COBRA does not capture potential electricity market effects, users should exercise care when interpreting the impacts of an emissions change in a specific location.

The **Map** tab shows the results from the previous table on a map. When you click on the tab you will see a map of the United States. To display a result, select an outcome from the drop-down menu labeled ‘Select the field that is to be mapped.’ Below, the map of Delta PM<sub>2.5</sub> shows the change in the particulate matter concentration between the baseline and control scenarios. The darker the shade of blue, the greater the change in concentration. As in the other results tables, a positive number indicates a *decrease* from the baseline scenario. You can also view any of the other results on the map by selecting them from the drop-down list under ‘Select the field that is to be mapped’. See Chapter 6 for more details on how to use COBRA’s mapping functionality.



### **Export and save your results.**

You may want to look at and manipulate the results data outside of COBRA. You can export data sets into a CSV format, which can be used with spreadsheet programs.

### **Emissions Scenario Definition in Table Form**

To save the definition of your emissions changes scenario go to the **2. Create Emissions Scenario** tab. In the top right panel, click **Save Scenario**. In the following window, browse to the file location where you want to save your data, and select 'CSV files' in the

'Save as type' drop-down box. You can then enter a name for the file in the box above. Click **Save** and COBRA will save the file.

### Results in Table Form

To export the results table, go to the **4. View Health Effects and Valuation Results** tab and then to the **Table** tab. Click on the **Export to CSV** or **Export to Excel** button, depending on your preferred format. In the following window, browse to the file location where you want to save your data, enter a name for the file, and click **Save**.

### Results in Map Form

To save a map for use in another mapping application, navigate to the map and right-click on the 'US counties' data in the legend panel and then click **Data... Export Data**. In the following window, browse to the file location where you want to save your data, enter a file name, and select '.shp' in the 'Save as type:' drop-down menu. Click **Save** and COBRA will save the file.

To save the map as an image, click the 'Print' button in the toolbar. In the following window, click **File... Save**. Then browse to the file location where you want to save your data and select 'DotSpatial Layout File' or 'Portable Network Graphics' in the 'Save as type' drop-down box. You can then enter a name for the file in the box above (e.g., type 'Penn Utility Reduction 2023 - 3%'). Click **Save** and COBRA will save the file.

## CHAPTER 3. Baseline Data

Click on the **1. Select Analysis Year** tab to select an analysis year.

### Basic Options

The COBRA model contains detailed 2016, 2023, and 2028 baseline emissions data for every county in the U.S., by state, county, tier category, and pollutant type (see Appendix A for details on the baseline data). It is recommended that new COBRA users start by using the default 2016, 2023, and 2028 baseline emissions database. To use the 2023 default emissions, go to the **Basic Options** tab, select 2023 in the “Select analysis year” drop down menu, and click **Apply selected analysis year**. COBRA will automatically use the population, baseline health incidence, baseline emissions, and health impact valuation datasets that correspond to the analysis year you select.

### Advanced Options

Users can also import custom datasets for population, baseline health incidence, baseline emissions, health effect functions, and health impact valuation.

### *Custom Baseline Emissions*

There are two ways for users to import custom emissions: (1) load a file containing only baseline emissions or (2) load a file containing emissions for both the baseline and your control scenario. It is important for input files to correspond to the same year, so if you choose to import custom baseline emissions data for a year that is not preloaded, you would also want to modify population data and incidence data to represent that same year. For example, running COBRA with a 2023 baseline emissions inventory, 2023 population data, and 2023 incidence data would result in COBRA estimating 2023 health benefits. However, running COBRA with a 2020 baseline emissions inventory and 2023 population and incidence data would result in theoretical annual health benefits that do not correspond to any calendar year. Alternatively, running COBRA with a projected emissions inventory for 2030 and the default 2023 population and incidence data would assume that population and incidence are constant from 2023 to 2030.

COBRA requires that imported baseline emission files be in a specific format. To create a properly formatted custom baseline emissions file, create a CSV file with the column headings shown in Exhibit 2. Each row of the file should correspond to a different source in a different county.

**Exhibit 2. Format of Baseline Emissions File.**

<b>Column Heading</b>	<b>Description of Column Headings</b>
typeindx <sup>1</sup>	Stack height associated with the emission
sourceindx <sup>2</sup>	Source index, which COBRA uses in its source receptor model
stid	FIPS state ID (e.g., the state FIPS code for Pennsylvania is 42)
cyid	FIPS county ID (e.g., the county FIPS code for Swarthmore, PA is 045)
TIER1 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 1 level
TIER2 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 2 level
TIER3 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 3 level
NO2	NOx emissions from each source in the baseline
SO2	SO2 emissions from each source in the baseline
NH3	NH3 emissions from each source in the baseline
PM25	Primary PM2.5 emissions from each source in the baseline
VOC	VOC emissions from each source in the baseline

## Notes:

<sup>1</sup> A table of typeindx and name is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/typeindx – stack heights.csv.

<sup>2</sup> A table of sourceindx and FIPS is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/SOURCEINDEX to FIPS crosswalk.csv.

<sup>3</sup> A table of tier definitions and tier numbers is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/EmissionsTier Definitions.csv.

To see the template for a baseline emissions file, navigate to the folder where you installed COBRA – the default location is C:/Program Files/COBRA/. From there, navigate to input files/emissions/Emissions\_2023.csv. This is the default COBRA 2023 emissions baseline, so do not make any changes directly to this file.

	A	B	C	D	E	F	G	H	I	J	K	L	M
	typeindx	sourceindx	stid	cyid	TIER1	TIER2	TIER3	NO2	SO2	NH3	PM25	VOC	
1	1	1	1	1	1	2	1	3	0	0	0	0	0
2	1	1	1	1	1	2	1	99	3.057423	43.36646	0.137616	3.110278	0.002523
3	1	1	1	1	1	2	2	1	0.091093	0.044303	0.003446	0.004898	1.21E-06
4	1	1	1	1	1	2	2	2	1.574823	1.34E-05	0.021663	0.957337	5.42E-06
5	1	1	1	1	1	2	3	1	14.15869	0.283776	1.513474	0.283776	2.566108
6	1	1	1	1	1	2	4	1	19.4424	11.62828	3.25591	207.9131	7.907229
7	1	1	1	1	1	2	4	99	0.066413	0.107956	0.001851	0.003482	0.001345
8	1	1	1	1	1	3	1	99	0	0	0	0	0
9	1	1	1	1	1	3	2	99	0.975228	0.000396	0.001277	0.055387	5.73E-07
10	1	1	1	1	1	3	3	99	8.337391	0.046515	0.037987	0.033336	0.420623
11	1	1	1	1	1	3	4	99	0.62026	0.07789	0.013863	1.219766	0.046901
12	1	1	1	1	1	3	5	99	2.165133	0.28277	1.035349	18.13988	22.83828
13	1	1	1	1	1	3	6	1	0.123392	0.292028	0.006855	0.014601	0.004799
14	1	1	1	1	1	3	6	1	0.123392	0.292028	0.006855	0.014601	0.004799

To import custom baseline datasets, click on the **Advanced Options** tab at the top of the **1. Select Analysis Year** screen. To import a properly formatted baseline emissions file into COBRA, click **Load a Baseline Emissions CSV File**. In the pop-up window, navigate to the location of your baseline emissions CSV file, click the file, and then click **Open**. If you try to import a baseline emissions file that is incorrectly formatted, COBRA will display an error message that an “unhandled exception has occurred in your application.”

To create a properly formatted file with both custom baseline and control scenario emissions, create a CSV file with the headings shown in Exhibit 3. Each row of the file should correspond to a different source in a different county.

**Exhibit 3. Format of Baseline and Scenario Emissions File**

Column Heading	Description of Column Headings
typeindx <sup>1</sup>	Stack height associated with the emission
sourceindx <sup>2</sup>	Source index, which COBRA uses in its source receptor model
stid	FIPS state ID (e.g., the state FIPS code for Pennsylvania is 42)
cyid	FIPS county ID (e.g., the county FIPS code for Swarthmore, PA is 045)
TIER1 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 1 level
TIER2 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 2 level
TIER3 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 3 level



Column Heading	Description of Column Headings
BASE_NO2	NO <sub>x</sub> emissions from each source in the baseline
BASE_SO2	SO <sub>2</sub> emissions from each source in the baseline
BASE_NH3	NH <sub>3</sub> emissions from each source in the baseline
BASE_PM25	Primary PM <sub>2.5</sub> emissions from each source in the baseline
BASE_VOC	VOC emissions from each source in the baseline
CTRL_NO2	NO <sub>x</sub> emissions from each source in the control scenario
CTRL_SO2	SO <sub>2</sub> emissions from each source in the control scenario
CTRL_NH3	NH <sub>3</sub> emissions from each source in the control scenario
CTRL_PM25	Primary PM <sub>2.5</sub> emissions from each source in the control scenario
CTRL_VOC	VOC emissions from each source in the control scenario
TIER1NAME	Name of emissions category to which emissions source corresponds, at tier 1 level
TIER2NAME	Name of emissions category to which emissions source corresponds, at tier 2 level
TIER3NAME	Name of emissions category to which emissions source corresponds, at tier 3 level
FIPS	FIPS state-county ID (e.g., the state FIPS code for Pennsylvania is 42 and the county FIPS code for Swarthmore, PA is 045, so the state-county FIPS ID is 42045)
STATE	State
COUNTY	County
TYPE	Type denotes the stack height associated with the emission

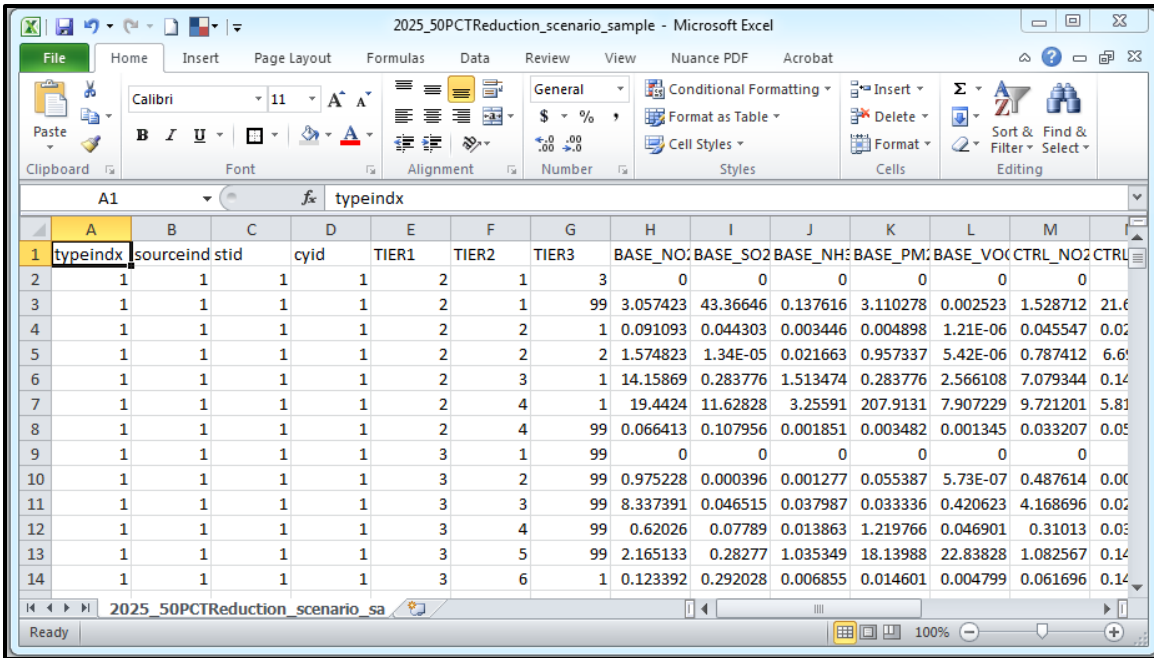
## Notes:

<sup>1</sup> A table of typeindx, type, and name is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/typeindx – stack heights.csv.

<sup>2</sup> A table of sourceindx and FIPS is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/SOURCEINDEX to FIPS crosswalk.csv

<sup>3</sup> A table of tier definitions and tier numbers is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/EmissionsTier Definitions.csv

To see an example baseline and control scenario emissions file, navigate to your Documents folder in Windows. From there, navigate to input files/emissionscenarios/2023\_50PCTReduction\_scenario\_sample.csv.



To import a properly formatted baseline and control emissions CSV file into COBRA, click **Load a Baseline Control Scenario Emissions CSV File**. In the pop-up window, navigate to the location of your baseline emissions CSV file, click the file, and then click **Open**.

### Custom Population, Incidence, and Valuation Datasets

The **Advanced Options** tab allows users to select customized input files for population data, incidence data, and valuation functions. Note that baseline emissions, population, incidence, and valuation functions vary over time. It is important for input files to correspond to the same year. For example, running COBRA with a 2023 baseline emissions inventory, 2023 population data, and 2023 incidence data would result in COBRA estimating 2023 health benefits. However, running COBRA with a 2023 baseline emissions inventory and 2020 population data would result in theoretical annual health benefits that do not correspond to any calendar year. Alternatively, running COBRA with a projected emissions inventory for 2030 and the default 2023 population and incidence data would assume that population and incidence are constant from 2023 to 2030.

COBRA requires that customized input files be in a specific format. To create a properly formatted population dataset, create a CSV file where each row is a different county and includes the columns listed in Exhibit 4.

**Exhibit 4. Format of Population File**

Column Heading	Description of Column Headings
Year	Year of population data

Column Heading	Description of Column Headings
DestinationID <sup>1</sup>	DestinationID uses same pattern as SOURCEINDX to indicate destination locations.
FIPS	FIPS state-county ID (e.g., the state FIPS code for Pennsylvania is 42 and the county FIPS code for Swarthmore, PA is 045, so the state-county FIPS ID is 42045)
Age0	Population at age 0
...	Population at each age
Age99	Population older than or equal to 99

<sup>1</sup> See data dictionary file that is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/SOURCEINDX to FIPS crosswalk.csv

To create a properly formatted incidence dataset, create a CSV file where each row is a different county and includes the columns listed in Exhibit 5.

**Exhibit 5. Format of Incidence File**

Column Heading	Description of Column Headings
Year	Year of population data
DestinationID <sup>1</sup>	DestinationID uses same pattern as SOURCEINDX to indicate destination locations.
FIPS	FIPS state ID (e.g., the state FIPS code for Pennsylvania is 42 and the county FIPS code for Swarthmore, PA is 045, so the state-county FIPS ID is 42045)
Endpoint	Written descriptor indicating health endpoint associated with the recorded incidence rate. (e.g., Acute Bronchitis)
Age0	Incidence of a specific endpoint for population at age 0
...	Incidence of a specific endpoint for population at each age
Age99	Incidence of a specific endpoint for population older than or equal to 99

<sup>1</sup> See data dictionary file that is saved on your computer after installing COBRA in the default location, in C:/Program Files/COBRA/input files/data dictionary/SOURCEINDX to FIPS crosswalk.csv

To create a properly formatted valuation functions dataset, create a CSV file where each row represents a concentration-response function from a single study for a specific age group and includes the columns described in Exhibit 6.

**Exhibit 6. Format of Valuation Functions File.**

Column Heading	Description of Column Headings
CRFunctionID	ID for C-R functions (linked to the health effect function file)
Endpoint	String descriptor indicating Endpoint for the Valuation function.

Column Heading	Description of Column Headings
PoolingWeight	Functions are pooled accounting to Endpoint. PoolingWeight indicates the relative weight given to this function.
Seasonal_Metric	Daily or Annual are allowable entries. Indicate if computed effect is for a day or an entire year.
Study_Author	Study Author
Study_Year	Study Year
Start_Age	Beginning of age range to which health impact function applies
End_Age	End of age range to which health impact function applies
Function	Functional form that is to be computed. (e.g., $(1 - 1 / ((1 - \text{Incidence}) * \text{Exp}(\text{Beta} * \text{DELTAQ}) + \text{Incidence}))) * \text{Incidence} * \text{POP}$ )
Beta	Beta parameter supplied to functional form.
Adjusted	Not used
Parameter_1_Beta	Parameter_1_Beta parameter supplied to functional form.
A	A parameter supplied to functional form.
Name_A	Description of A parameter.
B	B parameter supplied to functional form.
Name_B	Description of B parameter.
C	C parameter supplied to functional form.
Name_C	Description of C parameter.
Cases	Not used
HealthEffect	Use same as IncidenceEndpoint
ValuationMethod	Note of valuation method used.
valat3pct	Valuation of health endpoint using 3% discount rate
valat7pct	Valuation of health endpoint using 7% discount rate
IncidenceEndpoint	Indicates Endpoint to use for lookup in Incidence data set.

### Custom Health Effect Function Datasets

The **Advanced Options** tab allows users to select customized health effect function datasets. The default health dataset in COBRA relies on an up-to-date assessment of the published scientific literature to ascertain the relationship between particulate matter and adverse human health effects, and can be used for all analysis years. Because of this, most users will not want to modify this input. If you use custom datasets to describe health effect functions, please list the sources of these datasets and any assumptions used to generate them when reporting your results.

If you do choose to import a custom health effect function dataset, COBRA requires that this file be in a specific format. To create a properly formatted health effect functions dataset, create a CSV file where each row represents a concentration-response function from a single study for a specific age group and includes the columns listed in Exhibit 7.

**Exhibit 7. Format of Health Effect Functions File.**

Column Heading	Description of Column Headings
FunctionID	ID for C-R functions (linked to the valuation function file)
Endpoint	String descriptor indicating endpoint for the Health/Concentration-Response function.
PoolingWeight	Functions are pooled accounting to Endpoint. PoolingWeight indicates the relative weight given to this function.
Seasonal_Metric	Daily or Annual are allowed entries. Indicate if computed effect is for a day or an entire year.
Study_Author	Study Author
Study_Year	Study Year
Start_Age	Beginning of age range to which health impact function applies
End_Age	End of age range to which health impact function applies
Function	Functional form that is to be computed. (e.g., $(1 - (1 - \text{Incidence}) * \text{Exp}(\text{Beta} * \text{DELTAQ}) + \text{Incidence})) * \text{Incidence} * \text{POP}$ )
Beta	Beta parameter supplied to functional form.
Adjusted	Not used
Parameter_1_Beta	Parameter_1_Beta parameter supplied to functional form.
A	A parameter supplied to functional form.
Name_A	Description of A parameter.
B	B parameter supplied to functional form.
Name_B	Description of B parameter.
C	C parameter supplied to functional form.
Name_C	Description of C parameter.
Cases	Not used
IncidenceEndpoint	Indicates Endpoint to use for lookup in Incidence data set.

## Exploring Baseline Emissions Data

You can explore the geographic distribution of pollutant concentrations in the baseline scenario by clicking on the **2. Create Emissions Scenario** tab and then clicking on the **View Emissions Map** tab.

At the top of this screen, you will see a drop-down menu labeled ‘Select the field that is to be mapped.’ The first five options listed (Base NH<sub>3</sub>, Base NO<sub>2</sub>, Base PM<sub>2.5</sub>, Base SO<sub>2</sub>, and Base VOC) correspond to the baseline emissions of ammonia (NH<sub>3</sub>), nitrogen oxides (NO<sub>x</sub>), fine particulate matter (PM<sub>2.5</sub>), sulfur dioxide (SO<sub>2</sub>), and Volatile Organic Compounds (VOCs), respectively, from all sectors. Select any of these emissions to view a map of county-level baseline emissions. See Chapter 6 for more details on how to use COBRA’s mapping functionality.

## CHAPTER 4. Creating a New Emissions Scenario

COBRA allows users to define new emissions scenarios and investigate the related changes in air quality and health effects. The scenario definition, results tables, and results maps can be exported for future use or archiving purposes.

The steps to creating a new scenario within the COBRA interface are simple:

**Step 1. Select the Analysis Year and Baseline Data.** See Chapter 3.

**Step 2a. Select the Geography and Tier Level.** You can specify emissions changes at the national, state, or county level from fourteen emission source categories.

**Step 2b. Enter Emissions Changes.** Changes can be made for each state or county, or the entire nation. You can group states and counties and make changes to them together, or make different changes to different geographic areas.

**Step 3. Run the Scenario.** COBRA will calculate the changes in ambient PM<sub>2.5</sub> between your selected baseline scenario and the new control scenario, and calculate the associated changes in health effects and monetary impacts.

**Step 4. Examine the results.** See Chapter 5.

### Selecting Scenario Geography

To define your scenario, click the **2. Create Emissions Scenario** tab and then the **Emissions Scenario** tab. In the top left panel you can select the geographic area(s) to which you want to apply emissions changes. Note that once your comparison is run, you will be able to see results (changes in ambient PM<sub>2.5</sub> levels and health effects) for the entire country. Click on the appropriate choice in the left panel.

- **US.** If you wish to make emissions changes to the entire United States, select 'US.'
- **Individual State.** If you wish to make emissions changes to just one state, click that state.
- **Individual County.** If you wish to make emissions changes to just one county, click that county.

The geographic areas you select determine where your emissions changes are made. Your selection does not affect the geographic area for which you can view results. Regardless of the geographic area you select for emissions changes, you can view results for the entire country.

- **Multiple Geographic Areas.** If you wish to make the same emissions changes to multiple geographic areas, click the check box next to each of those geographic areas in the ‘Tier and Location Selection’ panel. It is possible to select multiple states (e.g., all of California and Oregon), multiple counties (e.g., Dade County, Florida and Broward County, Florida), and combinations of states and counties (e.g., all of Massachusetts and Providence County, Rhode Island).

The ‘Pollutants’ panel in the top center of your screen will show the total baseline emissions for all the selected geographic areas.

When you apply an emissions change to multiple geographic areas as a percentage, your control scenario will consist of emissions in each county in the selected areas changing by that same percentage. When you apply an emissions change to multiple geographic areas in tons, your control scenario will consist of emissions in each county in the selected areas changing by different amounts that add up to the input total emissions change. COBRA divides the total emissions change across counties in proportion to the baseline emissions. For example, using the 2023 analysis year, if you input a 100 ton reduction in SO<sub>2</sub> emissions in the Fuel Combustion: Industrial tier in Florida, this would correspond to SO<sub>2</sub> emissions decreases in Alachua County in the Fuel Combustion: Industrial tier by 0.052 tons, emissions in Baker County would decrease by 0.02 tons, etc., so that the sum of the SO<sub>2</sub> emission reductions across all Florida counties is 50 tons.

If you have decided to apply different changes to different geographic areas categories, you will need to select the geographic area, select the tier level, and define the scenario emissions separately for each geographic area.

### Selecting Tier Level

Next, define the categories of emissions affected. The center panel contains a directory tree with all the tier 1, 2, and 3 source categories (see Appendix A for a list of source categories and their emissions). You can define emissions changes at any level, but each level always includes all the levels indented underneath it. Click the tier at which you would like to apply your emissions changes. A change entered for a tier category applies to all of the branches under it, but you must enter changes individually for tier categories on separate branches.

### Defining Scenario Emissions

After you have selected your tier and geographic area, you can create a new emissions scenario by defining changes to the baseline emissions scenario. This can either be done manually or by uploading an AVERT emissions change data file if you have EPA AVERT model outputs you would like to use.



### ***Manually Entering Emissions Changes***

To manually enter the changes to the baseline emissions, use the boxes in the right panel for each of the five pollutants potentially emitted by sources in that category. You can enter emissions increases or decreases by percent or by tons. To the right of the boxes you will see the total emissions for your selected tier and geographic area for the baseline and control scenarios. If your emissions scenario attempts to reduce emissions by more than exists in the baseline emissions inventory (e.g., if the SO<sub>2</sub> emissions baseline for Fuel Combustion: Electric Utilities in California is 4,065 tons and you try to reduce those emissions by 4,100 tons), you will get the error message “Please make sure you do not try to reduce emissions by more than actually present in the baseline.” Click **Apply Changes** to save your changes.

Once your emissions changes are entered for a given tier and location, you can go back and change them by selecting that tier and geographic location again in the directory trees. The emissions baseline and your previously saved control scenario changes will be displayed in the right panel. To overwrite your previously saved control scenario for that tier and location, enter your new emissions changes and click **Apply Changes**.

If you have decided to apply different changes to different geographic areas categories, click the **Clear Selected States or Counties** button and repeat the above steps for each geographic area. For example, if you want to decrease all California Off-Highway SO<sub>2</sub> emissions by 25% and all Oregon Off-Highway SO<sub>2</sub> emissions by 15%, you would have to enter the California emissions change, click **Apply Changes**, click **Clear Selected States or Counties**, enter the Oregon emissions change, and click **Apply Changes** again.

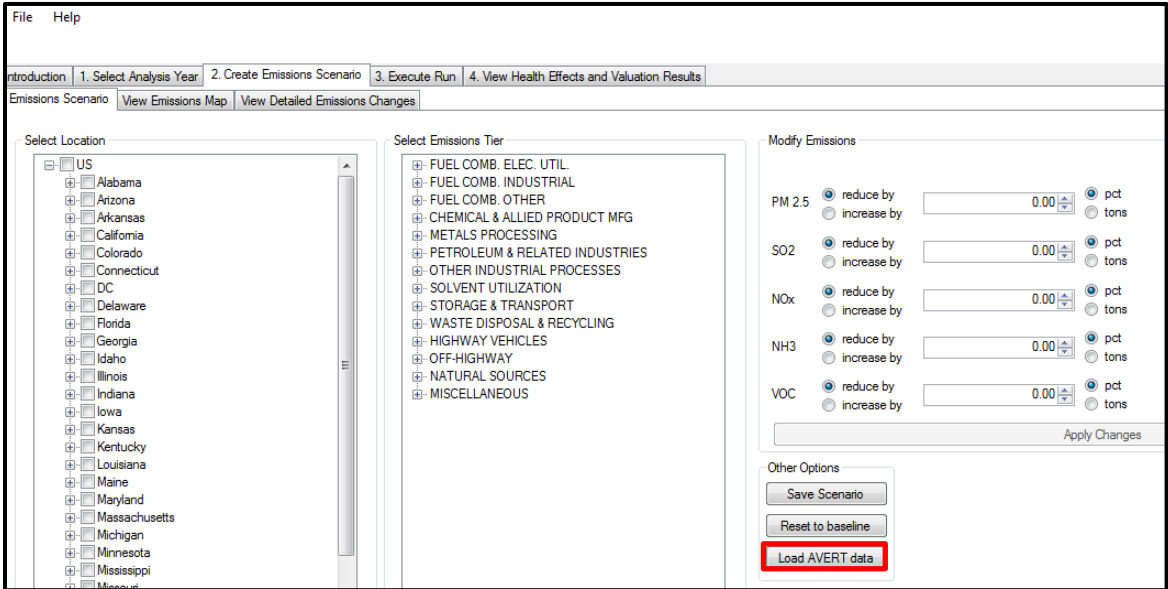
### ***Importing Emissions Changes from AVERT***

EPA’s AVOIDED Emissions and geneRation Tool ([AVERT](https://www.epa.gov/statelocalenergy/avoided-emissions-and-generation-tool-avert)) estimates displaced emissions (at the county, state, and regional levels) at electric power plants due to renewable energy or energy efficiency projects, policies, or programs. After running a renewable energy or energy efficiency scenario in AVERT, users can use the AVERT tool to generate a text file for input to COBRA that summarizes the emissions changes that result from that scenario. To access AVERT or to see AVERT output file generation instructions, available in EPA’s AVERT user manual, go to <https://www.epa.gov/statelocalenergy/avoided-emissions-and-generation-tool-avert>.

Once the AVERT output file is generated, it can be imported directly into COBRA by clicking the “**Load AVERT output file**” button in the **2. Create Emissions Scenario** tab in COBRA. The emissions reductions from each county from AVERT will be loaded into COBRA in the “Fuel Combustion: Electric Utilities” tier.

If you have entered any emissions changes in the user interface before loading an AVERT emissions scenario file, loading the AVERT file will make COBRA overwrite

emissions changes in the Fuel Combustion: Electric Utilities tier for the counties included in the AVERT file. Other tiers and counties will be unaffected.



The text file that AVERT generates is already formatted to directly import into COBRA without any modifications. If you choose to edit the AVERT output file, please make sure to maintain the original column headings and file format (example file shown below), such that each row summarizes the emissions reductions in tons for a given county and tier 1 category.

	A	B	C	D	E	F	G	H	I
	FIPS	STATE	COUNTY	TIER1NAME	NOX_REDUCTIONS_TONS	SO2_REDUCTIONS_TONS	PM25_REDUCTIONS_TONS		
2	6001	California	Alameda County	FUEL COMB. ELEC. UTIL.	-0.545	-0.03	-0.19		
3	6011	California	Colusa County	FUEL COMB. ELEC. UTIL.	-0.34	-0.065	-0.325		
4	6013	California	Contra Costa County	FUEL COMB. ELEC. UTIL.	-13.435	-0.21	-0.765		
5	6019	California	Fresno County	FUEL COMB. ELEC. UTIL.	-0.625	-0.025	-0.185		
6	6029	California	Kern County	FUEL COMB. ELEC. UTIL.	-3.01	-0.325	-0.985		
7	6031	California	Kings County	FUEL COMB. ELEC. UTIL.	-0.1	0	-0.005		
8	6037	California	Los Angeles County	FUEL COMB. ELEC. UTIL.	-27.755	-0.365	-1.87		
9	6053	California	Monterey County	FUEL COMB. ELEC. UTIL.	-1.355	-0.075	-0.8		
10	6059	California	Orange County	FUEL COMB. ELEC. UTIL.	-6.665	-0.03	-0.355		
11	6061	California	Placer County	FUEL COMB. ELEC. UTIL.	-0.115	0	-0.025		
12	6065	California	Riverside County	FUEL COMB. ELEC. UTIL.	-1.485	-0.06	-0.58		
13	6067	California	Sacramento County	FUEL COMB. ELEC. UTIL.	-0.62	-0.06	-0.31		
14	6071	California	San Bernardino County	FUEL COMB. ELEC. UTIL.	-2.5	-0.235	-3.615		
15	6073	California	San Diego County	FUEL COMB. ELEC. UTIL.	-2.56	-0.175	-1.43		
16	6077	California	San Joaquin County	FUEL COMB. ELEC. UTIL.	-0.145	-0.005	-0.13		
17	6085	California	Santa Clara County	FUEL COMB. ELEC. UTIL.	-1.455	-0.08	-0.42		
18	6088	California	Santa Clara County	FUEL COMB. ELEC. UTIL.	0.055	0	0.015		

Note that you can paste the results from multiple AVERT runs into a single text file and import it into COBRA. If there are any duplicate counties in the text file, COBRA will sum the emissions reductions before importing the file.

After loading the AVERT emissions scenario, it is possible to manually input additional emissions changes in other counties or tiers. In other words, you can still adjust other tiers besides the “Fuel Combustion: Electric Utilities” tier, such as the “Highway Vehicles” tier. You can also enter emissions changes in the “Fuel Combustion: Electric Utilities” tier for counties not included in the AVERT output file. However, if you try to manually adjust the emissions changes in the “Fuel Combustion: Electric Utilities” tier for a county included in the AVERT output file, this will overwrite the emissions changes from the AVERT output.

In rare cases output from AVERT may call for more emission reduction in a given county than what is in the baseline in COBRA for that county in the “Fuel Combustion: Electric Utilities” tier. In these cases COBRA sets the “Fuel Combustion: Electric Utilities” emissions to 0 in that county.

It is also possible to enter a custom scenario definition that is not generated by AVERT into COBRA as a CSV. See the Advanced Options section of Chapter 3 for further instructions on this scenario definition method.

### Reviewing Scenario Definition

You can review your scenario definition by looking at the table in the **View Detailed Emissions Changes** tab. This will display all of your changes by state, county, and tier category. If the scenario is acceptable and you wish to save it for future reference, click on the **Emissions Scenario** tab and then click the **Save Scenario** button in the top right panel of the screen. This will export a comma-delimited file that contains the same information as shown in the **View Detailed Emissions Changes** table. Exporting this file is useful if you export any other tables from the results tabs; at a later date you will have a reference for what the results tables and maps are based on.

### Running Scenario

When you have made all of your desired changes, navigate to the **3. Execute Run** tab. This tab asks you to choose a discount rate for the COBRA session. The discount rate you select is used to express future economic values in present terms. Not all health effects and associated economic values occur in the year of analysis. Therefore, COBRA accounts for the ‘time value of money’ preference (i.e., a general preference for receiving economic benefits now rather than later) by discounting benefits received later. Based on EPA’s Guidelines for Preparing Economic Analyses (U.S. EPA, 2010a), it is recommended that COBRA users calculate monetized health benefits using both 3 and 7 percent discount rates and then evaluate whether the overall outcome of the analysis is

affected by the choice of discount rate. For more details on discount rates, see Appendix F.

After selecting a discount rate, click **Run using above options**.

The time to generate your results will vary, depending on the speed of your computer. COBRA may stop responding while it runs, and it may take 5 to 15 minutes to generate results. Once the results are generated, COBRA will navigate to the **4. View Health Effects and Valuation Results** tab, where you can view the results of your run (see Chapter 5).

## CHAPTER 5. Viewing Results

### Viewing Results in Table Form

Once you have defined your new scenario (called the control scenario) and run the comparison between the baseline and your scenario, you can view the results: changes in air quality and health effects between the baseline and control scenarios. You can view results in table form in the **Table** tab or in map form in the **Map** tab.

The **Table** tab describes the changes in air quality (i.e., particulate matter concentration) between the baseline emissions scenario and your scenario (the control scenario), and the corresponding changes in health effects (incidence and monetized values). Note that if you have run a state-specific scenario, changes in air quality for other states will typically decrease as the distance from the state increases, since the emissions changes were only made there. You can navigate through the table data in several ways:

- **Scroll** through the data using the scroll bar on the right and bottom of the window.
- **Change the sort order** by clicking on the heading of any column. Click once to sort from smallest to largest and click twice to sort from largest to smallest.
- **Filter column variables** by clicking on the filter button at the top of any column. You can use this functionality to filter down to a specific state and county.
- **Change the column order** by clicking on the column name and dragging it to a new position. When you see a black line, you can drop the column there. Note that the sort order of the table will not change.
- **Change the width of a column** by moving your mouse to the column header and pointing to the dividing line between two columns. The mouse cursor will change to two arrows, indicating that you can drag the column line to condense or expand the column.
- **View totals** by navigating to the top or bottom of the table. If you filter the table, the totals row will adjust in real time to display the totals for all values that are currently visible. For example, if you have not filtered the table, the totals row will show total health effects for the entire United States. If you have filtered the table to only show results for a specific state, the totals row will show total health effects for that state.

For each county, the table lists the annual average PM<sub>2.5</sub> concentration for the control scenario and the baseline scenario, as well as the change between the two scenarios (Delta PM<sub>2.5</sub>) in units of micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ). The table also displays the

change in the number of cases for each health effect between the baseline emissions scenario and your scenario. These changes are derived using the health impact functions described in Appendix C. The table also displays an estimate of the economic value of the change in the number of cases for each health effect. For more information, see Appendix F. Exhibit 8 describes the health endpoints and valuations that are included in the health effects tables in COBRA.

**Exhibit 8. Description of Health Effects and their Economic Values**

<b>Health Effect</b>	<b>Description</b>
<b>Mortality (low estimate)</b>	Low estimate of the number of deaths, based on Krewski et al. (2009)
<b>\$ Mortality (low estimate)</b>	Low estimate of the economic value of the number of deaths, using Krewski et al. (2009) and a discount rate of 3% or 7%
<b>Mortality (high estimate)</b>	High estimate of the number of deaths, based on Lepeule et al. (2012)
<b>\$ Mortality (high estimate)</b>	High estimate of the economic value of the number of deaths, using Lepeule et al. (2012) and a discount rate of 3% or 7%
<b>Infant Mortality</b>	Number of infant deaths
<b>\$ Infant Mortality</b>	Economic value of the number of infant deaths
<b>Nonfatal Heart Attacks (low estimate)</b>	Low estimate of the number of non-fatal heart attacks, based on four acute myocardial infarction (AMI) studies
<b>\$ Nonfatal Heart Attacks (low estimate)</b>	Low estimate of the economic value of non-fatal heart attacks, based on four AMI studies and a discount rate of 3% or 7%
<b>Nonfatal Heart Attacks (high estimate)</b>	High estimate of the number of non-fatal heart attacks, based on Peter et al. (2001)
<b>\$ Nonfatal Heart Attacks (high estimate)</b>	High estimate of the economic value of non-fatal heart attacks, using Peter et al. (2001) and a discount rate of 3% or 7%
<b>Hospital Admits, All Respiratory</b>	Number of respiratory-related hospitalizations
<b>Hospital Admits, Asthma</b>	Number of asthma-related hospitalizations
<b>Hospital Admits, Chronic Lung Disease</b>	Number of hospitalizations related to chronic lung disease
<b>\$ Hospital Admits, All Respiratory</b>	Economic value of respiratory-related hospitalizations (total across respiratory-related, asthma-related, and chronic lung disease hospitalizations)
<b>Hospital Admits, Cardiovascular (except heart attacks)</b>	Number of cardiovascular-related hospitalizations (ICD codes 390-409, 411-429); ICD code 410 (nonfatal heart attacks) is counted only in 'Non-fatal Heart Attacks'

Health Effect	Description
<b>\$ Hospital Admits, Cardiovascular</b>	Economic value of cardiovascular-related hospitalizations
<b>Acute Bronchitis</b>	Cases of acute bronchitis
<b>\$ Acute Bronchitis</b>	Economic value of acute bronchitis cases
<b>Upper Respiratory Symptoms</b>	Episodes of upper respiratory symptoms (runny or stuffy nose; wet cough; and burning, aching, or red eyes)
<b>\$ Upper Respiratory Symptoms</b>	Economic value of episodes of upper respiratory symptoms
<b>Lower Respiratory Symptoms</b>	Episodes of lower respiratory symptoms: cough, chest pain, phlegm, or wheeze
<b>\$ Lower Respiratory Symptoms</b>	Economic value of episodes of lower respiratory symptoms
<b>Emergency Room Visits, Asthma</b>	Number of asthma-related emergency room visits
<b>\$ Emergency Room Visits, Asthma</b>	Economic value of asthma-related emergency room visits
<b>Minor Restricted Activity Days</b>	Number of minor restricted activity days (days on which activity is reduced, but not severely restricted – e.g., missing work or being confined to bed is too severe to be MRAD).
<b>\$ Minor Restricted Activity Days</b>	Economic value of minor restricted activity days
<b>Work Loss Days</b>	Number of work days lost due to illness
<b>\$ Work Loss Days</b>	Economic value of work days lost due to illness

*Notes:* \* For adult mortality and nonfatal heart attacks, COBRA contains multiple health impact functions that relate PM<sub>2.5</sub> and each health effect. Therefore, there are high and low estimates of the cases avoided and their economic values for each of these health effects. More details on the underlying health impact functions are available in Appendix C of the user manual. In addition, future costs are calculated using a discount rate (3% or 7%) that you selected before running the scenario.

The health effects table includes low and high estimates for the changes in the number of cases and the corresponding economic values for adult mortality and non-fatal heart attacks. The low and high estimates are derived using two sets of assumptions about the sensitivity of adult mortality and non-fatal heart attacks to changes in ambient PM<sub>2.5</sub> levels. Specifically, the high estimates are based on studies that estimated a larger effect of changes in ambient PM<sub>2.5</sub> levels on the incidence of these health effects. The low and high estimates for each of these values are derived as follows:

- **Adult Mortality.** EPA (2009) used two studies when analyzing proposed NO<sub>2</sub> National Ambient Air Quality Standards; EPA presented the results separately for

each study. Following EPA, COBRA reports the results of two health impact functions that relate PM<sub>2.5</sub> and mortality: Krewski et al. (2009) and Lepeule et al. (2012). In the health effects table, **Adult Mortality (Low)** and **\$ Adult Mortality (Low)** represent estimates of adult deaths avoided and their economic value, respectively, based on Krewski et al. (2009). **Adult Mortality (High)** and **\$ Adult Mortality (High)** represent estimates of adult deaths avoided and their economic value, respectively, based on Lepeule et al. (2012). More details on these two studies are available in Appendix C of the user manual.

- **Nonfatal Heart Attacks.** COBRA calculates two estimates of the non-fatal heart attack cases avoided (**Nonfatal Heart Attacks**) and their economic value (**\$ Nonfatal Heart Attacks**). The low estimate is based on Peter et al. (2001), while the high estimate is based on pooling of the effect estimates of the following four studies: Sullivan et al. (2005), Pope et al. (2006), Zanobetti et al. (2009), and Zanobetti & Schwartz (2006). More details on the studies are available in Appendix C of the user manual.

The value in each health effects column represents the total change in the number of cases of each health endpoint in a county. A value of 3.00 in the Adult Mortality (low) column, for instance, indicates that in your scenario there would be an estimated 3 fewer cases of premature mortality compared to the baseline emissions scenario over the following 20 years. *Note, however, that a negative number signifies an increase in cases.* Therefore, - 3.00 in the Adult Mortality (low) column indicates that in your scenario there would be 3 additional cases of premature mortality over the following 20 years compared to the baseline emissions scenario.

*Interpreting positive and negative results:*

In the health effects table, positive numbers indicate reductions in the number of cases of adverse health effects and the associated monetary benefits of your scenario. Negative numbers signify increases in the number of cases of health effects and the resulting costs.

All health effects are monetized. However, to prevent double-counting, the calculation of asthma exacerbations only includes asthma effects occurring in children aged 6 to 18 years. This approach follows the recommendations of EPA's Science Advisory Board Health Effects Subcommittee (SAB-HES) for valuing asthma exacerbations, as described in the benefits analysis for the 2006 Regulatory Impact Analysis for the revised PM<sub>2.5</sub> National Ambient Air Quality Standard (U.S. EPA, 2006). Studies of the general population include asthmatics, so estimates based solely on the adult asthmatic population cannot be directly added to the general population numbers without double-counting. Instead, asthma exacerbations occurring in adults were assumed to be accounted for in health effects for the general population, such as WLDs and MRADs



(U.S. EPA, 2006). Since the health effects for the general population do not include asthma effects in children, the analysis of asthma exacerbations for children does not lead to double-counting (see Appendix C for details).

### Viewing Results in Maps Form

The **Map** tab displays the results of your scenario geographically. The left ‘Select the field that is to be mapped:’ drop-down menu allows you to change the values shown on the map. You can display the change in PM<sub>2.5</sub> between your scenario and the baseline emissions (the same values shown as ‘Delta PM<sub>2.5</sub>’ in the **Table** tab), or any of the health endpoints included in the model. The values displayed for each health endpoint are the change in the number of cases (or deaths for ‘Adult Mortality (Low)’, ‘Adult Mortality (High)’, and ‘Infant Mortality’) and the economic valuation of these cases from the scenario, as displayed in the **Table** tab.

### Saving Results

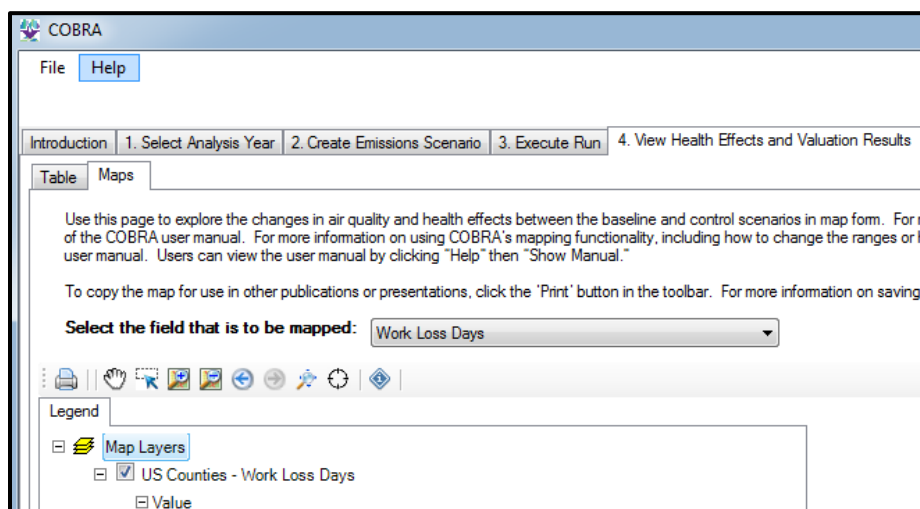
To save your results for use outside of the COBRA environment, you can export the results tables by clicking **Export to CSV** in the **Table** tab. The maps can also be customized within COBRA and then exported for use in documents and presentations (see Chapter 6).

## CHAPTER 6. Using Mapping Functionality.

COBRA can create maps of baseline emissions and air quality, control scenario emissions and air quality, changes in air quality, adverse health effects avoided, and the economic value of adverse health effects avoided. To view maps of baseline or control emissions, navigate to the **2. Create Emissions Scenario** tab and click **View Emissions Map** at the top of the page. To view maps of baseline and control air quality, changes in air quality, adverse health effects avoided, and the economic value of adverse health avoided, navigate to the **4. View Health Effects and Valuation Results** tab and click on **Map** tab at the top of the page. Maps of emissions can be viewed before running COBRA, but maps of air quality and health effects can only be viewed after running COBRA.

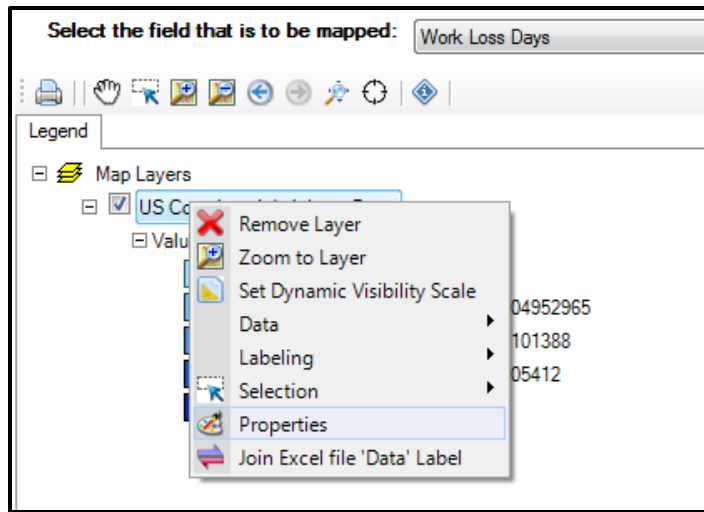
The mapping interface works the same way in both screens. Below, we describe the major capabilities of the mapping tool and show how to use it for the Pennsylvania example described in Chapter 2.

To create a map, select the variable you would like to map in the ‘Select the field that is to be mapped’ drop-down menu. For this example, you will examine avoided work loss days.

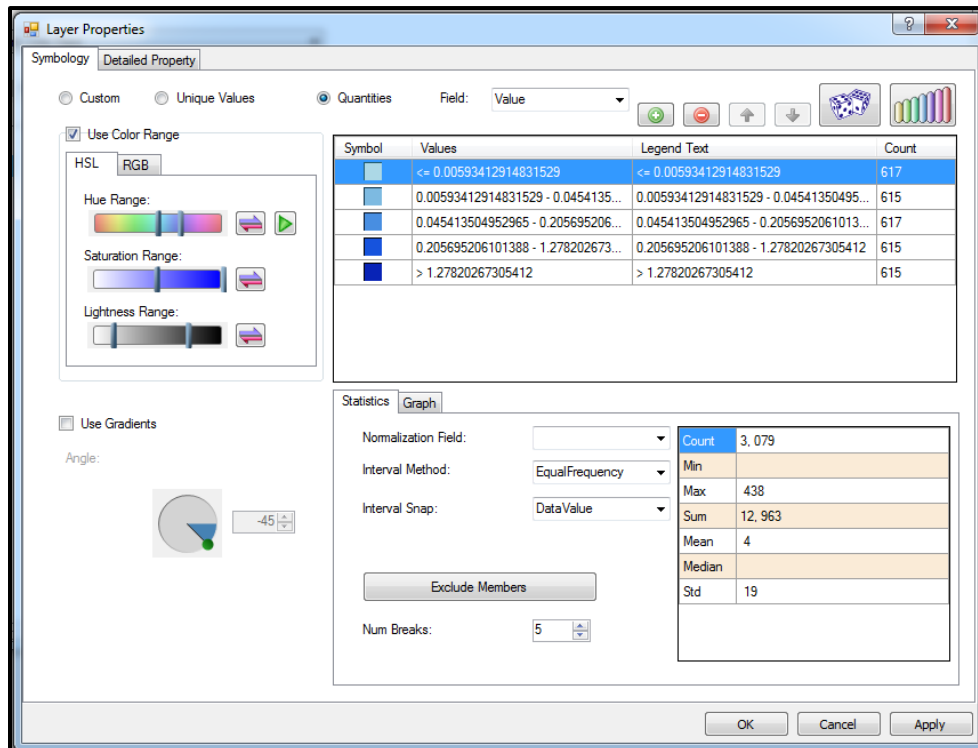


### Customizing Map Appearance

To modify the color scheme of the map, right-click ‘US Counties’ in the Legend panel and then click ‘Properties.’

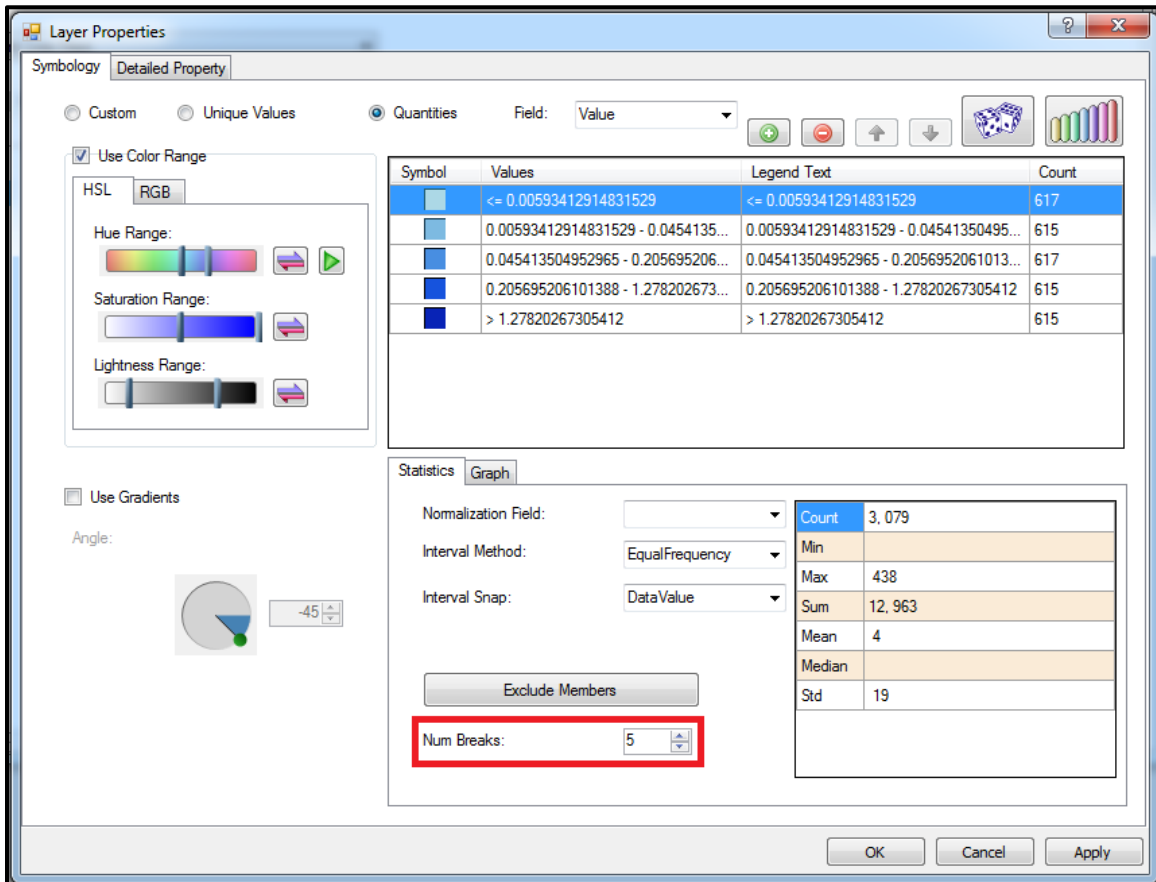


The menu that pops up has three panels. The top left panel allows you to select the overall color scheme that will define the map. The top right panel summarizes the color classification scheme (in this example, the shading that indicates larger numbers of avoided work loss days). The bottom right panel allows you to modify the ranges (or “interval breaks”) used in the color classification scheme.



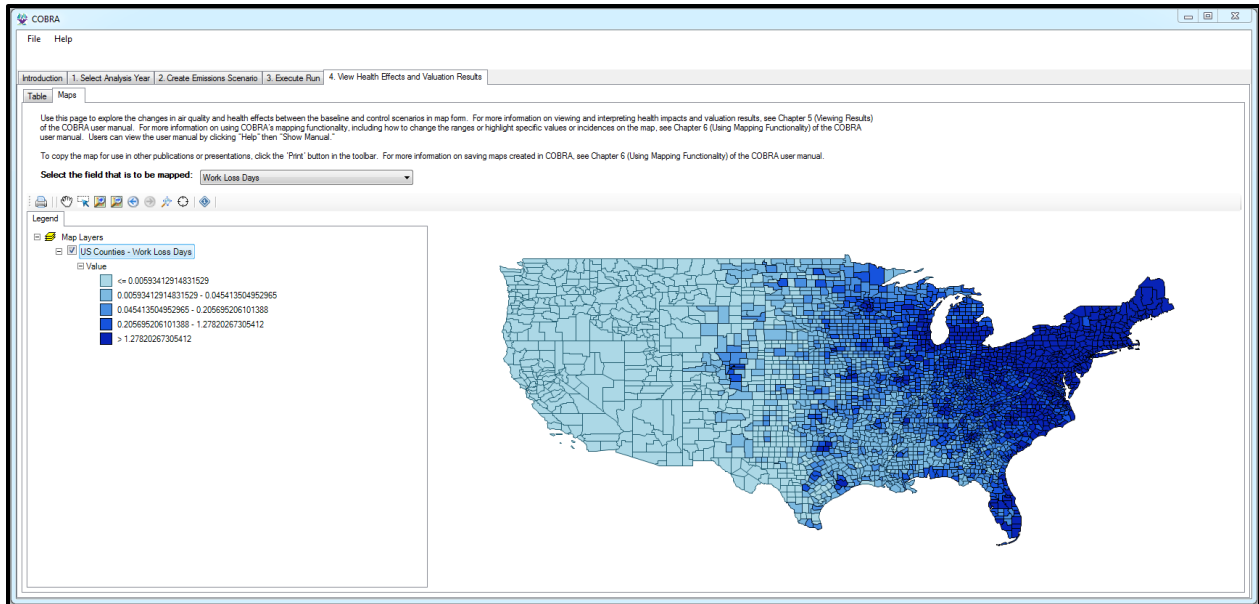
## Defining Interval Breaks

You can adjust how color intervals are defined in the bottom right panel. In the box to the right of 'Num Breaks' (indicated in red below), enter the number of intervals you would like your map to have.

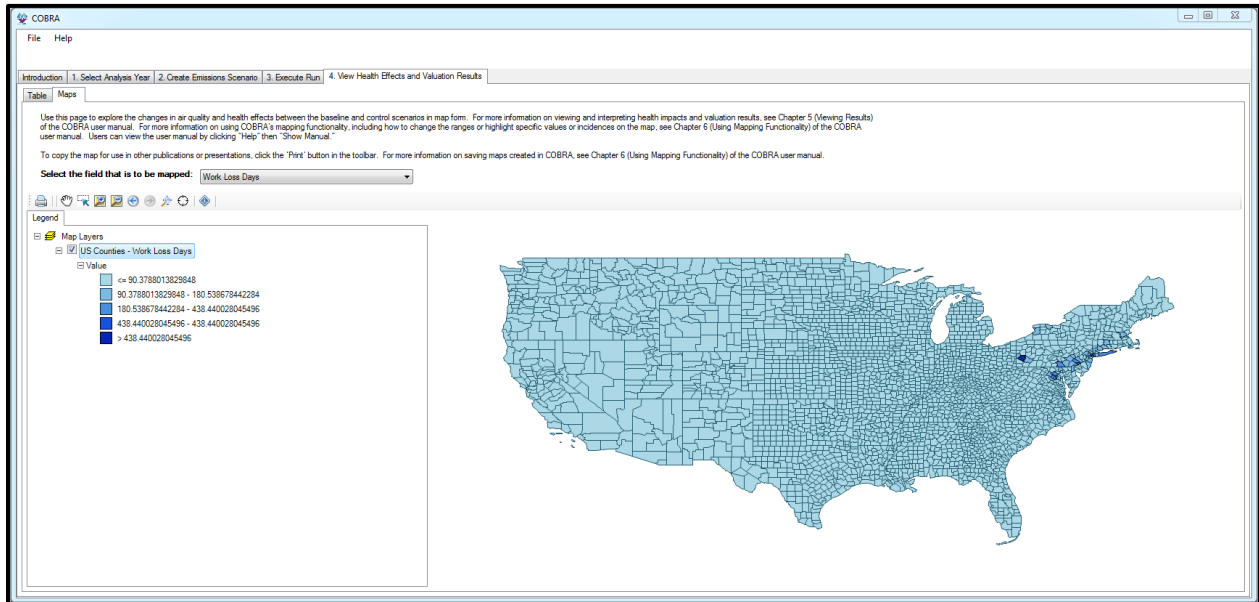


If you select 1, all counties will be in the same interval and the map will be a single color. If you select 10, there will be 10 intervals that can be 10 different colors. In this example, you should select 5. Next, select how COBRA should define the intervals. There are four possible methods, described below.

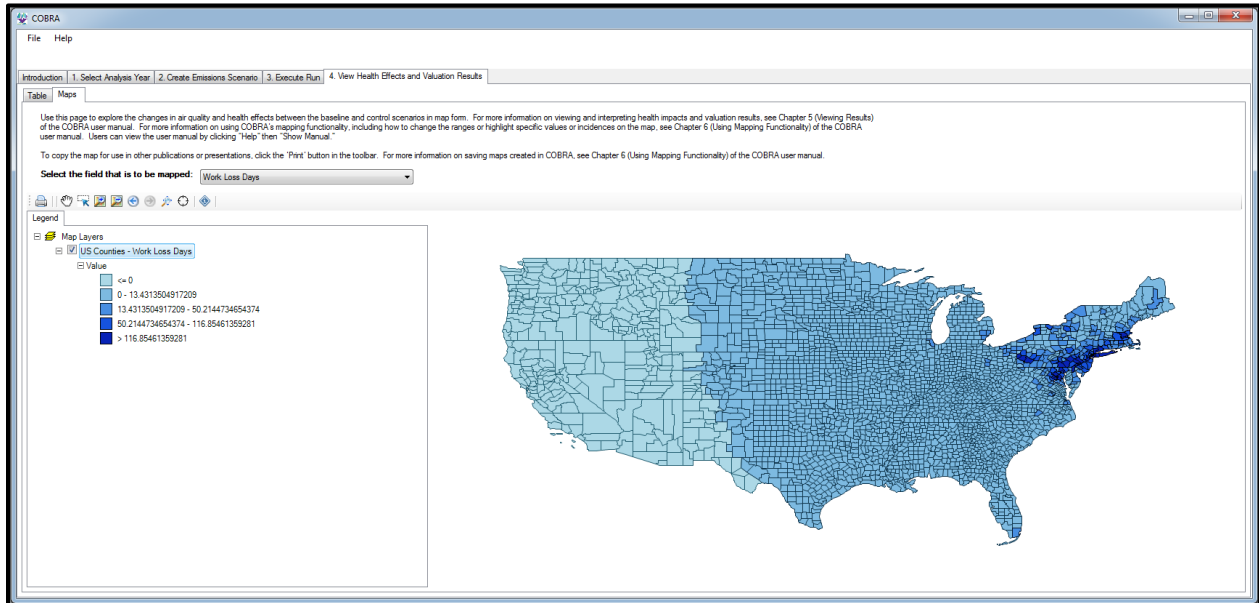
- Equal Frequency.** This method takes the range of the values, and, using the number of breaks you selected, splits the range into intervals where each interval contains the same number of counties. Below is the map of avoided work loss days where intervals are defined using the equal frequency option. This is COBRA's default setting.



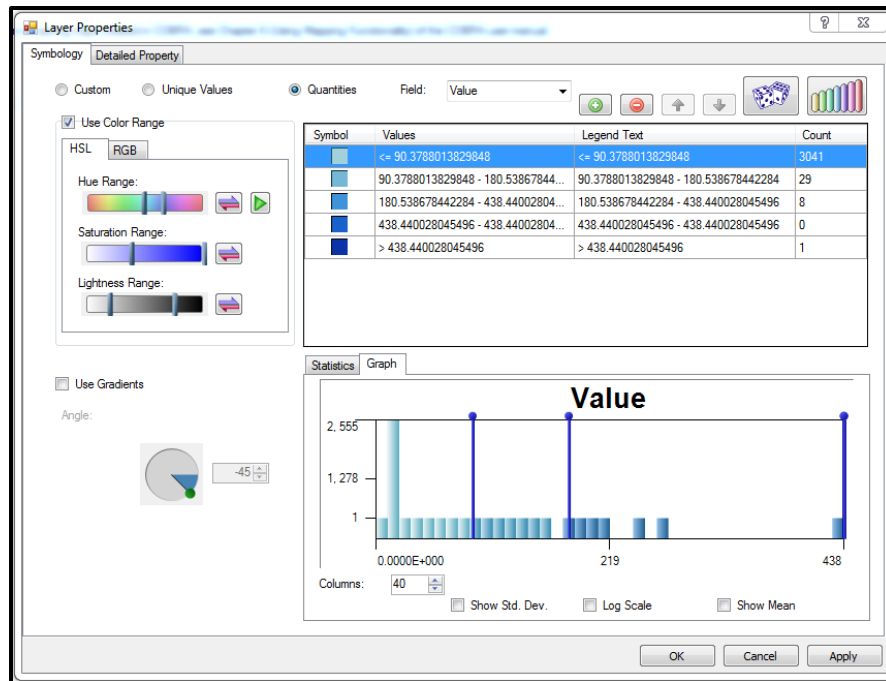
- Equal Intervals.** This method takes the range of the values for all counties in the U.S., and, using the number of breaks you selected, splits the range into equal intervals. Below is the map of avoided work loss days where intervals are defined using the equal intervals option.



- Natural Breaks.** This method divides counties into groups where the values (e.g., for avoided work loss days) are similar within groups and less similar to counties in other groups (based on variance in the values). Below is the map of avoided work loss days where intervals are defined using the natural breaks option.



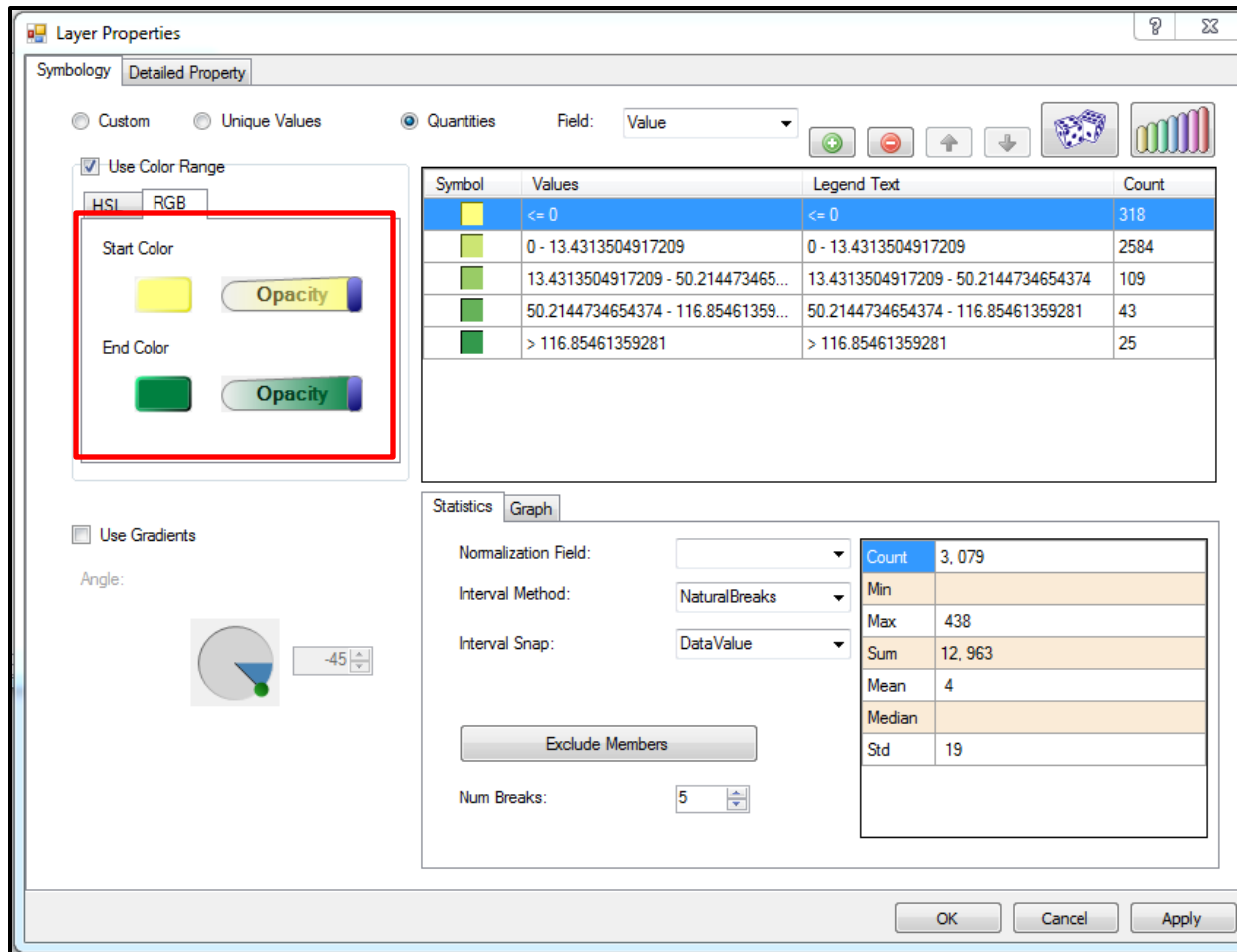
**Manual.** This method allows you to manually enter the range of each interval. To do so, click the **Graph** tab in the bottom right panel. This displays the distribution of values across all counties in the United States in a histogram. The x-axis is the value you are mapping (in this example, work loss days avoided) and the y-axis is the number of counties. The height of each rectangle is the number of counties with avoided work loss days within the range defined by the base of that rectangle. The blue lines are the boundaries defining the intervals for your color scheme. To change these boundaries, click on one of the blue lines and drag it to the boundary you want.



The method used to define a map's interval boundaries affects the conclusions that are likely to be drawn from the map, so it is important that users exercise care in selecting interval definition schemes. In this example, you can choose to define intervals using the Natural Breaks method.

### Changing Color Scheme








Navigate to the top left panel to change the map's color scheme. To create a simple spectrum that fades from one color to another color, click the **RGB** tab. Click the 'Start Color' box and select a color to represent the boxes with the smallest value. Then, click the 'End Color' box and select another color to represent the color with the largest value. In this example, choose yellow to represent counties with the smallest number of avoided work loss days and green to represent counties with the largest number of work loss days avoided.



After selecting interval breaks and deciding on a color scheme, you can review your selections in the top right panel. The 'Symbol' column shows the color that will represent all counties within each interval. The 'Values' column summarizes the definition of each interval. The 'Count' column lists the number of counties within each interval. The 'Legend Text' column displays the text that will be displayed in the legend. You can modify the legend text by double-clicking on the text.

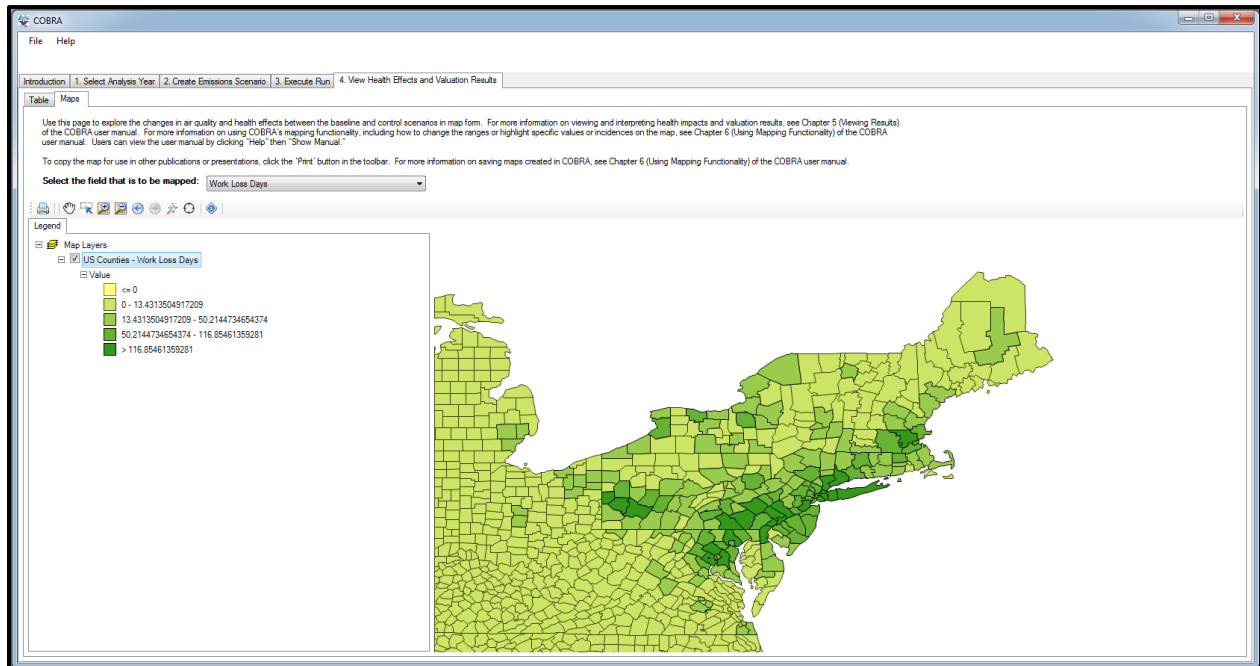
After creating the desired color scheme, click **Apply**.

### Modifying Map Scale

You can zoom in or out on the map by selecting **Zoom In** () or **Zoom Out** () in the top left toolbar and then clicking anywhere on the map. You can also zoom in on a specific area by clicking the **Zoom In** tool and then drawing a box with left mouse button depressed. To pan, click the **Pan** button () and then click and drag the map to the portion you wish to view. You can toggle between extents (i.e. map scales) by clicking **Zoom to Previous Extent** () and **Zoom to Next Extent** (). To zoom out to the full U.S., click **Zoom to Maximum Extents** () ). To center the map at a specific location, click **Zoom to Coordinates** () and input the

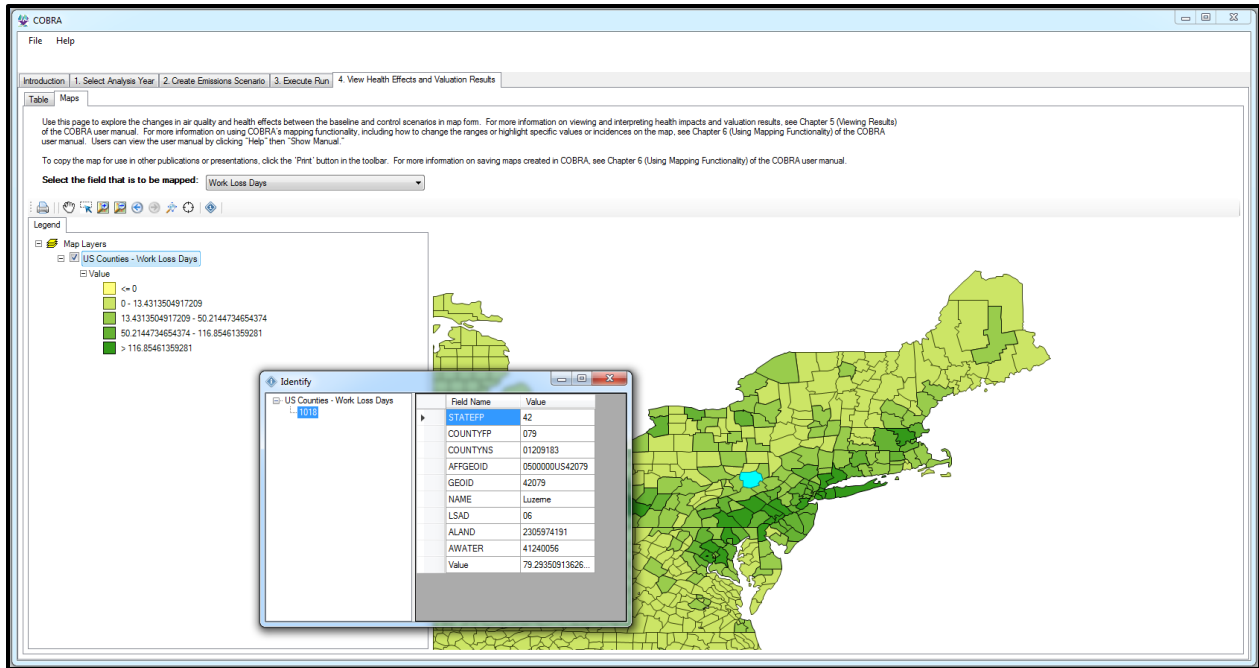


relevant latitude and longitude coordinates. Because this scenario focused on emissions reductions in Pennsylvania, in this example you will zoom in to the Northeast United States.

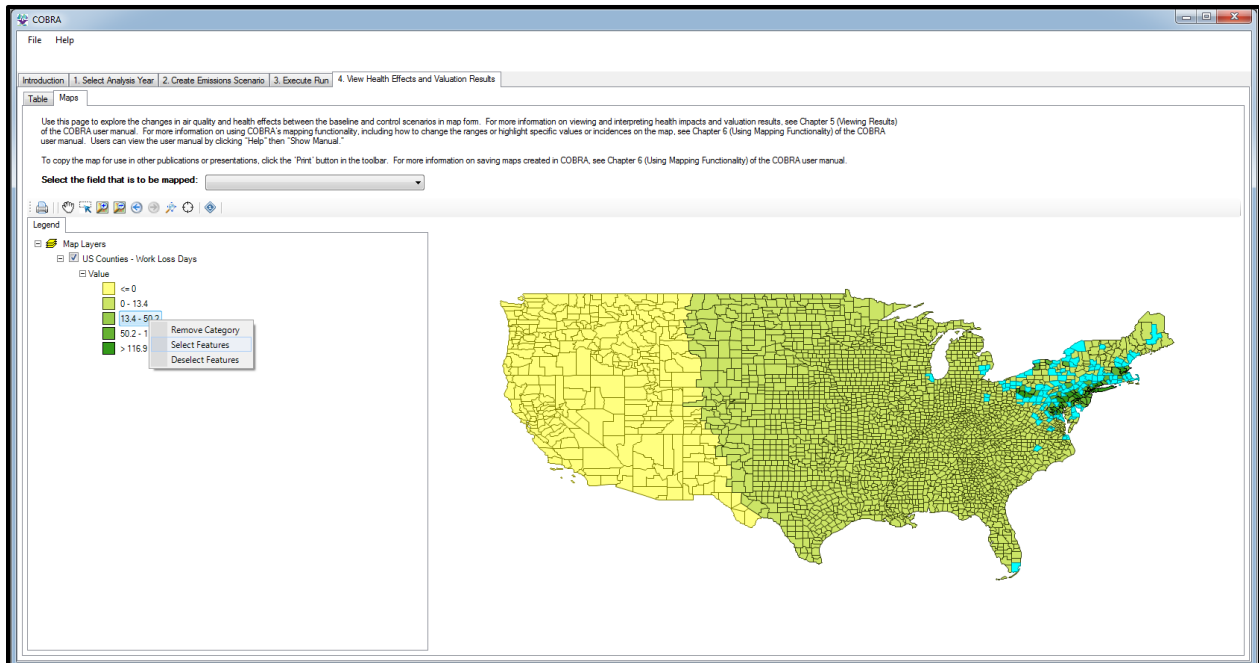


### Exploring Data

To get map information about a specific county, click the **Identifier** button (📍) in the top left toolbar and then click the county of interest. The selected county will be highlighted in teal and a box will pop up that contains the name of the county, the estimate for the value you are mapping for this county (e.g., avoided work loss days), and other identifying information for the county. For example, if you select Berks county, Pennsylvania you will see that the emissions reduction scenario will avoid about 134 work loss days in this county.



To select all counties that have values in a given interval (as described in the “Defining Interval Breaks” section above), right click on the interval in the Legend and then click ‘Select Features.’



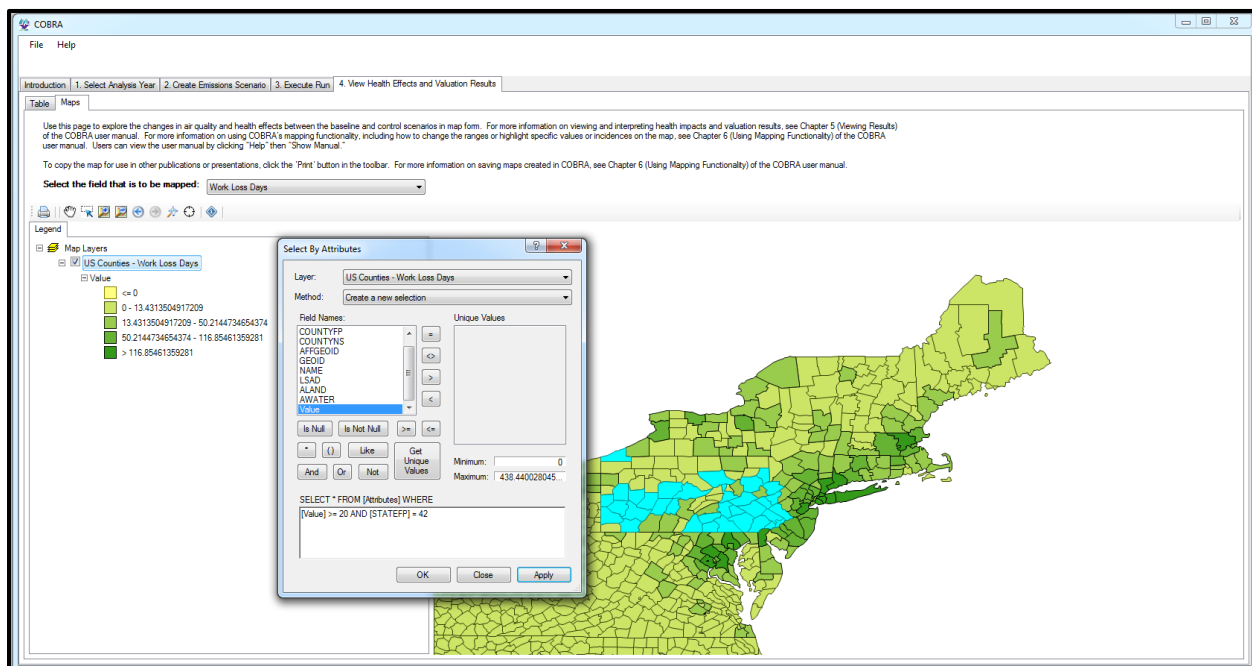
To find counties that match some criterion, right click the ‘US Counties’ drop-down, then click **Selection...Select by Attributes**. You can fill the box with the criteria you are interested in. For example, if you want to see which counties have more than 20 avoided work loss days and are

also located in Pennsylvania (which has a state FIPS code of 42<sup>10</sup>), you can make selections from the ‘Field Names’ list in the upper left of the pop up box. For this example, you would:

- First, double-click Value from the ‘Field Names’ list, click the ‘>=’ button, and then type 20. This sets the criterion for at least 20 avoided work loss days.
- You would then add the criterion for counties located in Pennsylvania as follows: click the ‘And’ button, double-click STATEFP from the ‘Field Names’ list, click the ‘=’ button, and then type 42.

Alternatively, once you are familiar with this functionality, you could simply write this equation in the box at the bottom of the window: “[Value]>=20 AND [STATEFP]=42”.

Once you have described your selection, click **Apply**. COBRA will highlight the counties that fit your criteria in teal.



## Exporting Map

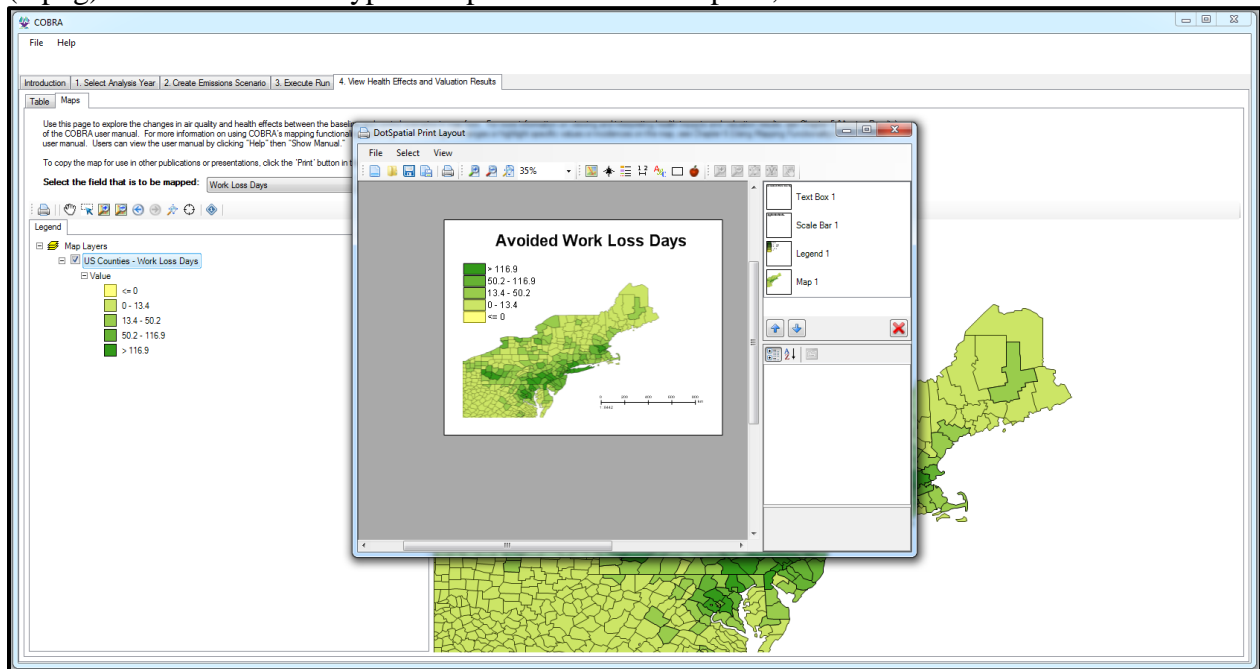
### *Printing or Exporting Map as Image*

To print the map or export a map as an image, click the **Print** button (🖨️) in the toolbar. To modify how much space the map takes up on the page, click the map, then click the bottom right

<sup>10</sup> FIPS codes uniquely identify United States counties and states. Codes can be accessed at: <https://www.census.gov/geo/reference/codes/cou.html>.

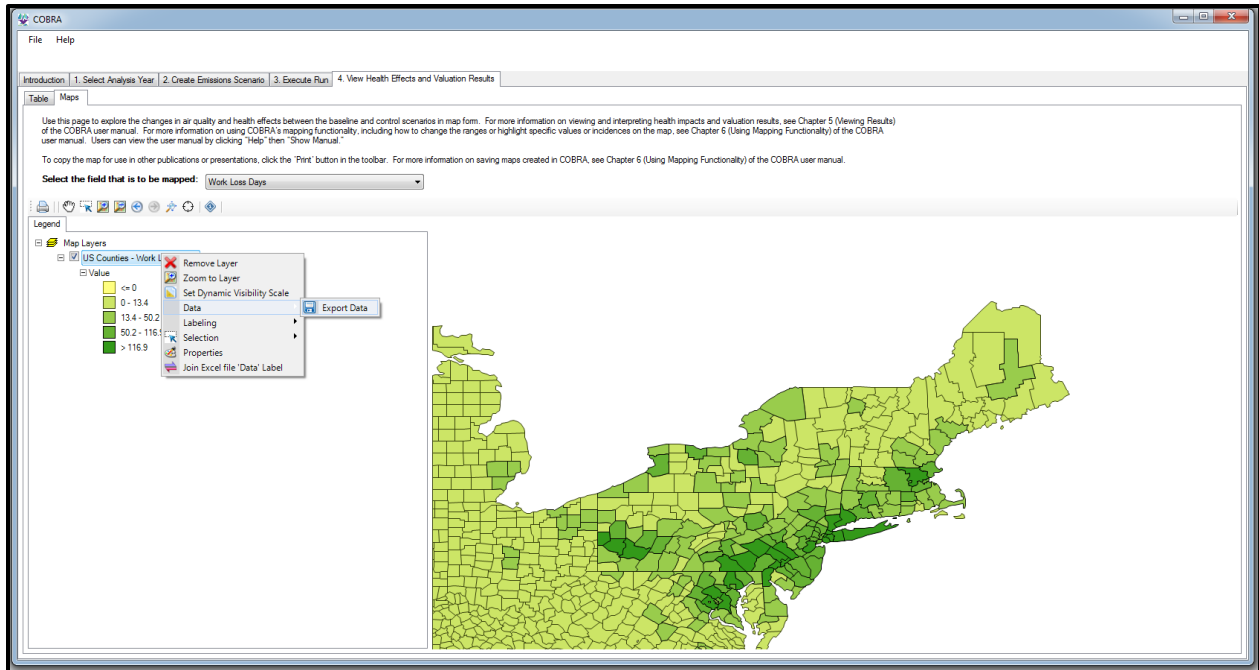
corner of the map and drag the rectangle. You can also click the center of the map and move it elsewhere on the page. You can zoom in and out and pan using the tools in the right hand side of the screen. To change the orientation of the page, click **File...Page Setup** then click Landscape. You can also add a compass, legend, scale bar, or text box from the toolbar. To edit the text box, click the text box and then edit the “Text” row in the bottom right panel. For example, you can add a title to the map “Avoided Work Loss Days” in a text box.

To save the map as an image, click **File...Save As** and then click Portable Network Graphics (\*.png) from the ‘Save as type:’ drop-down menu. To print, click **File...Print**.



### ***Exporting Map for Use in Other Mapping Software***

To export the map as a shapefile, right click ‘US Counties’ in the Legend tab. Then click **Data...Export Data**. Click the folder icon to navigate to the folder where you would like to save the map, and select ‘DotSpatial.Shapefile – Shapefiles (\*.shp)’ from the ‘Save as type:’ drop-down menu. Click **Save**.



## Glossary

**Baseline emissions scenario:** The emissions estimates for a given year in absence of a policy, ambient pollution levels and health impacts for that year. The baseline emissions scenario is compared to the control scenario when running COBRA.

**Control scenario:** A hypothetical scenario that factors in user-specified emissions changes (to 'control' emissions). In COBRA, the control scenario is compared to the baseline scenario.

**Delta PM<sub>2.5</sub>:** The difference in ambient concentrations of particulate matter that is less than or equal to 2.5 microns in diameter.

**Federal Information Processing Standards (FIPS) geographic codes:** codes which uniquely identify United States counties and states. Codes can be accessed at <https://www.census.gov/geo/reference/codes/cou.html>.

**Health impact function:** An equation that calculates the change in adverse health effects associated with a change in exposure to air population. A typical health impact function has inputs specifying the change in the air pollutant, an effect coefficient (specifying the percent change in an adverse health effect per unit change of a pollutant), the age of the population affected, and the incidence rate of the adverse health effect.

**Scenario definition:** A table of all edits made to the baseline emissions when defining a control scenario. The table can be viewed within COBRA or can be exported for future reference.

**Sensitivity analyses:** Comparison of analyses performed with varied assumptions or decisions to determine whether the assumptions/decisions have a major effect on the results of the analysis.

**Source-receptor matrix:** An air quality model built into COBRA that calculates the change in PM<sub>2.5</sub> levels for any given change in emissions. Appendix A discusses this model in more detail.

**Tier category:** Classification used by EPA for emission inventories.

# APPENDICES

## Appendix A: Dispersion Modeling in COBRA

COBRA estimates particulate matter levels using the Phase II Source-Receptor (S-R) Matrix. The S-R Matrix consists of fixed transfer coefficients that reflect the relationship between annual average PM<sub>2.5</sub> concentration values at a single receptor in each county (a hypothetical monitor located at the county centroid) and the contribution by PM<sub>2.5</sub> species to this concentration from each emission source (E.H. Pechan & Associates Inc., 1994).

Levy et al. (2003) found that an earlier version of the S-R Matrix predicted public health benefits that were similar to those predicted by CALPUFF, a comparatively more sophisticated model often used in risk assessments. Using the emission impacts from seven power plants in northern Georgia, Levy et al reported that the two models yielded generally similar results for sulfates or primary PM<sub>2.5</sub>, with somewhat greater differences for nitrates. However, they carefully noted that this result may differ depending on the location of the emissions, as temperature and humidity are important considerations in the formation of ambient particles.

Because of the limited validation studies of the S-R Matrix, it should be treated as a screening tool that provides a crude estimate of the likely impact of a change in emissions on ambient PM<sub>2.5</sub> levels. More sophisticated atmospheric dispersion models should be used to obtain detailed estimates of ambient air quality changes.

The sections below summarize the development of the S-R matrix and the steps taken to apply the matrix in COBRA in order to derive the changes in air quality resulting from changes in emissions.

### Development of the S-R Matrix

The S-R matrix is based on the Climatological Regional Dispersion Model (CRDM), which uses assumptions similar to the Industrial Source Complex Short Term model (ISCST3), an EPA-recommended short range Gaussian dispersion model (U.S. EPA, 1995). The CRDM incorporates terms for wet and dry deposition of primary and secondary species that constitute PM<sub>2.5</sub> and uses meteorological summaries (annual average mixing heights and joint frequency distributions of wind speed and direction) from 100 upper air meteorological sites throughout North America. This analysis employs meteorological data collected in 1990.

Relative to more sophisticated and resource-intensive three-dimensional modeling approaches, the CRDM does not fully account for all the complex chemical interactions that take place in the atmosphere in the secondary formation of PM<sub>2.5</sub>. Instead it relies on more simplistic species dispersion-transport mechanisms supplemented with chemical conversion at the receptor location.

The CRDM uses Turner's sector-average approach (Turner, 1970), a probabilistic method in which relative frequencies of occurrence of combinations of wind and stability conditions at the emissions source are used to calculate the relative frequencies of transport in various sectors.



This method is recommended for the estimation of long-term average pollutant concentrations (E.H. Pechan & Associates Inc., 1997).

The pollutant concentration in a destination sector is estimated as follows:

$$C_j(r) = \frac{2Q(r)}{y\sqrt{2\pi}} \sum_{i,k} \frac{f_{i,j,k}}{u_i \sigma_{z,k}} \exp \left[ -\frac{1}{2} \left( \frac{H}{\sigma_{z,k}} \right)^2 \right] \quad (1)$$

where:

- $C_j(r)$  = atmospheric concentration in destination sector  $j$  at distance  $r$
- $Q(r)$  = pollutant mass flux at distance  $r$
- $y$  = sector width at distance  $r$
- $f_{i,j,k}$  = joint frequency of wind speed class  $i$ , wind direction  $j$ , and stability category  $k$
- $\sigma_{z,k}$  = vertical diffusion coefficient for stability category  $k$
- $u_i$  = wind speed for wind class  $i$
- $H$  = effective stack height of emissions source (= 0 for ground-level sources)

The sector width is calculated as:

$$y = \left( \frac{2\pi}{16} \right) r \quad (2)$$

Primary emissions from a county are assumed to always impact the county source county itself and are evenly distributed over a square with the same area as the county. A simple box model is used for each combination of wind speed and stability category. The vertical diffusion coefficient,  $\sigma_z$ , is then calculated at a downwind distance corresponding to the side of the square.<sup>11</sup> These assumptions are necessary since the spatial variation of emissions within a county cannot be provided for a national scale model.<sup>12</sup>

Additional adjustments are made to ensure a consistent distribution of pollutant species among areas in close proximity to the emissions source. Receptors at a distance less than the square root

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<sup>11</sup> The vertical diffusion coefficient  $\sigma_z$  was calculated using a subroutine from EPA's ISC3 model. Atmospheric stabilities were assumed to be *C* class (slightly unstable) during the day and *E* class (slightly stable) at night. However, for wind speeds in excess of 6 m/s, stability was assumed to be neutral (class *D*).

<sup>12</sup> Actual measured concentrations would be expected to be higher than those modeled with these assumptions for a monitor located in, or generally downwind from, a portion of the county with emission densities much higher than the county average. On the other hand, concentrations would be expected to be lower if a monitor is located at the prevailing upwind edge of the county or in an area of relatively low emission density.

of the source area are assumed to receive the same concentration of pollutants as the source area. In addition, the destination sector width is constrained to be at least equal to the square root of the source area.

Equation (1) is applicable to both point and area sources, either ground-level or elevated, and results in a Gaussian distribution of pollutant mass in the vertical dimension. However, for long-range transport, emissions are distributed uniformly in the vertical between the top of the mixed layer and the ground. This occurs when the vertical diffusion parameter,  $\sigma_z$ , is equal to the height of the mixed layer,  $h_m$ . For such long-range situations, the sector-average limited mixing model of Turner (1970) estimates pollutant concentrations at a downward distance  $r$  from the source as:

$$C_j(r) = \frac{Q(r)}{h_m y} \sum_{i,k} \frac{f_{i,j,k}}{u_i} \quad (3)$$

The mass flux of a directly emitted primary species at distance  $r$  from the source is a function of the material initially emitted, the amount chemically converted to a secondary pollutant, and the amount deposited by wet and dry processes during the period of transport (time  $t$ ) from the emission point to the receptor. This is calculated by solving the relevant differential equation (Latimer, 1993):

$$Q_p(t) = Q_0 e^{-(k_c+k_p)t} \quad (4)$$

where:

- $Q_p(t)$  = primary pollutant mass flux at transport time  $t$
- $Q_0$  = initial emission rate
- $k_c$  = pseudo-first-order rate constant for chemical conversion of the primary species to the secondary species
- $k_p$  = pseudo-first-order rate constant for deposition of primary species, equal to the sum of the dry and wet deposition rate constants ( $k_{pd} + k_{pw}$ )
- $t$  = transport time

The mass flux of secondary pollutants is dependent upon the fraction of the primary species that is chemically converted in the atmosphere to the secondary species and the amount of the secondary species that is deposited by wet and dry deposition processes during the transport time  $t$  from the stack to the downwind receptor point at distance  $r$ . This is also calculated by solving the relevant differential equation (Latimer, 1993):

$$Q_s(t) = \frac{k_c Q_0}{k_c + k_p - k_s} \left( e^{-k_s t} - e^{-(k_c+k_p)t} \right) \quad (5)$$

where:

- $Q_s(t)$  = mass flux of the secondary species at transport time  $t$
- $Q_0$  = initial emission rate
- $k_c$  = pseudo-first-order rate constant for chemical conversion of the primary species to the secondary species
- $k_p$  = pseudo-first-order rate constant for deposition of primary species, equal to the sum of the dry and wet deposition rate constants ( $k_{pd} + k_{pw}$ )
- $k_s$  = pseudo-first-order rate constant for deposition of secondary species, equal to the sum of the dry and wet deposition rate constants ( $k_{sd} + k_{sw}$ )
- $t$  = transport time

The model parameters used to estimate mass flux are detailed in Exhibit A-1. Note that the pseudo-first-order rate constant for deposition,  $k_p$ , is estimated from the dry and wet deposition velocities by dividing them by the mixing height ( $h_m$ ).

**Exhibit A-1. Pollutant-specific Model Parameters**

	PM <sub>2.5</sub> , SOA*	SO <sub>2</sub> **	NO <sub>2</sub>	NH <sub>3</sub>
Chemical Conversion Rate, $k_c$ (%/hr) [RH = relative humidity (%)]	0	0.5 if RH < 40 1.5 if RH > 70 ((RH - 40)/30) + 0.5 Otherwise	2	0
Dry Deposition Velocity (cm/s)	0.1	0.5	1	1
Wet Deposition Velocity (cm/s)*** [P = annual precipitation rate (in.)]	0.01 P	0.003 P	0.0003 P	0.0003 P

\* Secondary organic aerosols.

\*\* The chemical conversion rate for SO<sub>2</sub> was parameterized as a function of relative humidity to account for greater atmospheric conversion rates in areas of the country with higher humidity.

\*\*\* Wet deposition velocities are from (Yamartino, 1985).

## Details of S-R Matrix Implementation in COBRA

In subsections below we provide the following implementation details on: (i) processing of the EPA's emissions data to create COBRA emissions baseline; (ii) meteorological data sources and processing; (iii) generating S-R transfer coefficients; (iv) approach taken to model secondary PM<sub>2.5</sub> formation (atmospheric chemistry); and (v) calibration of dispersion model outputs to the monitored PM<sub>2.5</sub>.

### ***Emissions Data***

We use emissions data from EPA's 2016v1 Air Emissions Modeling Platform (2016v1 platform) to forecast ambient 2016, 2023, and 2028 PM<sub>2.5</sub> levels in COBRA.<sup>13</sup> The 2016, 2023, and 2028 emissions inventories contain predicted emissions that reflect federal and state measures (promulgated or under reconsideration) as of May 2018.<sup>14</sup> The 2016, 2023, and 2028 base cases include:

- electrical generating unit emissions (reflecting the implementation of the Cross State Air Pollution Rule Update),
- the Mercury and Air Toxics Rule (MATS),
- the Standards of Performance for Greenhouse Gas Emissions from New, Modified, and Reconstructed Stationary Sources,
- mobile emissions (reflecting changes in activity data and the impacts the Tier 3 Motor Vehicle Emission and Fuel Standards Rule and local inspection and maintenance programs), and
- base year-specific fire data for 2016.

In addition, we used the 2016 base case emissions inventory to help develop calibration factors (discussed in more detail in a later section). Exhibit A-2a, Exhibit A-2b, and Exhibit A-2c summarize the 2016, 2023, and 2028 emissions data for the continental U.S. that we used.

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<sup>13</sup> Note that 2016 county-level biogenic emissions (from plants and soil) were estimated using the county total annual emissions by sector (available for download at <ftp://ftp.epa.gov/EmisInventory/2011v6/v2platform/reports/>) for EPA's 2011 Version 6.2 Air Emissions Modeling Platform.

<sup>14</sup> More details about the development of the 2016, 2023, and 2028 baseline emissions case are available in the supporting information for the 2016v1 Emissions Modeling Platform, available here: <https://www.epa.gov/air-emissions-modeling/2016v1-platform>

**Exhibit A-2a. 2016 Emissions Inventory Summary, by Tier 1 (tons/year)**

<b>Tier 1</b>	<b>NO<sub>x</sub></b>	<b>SO<sub>2</sub></b>	<b>PM<sub>2.5</sub></b>	<b>VOC</b>	<b>NH<sub>3</sub></b>
Fuel Combustion Electric Utilities	1,213,176	1,509,524	123,023	33,346	22,301
Fuel Combustion Industrial	962,831	367,909	179,510	105,988	15,420
Fuel Combustion Other	521,545	85,937	346,201	373,681	62,034
Chemical & Allied Product Manuf.	41,867	111,625	14,476	79,541	21,934
Metals Processing	60,542	85,099	34,984	22,838	638
Petroleum & Related Industries	593,419	80,056	25,818	2,873,249	1,404
Other Industrial Processes	319,965	138,608	260,264	336,096	28,362
Solvent Utilization	1,248	65	3,745	3,035,948	422
Storage & Transport	2,960	818	15,426	661,600	3,827
Waste Disposal & Recycling	109,378	30,976	225,040	229,311	29,399
Highway Vehicles	3,630,548	27,559	117,758	1,852,260	100,318
Off-Highway Vehicles	2,173,402	33,326	136,316	1,258,667	2,190
Natural Sources	965,761	0	0	42,133,700	0
Miscellaneous	239,994	115,256	3,723,122	3,324,005	3,700,883
<b>Total</b>	<b>10,836,636</b>	<b>2,586,758</b>	<b>5,205,683</b>	<b>56,320,230</b>	<b>3,989,132</b>

**Exhibit A-2b. 2023 Emissions Inventory Summary, by Tier 1 (tons/year)**

<b>Tier 1</b>	<b>NO<sub>x</sub></b>	<b>SO<sub>2</sub></b>	<b>NH<sub>3</sub></b>	<b>PM<sub>2.5</sub></b>	<b>VOC</b>
Fuel Combustion Electric Utilities	784,566	790,808	38,924	122,222	37,822
Fuel Combustion Industrial	929,186	280,440	12,628	178,905	115,012
Fuel Combustion Other	516,645	48,594	61,375	332,817	362,513
Chemical & Allied Product Manuf.	40,321	103,924	21,555	14,802	82,224
Metals Processing	54,640	67,825	638	35,047	22,970
Petroleum & Related Industries	607,132	92,748	1,412	28,081	3,291,678
Other Industrial Processes	312,538	129,497	28,311	268,509	345,611
Solvent Utilization	1,249	65	422	3,754	3,139,345
Storage & Transport	2,648	847	3,825	15,411	619,369
Waste Disposal & Recycling	106,723	32,391	29,525	225,221	229,983
Highway Vehicles	1,750,937	12,484	89,285	72,468	1,098,966
Off-Highway Vehicles	1,694,745	38,165	2,380	96,121	987,588
Natural Sources	965,761	0	0	0	42,133,700
Miscellaneous	240,053	115,265	3,834,279	3,750,534	3,334,678
<b>Total</b>	<b>8,007,144</b>	<b>1,713,053</b>	<b>4,124,559</b>	<b>5,143,892</b>	<b>55,801,459</b>

**Exhibit A-2c. 2028 Emissions Inventory Summary, by Tier 1 (tons/year)**

<b>Tier 1</b>	<b>NO<sub>x</sub></b>	<b>SO<sub>2</sub></b>	<b>NH<sub>3</sub></b>	<b>PM<sub>2.5</sub></b>	<b>VOC</b>
Fuel Combustion Electric Utilities	766,652	808,476	42,154	121,562	37,971
Fuel Combustion Industrial	929,420	285,579	13,078	188,424	117,610
Fuel Combustion Other	508,511	47,450	61,127	328,496	358,818
Chemical & Allied Product Manuf.	45,755	103,938	21,572	15,125	82,776
Metals Processing	54,690	68,071	641	35,099	23,018
Petroleum & Related Industries	582,205	95,879	1,419	28,339	3,420,707
Other Industrial Processes	313,274	129,739	28,298	273,239	347,825
Solvent Utilization	1,252	65	424	3,764	3,229,409
Storage & Transport	2,651	849	3,827	15,426	570,872
Waste Disposal & Recycling	106,354	32,379	29,631	225,214	230,322
Highway Vehicles	1,246,019	11,703	87,913	58,925	836,112
Off-Highway Vehicles	1,524,774	42,668	2,465	81,990	938,552
Natural Sources	965,761	0	0	0	42,133,700
Miscellaneous	240,101	115,272	3,855,188	3,763,155	3,336,351
<b>Total</b>	<b>7,287,419</b>	<b>1,742,068</b>	<b>4,147,737</b>	<b>5,138,758</b>	<b>55,664,043</b>

We estimate the formation of SOA using a fixed relationship between SOA and VOC for each Tier 3 emission category.<sup>15</sup> EPA’s 2016v1 platform estimated VOC but did not estimate SOA, so we developed a simple approach to estimate the conversion of VOC to SOA, though this conversion actually depends upon a number of factors including climate and the type of VOC. We used the 2010 base case inventory of SOA and VOC emissions generated for the Clear Skies Act (CSA) of 2003 (U.S. EPA, 2003b). For each Tier 3 emission category in this inventory, we calculated the ratio of SOA to VOC. We then used these Tier 3 category-specific ratios to estimate SOA in the emissions inventory from the 2016v1 platform:

$$SOA_{2016v1, \text{ Tier 3}} = VOC_{2016v1, \text{ Tier 3}} \cdot \left( \frac{SOA_{CSA, \text{ Tier 3}}}{VOC_{CSA, \text{ Tier 3}}} \right)$$

When modeling emission sources, we categorized them into elevated point sources and area/mobile sources. For each, we calculate an “effective stack” height, which takes into account the actual stack height, gas temperature and velocity, stack diameter, and other factors. The effective stack height is important as it is one of the greatest determinants<sup>16</sup> of how far emissions will disperse – generally the taller the effective stack the further the emissions might travel from

<sup>15</sup> The emissions inventory in COBRA has fourteen broad Tier 1 categories (e.g., on-road motor vehicles), and within each of these larger categories there are Tier 2 (e.g., diesels), and Tier 3 (e.g., heavy duty diesels) categories.

<sup>16</sup> The other determinants include wind speed and direction as well as atmospheric chemistry.

the source. In calculating effective stack height, we assume an average wind speed of 5 meters per second using the plume rise algorithm from ISCST3 (U.S. EPA, 1995).

We group stationary point source emissions for each county into three groups based on effective stack height: (1) less than 250 meters, (2) 250 to 500 meters, and (3) greater than 500 meters.<sup>17</sup> We assume that emissions from the two groups less than 500 meters originate from the center of the county in which they are located. For point sources with effective stack heights greater than 500 meters, we use their true latitude and longitude coordinates when modeling the dispersion of emissions.<sup>18</sup>

Emissions from both ground-level mobile and area sources in the contiguous U.S. are combined at the county-level and modeled as emissions from stacks with an effective stack height of zero located at the source county centroid. Exhibit A-3 summarizes these emission categories.

**Exhibit A-3. Emissions Categories for the S-R Matrix**

<b>Emissions Category</b>	<b>Effective Stack Height</b>	<b>Modeled Location</b>
U.S. area and mobile emissions	0 m	County center
U.S. elevated point emissions	0-250 m	County center
U.S. elevated point emissions	250-500 m	County center
U.S. elevated point emissions	>500 m	True location

### ***Meteorological Data***

Meteorological variables were calculated from rawinsonde data on the NAMER-WINDTEMP tapes<sup>19</sup> obtained from the National Climatic Data Center. Winds for each of 100 sites throughout North America were averaged for the following layers: the surface to 250 meters above ground level (m AGL), 250-500 m AGL, 500-1000 m AGL, 1000-2000 m AGL, and 2000-4000 m AGL. For each of these levels and for each of the 100 meteorological sites, a joint frequency distribution of wind direction (16 cardinal directions) and wind speeds (11 speeds in 1 m/s increments) was calculated for 1990.

These distributions were calculated separately for the twice-daily soundings. The early morning soundings were assumed to be associated with the *E* stability category, and the late afternoon soundings were assumed to be associated with the *C* stability category. Mixing heights were determined from each sounding by calculating the virtual potential temperature. The annual

<sup>17</sup> In a very small number of cases (0.32% of records in the emissions baseline), there are point source emissions in multiple stack height groups for the same Tier 3 emission category in the same county. When emissions changes are entered in the COBRA model for these cases, the changes are apportioned to stack height groups in proportion to the baseline scenario emissions.

<sup>18</sup> For some counties, the emissions inventory contained more than one emission source with stack height greater than 500m. These emission sources normally have different locations and stack heights. To create a composite county-level emissions source with stack height greater than 500m, we used the latitude and longitude of the source with the tallest stack, whereas the composite stack height was an emissions-weighted average.

<sup>19</sup> Refers to North America wind and temperature. These are standard data tapes for upper-air (rawinsonde) data collected twice daily throughout North America. Rawinsondes are radar-tracked wind balloons.

average afternoon mixing heights were calculated for each of the 100 meteorological sites and were used to calculate the upper limit of vertical diffusion ( $h_m$ ). The appropriate wind layer for concentration calculations was determined using the centroid of the diffusing plume:  $\sigma_z$  for a ground-based plume that has not yet mixed uniformly in the vertical,  $H$  for an elevated source, and  $h_m/2$  for a uniformly mixed plume (E.H. Pechan & Associates Inc., 1994).

### **S-R Transfer Coefficients**

The S-R matrix used in COBRA estimated the transport of the following emissions species: (1) directly emitted PM<sub>2.5</sub> and secondary organic aerosols (SOA), (2) sulfur dioxide (SO<sub>2</sub>), (3) nitrogen dioxide (NO<sub>2</sub>), and (4) ammonia (NH<sub>3</sub>). These species were then used in the calculation of ambient concentrations of PM<sub>2.5</sub>.

A matrix of source-receptor coefficients (in units of s/m<sup>3</sup>) spanning the entire contiguous U.S. was developed for each of the four pollutants using the CRDM. For a unique combination of source and receptor sites, a S-R transfer coefficient represents the incremental ambient air quality impact in µg/m<sup>3</sup> at the receptor resulting from a 1 µg/s unit emission from the source. The S-R matrix therefore provides a link between emission reductions and resulting air quality concentrations. Concentration reductions that occur in proportion to a decrease in emissions at a source are determined by the S-R coefficients for a given source and all receptors.

The pollutant concentration at a destination county is given by:

$$D_j^s = \sum_i \sum_c E_{c,i}^s T_{c,i,j}^s \cdot F^s \cdot F_{unit} \quad (6)$$

where:

- $D_j^s$  = Concentration of pollutant  $s$  at destination county  $j$  (µg/m<sup>3</sup>)
- $E_{i,c}^s$  = Emission of pollutant  $s$  from emissions category  $c$  in source county  $i$  (tons/year)
- $T_{i,j}^s$  = Transfer coefficient for pollutant  $s$  from source county  $i$  to destination county  $j$  for emissions category  $c$  (sec/m<sup>3</sup>)
- $F^s$  = Ionic conversion factor for pollutant  $s$
- $F_{unit}$  = Unit conversion factor (28,778 µg-year/ton-sec)

The ionic conversion factors are molecular weight ratios used to adjust the transfer coefficients to reflect the concentration of precursors to secondarily-formed particulate species. Standard molecular weights along with the ionic conversion factors used in this analysis are given in Exhibit A-4 and Exhibit A-5.



**Exhibit A-4. Standard Molecular Weights**

Species	Symbol	Standard molecular weight <sup>20</sup>
Nitrate ion	NO <sub>3</sub> <sup>-</sup>	62.0049
Sulfate ion	SO <sub>4</sub> <sup>2-</sup>	96.0626
Bisulfate	HSO <sub>4</sub>	97.07054
Sulfur Dioxide	SO <sub>2</sub>	64.0638
Nitrogen Dioxide	NO <sub>2</sub>	46.0055
Ammonia	NH <sub>3</sub>	17.03052
Ammonium ion	NH <sub>4</sub> <sup>+</sup>	18.03846
Ammonium Nitrate	NH <sub>4</sub> NO <sub>3</sub>	80.04336
Ammonium Bisulfate	NH <sub>4</sub> HSO <sub>4</sub>	115.109
Ammonium Sulfate	(NH <sub>4</sub> ) <sub>2</sub> SO <sub>4</sub>	132.13952

**Exhibit A-5. Ionic Conversion Factors**

Species	Ionic conversion factor, F <sup>s</sup>
PM <sub>2.5</sub> , SOA	1
SO <sub>2</sub> → SO <sub>4</sub> <sup>2-</sup>	96.0626 / 64.0638
NO <sub>2</sub> → NO <sub>3</sub> <sup>-</sup>	62.0049 / 46.0055
NH <sub>3</sub> → NH <sub>4</sub> <sup>+</sup>	18.03846 / 17.03052

### **Atmospheric Chemistry**

This section describes how secondary reactions are modeled in COBRA, including formation of ammonium bisulfate (NH<sub>4</sub>HSO<sub>4</sub>), ammonium sulfate ((NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub>), and ammonium nitrate (NH<sub>4</sub>NO<sub>3</sub>). Note that the COBRA treats atmospheric chemistry involved in the formation of these pollutants in a more simplified fashion than state-of-the-art air quality models<sup>21</sup> (e.g., CALPUFF, AERMOD, CMAQ). We try to address this problem by calibrating COBRA modeling results to measured PM<sub>2.5</sub> concentrations as described in later in this Appendix. Nevertheless uncertainty remains.

For the atmospheric chemistry in COBRA, in the presence of sulfate (SO<sub>4</sub><sup>2-</sup>) and nitrate (NO<sub>3</sub><sup>-</sup>), ammonium (NH<sub>4</sub><sup>+</sup>) reacts preferentially with SO<sub>4</sub><sup>2-</sup> to form NH<sub>4</sub>HSO<sub>4</sub> and (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub>. NH<sub>4</sub>NO<sub>3</sub> is only formed under conditions of excess NH<sub>4</sub><sup>+</sup> and low temperatures. In each destination county, the relative amounts of each secondary particle are subject to the following assumptions:

- SO<sub>4</sub><sup>2-</sup> is always assumed to be a particle;
- NO<sub>3</sub><sup>-</sup> is assumed to be a gas, unless it combines with NH<sub>4</sub><sup>+</sup>;

<sup>20</sup> Standard atomic weights from Coursey, et al. (2011).

<sup>21</sup> See U.S. [EPA \(2012\)](#).

- $\text{NH}_4^+$  reacts first with  $\text{SO}_4^{2-}$ . The nature of the reaction depends on the relative amounts of  $\text{NH}_4^+$  and  $\text{SO}_4^{2-}$ :
  - If there is a little  $\text{NH}_4^+$ , then  $\text{SO}_4^{2-}$  will be converted to  $\text{NH}_4\text{HSO}_4$  with potentially some leftover  $\text{SO}_4^{2-}$ ;
  - If there is an intermediate amount of  $\text{NH}_4^+$ , then a combination of  $\text{NH}_4\text{HSO}_4$  and  $(\text{NH}_4)_2\text{SO}_4$  will be obtained;
  - If there is a lot of  $\text{NH}_4^+$ , then  $\text{SO}_4^{2-}$  will be completely converted to  $(\text{NH}_4)_2\text{SO}_4$ ;
- After all reactions between  $\text{NH}_4^+$  and  $\text{SO}_4^{2-}$  occur, any remaining  $\text{NH}_4^+$  reacts with  $\text{NO}_3^-$  to form  $\text{NH}_4\text{NO}_3$ .

Below we lay out the specifics of our approach:

**Step 1:** Calculate the mole ratio of  $\text{NH}_4^+$  to  $\text{SO}_4^{2-}$ .

$$R = (\text{NH}_4^+ / 18.03846) / (\text{SO}_4^{2-} / 96.0626):$$

**a)** If  $R < 1$  then we assume that a portion of  $\text{SO}_4^{2-}$  converts to  $\text{NH}_4\text{HSO}_4$  ( $\text{SO}_4^{2-} + \text{NH}_4^+ \rightarrow \text{NH}_4\text{HSO}_4$ ), while the rest remains as  $\text{SO}_4^{2-}$

- Resulting concentration of  $\text{NH}_4\text{HSO}_4$  is  
 $15.109 \cdot \min\{(\text{NH}_4^+ / 18.03846), (\text{SO}_4^{2-} / 96.0626)\}$

- Resulting concentration of remaining  $\text{SO}_4^{2-}$  is  
 $96.0626 \cdot ((\text{SO}_4^{2-} / 96.0626) - (\text{NH}_4^+ / 18.03846))$

**b)** If  $1 \leq R < 2$  then we assume that all  $\text{SO}_4^{2-}$  converts to  $\text{NH}_4\text{HSO}_4$  ( $\text{SO}_4^{2-} + \text{NH}_4^+ \rightarrow \text{NH}_4\text{HSO}_4$ ) and a portion of  $\text{NH}_4\text{HSO}_4$  converts to  $(\text{NH}_4)_2\text{SO}_4$  ( $\text{NH}_4\text{HSO}_4 + \text{NH}_4^+ \rightarrow (\text{NH}_4)_2\text{SO}_4$ ). The second reaction will occur if there is enough  $\text{NH}_4^+$  remaining after the first reaction.

- Resulting concentration of  $\text{NH}_4\text{HSO}_4$  is  
 $115.109 \cdot (2(\text{SO}_4^{2-} / 96.0626) - (\text{NH}_4^+ / 18.03846))$

- Resulting concentration of  $(\text{NH}_4)_2\text{SO}_4$  is  
 $132.13952 \cdot ((\text{NH}_4^+ / 18.03846) - (\text{SO}_4^{2-} / 96.0626))$

**c)** If  $R \geq 2$  then we assume that all  $\text{SO}_4^{2-}$  converts to  $(\text{NH}_4)_2\text{SO}_4$  ( $\text{SO}_4^{2-} + 2\text{NH}_4^+ \rightarrow (\text{NH}_4)_2\text{SO}_4$ ).

- Resulting concentration of  $(\text{NH}_4)_2\text{SO}_4$  is  
 $132.13952 \cdot (\text{SO}_4^{2-} / 96.0626)$

- Resulting concentration of  $\text{NH}_4^+$  (remaining) is  
 $18.03846 \cdot ((\text{NH}_4^+ / 18.03846) - 2(\text{SO}_4^{2-} / 96.0626))$

**Step 2:** If  $\text{NH}_4^+$  remains after Step 1 (c), then  $\text{NH}_4\text{NO}_3$  formation can take place. The number moles of  $\text{NO}_3^-$  neutralized in this reaction will be:

$$\text{moles of } \text{NO}_3^- \text{ (neutralized)} = \min\left\{\left(\text{NH}_4^+ \text{ (remaining)} / 18.03846\right), \left(\text{NO}_3^- / 62.0049\right)\right\}.$$

**Step 3:** Particulate  $\text{NH}_4\text{NO}_3$  is stable at relatively low temperatures. Following prior usage of the S-R Matrix (e.g.,  $\text{NO}_x$  SIP Call), we assume that nitrate converts to ammonium nitrate only a quarter of the time (i.e., the winter months). The annual average concentration of  $\text{NH}_4\text{NO}_3$  formed by the neutralization process is therefore:

$$80.04336 \cdot 0.25 \cdot \text{moles of } \text{NO}_3^- \text{ (neutralized)}.$$

**Step 4:** The concentration of  $\text{PM}_{2.5}$  at the destination county is estimated as the sum of concentrations of primary  $\text{PM}_{2.5}$ , SOA, remaining  $\text{SO}_4^{2-}$  (if any) and secondary  $\text{NH}_4\text{HSO}_4$ ,  $(\text{NH}_4)_2\text{SO}_4$ , and  $\text{NH}_4\text{NO}_3$ :

$$\begin{aligned} \text{PM}_{2.5} \text{ (total)} &= \text{PM}_{2.5} \text{ (primary)} + \text{SOA} \\ &\quad + \text{SO}_4^{2-} \text{ (remaining)} \\ &\quad + \text{NH}_4\text{HSO}_4 + (\text{NH}_4)_2\text{SO}_4 + \text{NH}_4\text{NO}_3 \end{aligned}$$

### ***Calibration of S-R Matrix Outputs to Monitoring Data***

We calibrated the S-R Matrix model estimates to actual monitoring data obtained from EPA. The county-level calibration factors were estimated using the 2016 emissions inventory from EPA's 2016v1 platform and 2016 data from EPA Federal Reference Method (FRM) monitor sites and EPA/National Park Service Visibility Interagency Monitoring of Protected Visual Environments (IMPROVE) program monitor sites.<sup>22</sup>

First, we used the S-R Matrix with the 2016 emissions inventory to estimate  $\text{PM}_{2.5}$  levels at the center of each county. Second, we spatially interpolated the  $\text{PM}_{2.5}$  monitor data to generate a monitor-based estimate for each county center as follows:

1. We pre-processed EPA monitoring data to ensure that it did not contain any values flagged as invalid and that minimum number of daily measurements per quarter was 11;<sup>23</sup>
2. We calculated quarterly average  $\text{PM}_{2.5}$  concentrations for all monitoring sites with sufficient data;
3. For each quarter, we used an automatic kriging routine from R project package 'automap' (Hiemstra, 2012) to interpolate quarterly average  $\text{PM}_{2.5}$  values to county centroids;

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<sup>22</sup> 2011 daily  $\text{PM}_{2.5}$  monitoring data from EPA were downloaded at [http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download\\_files.html](http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html) (U.S. EPA, 2011d).

<sup>23</sup> The choice of 11 as the minimum number of site-days per valid quarter corresponds to > 75% completeness for monitors on a 1 in 6 day schedule. This is a minimum number of samples that is routinely used in calculations of quarterly average concentrations by EPA.

4. At each county centroid we then average over the interpolated quarterly average  $PM_{2.5}$  values to generate an annual average  $PM_{2.5}$  value.

We calculated a “calibration factor” for each county by dividing our monitor estimate by the model estimate. These county-level calibration factors ranged from 0.19 to 3.32 with a mean value of 0.76. For each state, Exhibit A-6 gives the average of the county-level monitor and model values as well as the ratio of the two (the ratio being the average of the calibration factors).

When calculating future year  $PM_{2.5}$  levels in COBRA, we use the calibration factors to adjust our model estimate for each county in the following way:

$$PM_{2.5}(\text{calibrated model, 2016, 2023, or 2028}) \\ = PM_{2.5}(\text{model, 2016, 2023, or 2028}) \cdot \left( \frac{PM_{2.5}(\text{interpolated monitor, 2016})}{PM_{2.5}(\text{model, 2016})} \right)$$

To sum up, the steps involved in the calculation of 2016, 2023, or 2028 ambient  $PM_{2.5}$  levels in COBRA are the following. We start the process by running the CRDM model, which generates the S-R Matrix transfer coefficients. Emissions data for 2016 are run through the S-R Matrix and atmospheric chemistry calculations applied to generate un-calibrated 2016 model estimates. Monitoring data for 2016 were interpolated to the county-level, and were then compared with the 2016 model estimates to generate calibration factors. Estimates of 2016, 2023, or 2028 ambient  $PM_{2.5}$  levels can then be generated by running the 2016, 2023, or 2028 emissions data through the S-R Matrix. The resulting 2016, 2023, or 2028 model  $PM_{2.5}$  levels are then multiplied with the previously generated county-level calibration factors to calculate a best estimate of the 2016, 2023, or 2028 calibrated ambient  $PM_{2.5}$  levels.

**Exhibit A-6. Monitor and Model Average PM<sub>2.5</sub> Levels (ug/m<sup>3</sup>) in 2016 and Average of Monitor to Model Ratios by State**

State	Monitor	Model	Ratio	State	Monitor	Model	Ratio
AL	8.16	14.27	0.57	MT	4.37	3.85	1.13
AZ	4.58	5.03	0.91	NE	5.47	5.79	0.94
AR	7.93	13.00	0.61	NV	4.44	4.61	0.96
CA	6.84	6.20	1.10	NH	5.04	7.46	0.68
CO	3.90	5.94	0.66	NJ	7.59	11.63	0.65
CT	6.29	9.27	0.68	NM	3.75	5.64	0.66
DE	7.27	9.19	0.79	NY	5.99	8.28	0.72
DC	7.98	16.66	0.48	NC	7.43	11.49	0.65
FL	7.32	10.30	0.71	ND	3.75	4.58	0.82
GA	8.21	15.16	0.54	OH	7.79	13.43	0.58
ID	5.21	6.40	0.81	OK	7.06	9.82	0.72
IL	8.03	11.14	0.72	OR	4.95	5.20	0.95
IN	8.25	12.24	0.67	PA	8.20	10.31	0.79
IA	6.90	7.09	0.97	RI	5.70	8.10	0.70
KS	6.05	7.78	0.78	SC	8.16	12.62	0.65
KY	8.15	12.30	0.66	SD	4.68	4.79	0.98
LA	8.28	11.48	0.72	TN	7.91	12.84	0.62
ME	4.77	5.11	0.93	TX	7.13	7.96	0.90
MD	7.56	11.16	0.68	UT	4.71	6.02	0.78
MA	5.62	9.16	0.61	VT	4.59	6.12	0.75
MI	6.32	7.56	0.84	VA	7.04	12.53	0.56
MN	5.30	7.77	0.68	WA	4.65	4.61	1.01
MS	8.00	13.23	0.60	WV	7.21	11.73	0.62
MO	7.25	11.44	0.63	WI	5.89	8.43	0.70
				WY	3.86	4.98	0.78

## Appendix B: Derivation of Health Impact Functions

This Appendix reviews the steps we performed in taking models from the epidemiological study and converting them into health impact functions, which we then use to quantify the change in adverse health effects due to a change in air pollution exposure. The most common functional forms the log-linear and logistic, with a linear model used in some cases. All three are discussed below.

Note that the log-linear and logistic generally produce comparable results, so the fact that some health impacts are estimated with a logistic function and others with a log-linear function is not a cause for concern. Indeed, in some circumstances, such as for small changes in air pollution, the logistic and log-linear produce essentially the same result.

### The Linear Model

A linear model between the adverse health effect,  $y$ , and the pollutant concentration,  $x$ , is of the form

$$y = \alpha + \beta \cdot x$$

A linear model includes the factors that are believed to affect the incidence of the health effect, of which the pollutant would be one. So, the variable “ $\alpha$ ” in the linear function consists of all the other independent variables in the regression, typically evaluated at their mean values, times their respective coefficients.

The function describing the relationship between a change in  $x$  and the corresponding change in incidence (rate) of the health effect from the baseline level ( $y_b$ ) to the post-control level ( $y_c$ ) is then:

$$\Delta y = y_b - y_c = \beta \cdot (x_b - x_c) = \beta \cdot \Delta x$$

If  $y$  denotes an incidence rate, then  $\Delta y$  denotes the change in the incidence rate. If  $y$  denotes an incidence count, then the  $\beta$  is first divided the baseline study population to generate an incidence rate.  $\Delta x$  is the difference between the baseline level of the pollutant concentration and the control level of the pollutant concentration:  $x_b - x_c$ . (Note that typically a control strategy is intended to decrease the pollutant levels, so we expect  $\Delta x$  to be positive.) The expected number of cases avoided would then be calculated by multiplying  $\Delta y$  by the relevant population:

$$\text{Cases Avoided} = \beta \cdot \Delta x \cdot \text{population}$$

The coefficient,  $\beta$ , and standard error of  $\beta$  ( $\sigma_\beta$ ) are reported directly in studies presenting results from linear regression models.

## The Log-linear Model

The most commonly used functional form for criteria air pollutant concentration-response functions is the log-linear model. It defines the relationship between  $x$  and  $y$  to be of the form:

$$y = B \cdot \exp(\beta \cdot x)$$

or, equivalently,

$$\ln(y) = \alpha + \beta \cdot x,$$

where the parameter  $B$  is the incidence (rate) corresponding to the zero pollutant concentration ( $x = 0$ ); the coefficient  $\beta$  is the effect of pollutant  $x$  on the natural logarithm of the incidence (rate)  $y - \ln(y)$ ; and  $\alpha = \ln(B)$ .<sup>24</sup>

### Estimating Avoided Cases

The relationship between  $\Delta x$  and  $\Delta y$  is:

$$\Delta y = y_b - y_c = B(\exp(\beta x_b) - \exp(\beta x_c))$$

This may be rewritten as:

$$\Delta y = y_b \cdot \left(1 - \frac{1}{\exp(\beta \cdot \Delta x)}\right)$$

where  $y_b$  is the baseline incidence (rate) of the health effect – i.e., the incidence (rate) before the change in  $x$ . If  $y$  is incidence rate rather than incidence count, then the change in incidence rate,  $\Delta y$ , must be multiplied by the relevant population to get the expected number of cases avoided. For example, if  $y$  denotes the annual number of cases of the adverse health effect per 100,000 population then the expected number of cases avoided is calculated as:

$$\text{Cases Avoided} = \frac{y_b}{100,000} \cdot \text{population} \cdot \left(1 - \frac{1}{\exp(\beta \cdot \Delta x)}\right)$$

### Estimating the Coefficient ( $\beta$ )

Epidemiological studies that estimate log-linear concentration-response functions often report a relative risk for a specific  $\Delta x$ , rather than the coefficient,  $\beta$ , in the function itself. The relative risk ( $RR$ ) is simply the ratio of two risks corresponding to two levels of pollutant concentration –

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<sup>24</sup> Other covariates besides pollution clearly affect mortality. The parameter  $B$  might be thought of as containing these other covariates, for example, evaluated at their means. That is,  $B = B_0 \exp\{\beta_1 x_1 + \dots + \beta_n x_n\}$ , where  $B_0$  is the incidence of  $y$  when all covariates in the model are zero, and  $x_1, \dots, x_n$  are the other covariates evaluated at their mean values. The parameter  $B$  drops out of the model, however, when changes in  $y$  are calculated, and is therefore not important.

the “high” risk  $y_{high}$  (corresponding to the higher pollutant level,  $x = x_{high}$ ) and the lower risk  $y_{low}$  (corresponding to the lower pollutant level,  $x = x_{low}$ ):

$$RR = \frac{y_{high}}{y_{low}}$$

Using the original log-linear function above, it can be shown that the relative risk associated with a specific change in pollutant concentration of  $\Delta x^* = x_{high} - x_{low}$  can be written as

$$RR|_{\Delta x^*} = \frac{y_{high}}{y_{low}} = \exp(\beta \cdot \Delta x^*)$$

Taking the natural log of both sides, the coefficient in the function underlying the relative risk can be derived as:

$$\beta = \frac{\ln(RR)}{\Delta x^*}$$

Once the pollutant coefficient,  $\beta$ , has been calculated, the change in incidence (rate),  $\Delta y$ , corresponding to any change in pollutant concentration,  $\Delta x$ , can be calculated, using the relationship between  $\Delta x$  and  $\Delta y$  given above, the baseline incidence (rate) and assessment population.

There are instances when epidemiological studies report percent increase in the relative risk, rather than relative risk itself. Given a reported  $x$  percent increase in the relative risk, we back-calculate the relative risk as  $RR = 1 + x/100$ . Then we proceed to calculating  $\beta$  as described above. Note that some epidemiological studies (see, e.g., Moolgavkar [2003]) further define  $x$  to be  $\log(RR) \times 100$ . In these cases, our approach to computing the RR is an approximation, although the error introduced is small. This approximation is consistent with EPA’s PM<sub>2.5</sub> health benefits model implemented in EPA’s BenMAP-CE.

### **Estimating the Standard Error of $\beta$ ( $\sigma_\beta$ )**

The standard error of  $\beta$  ( $\sigma_\beta$ ) is not often directly reported in studies presenting results from log-linear regression models. Results are most commonly presented as a relative risk and 95% confidence interval. The 95% confidence interval is defined as follows:

$$CI_{95\%} = \exp(\beta \cdot \Delta x \pm 1.96 \cdot \sigma_\beta \Delta x)$$

Based on this equation, the standard error of  $\beta$  ( $\sigma_\beta$ ) can be estimated from the relative risk (RR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:



$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{(\ln(UL)/\Delta x - \ln(RR)/\Delta x)}{1.96} \quad \text{and} \quad \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{(\ln(RR)/\Delta x - \ln(LL)/\Delta x)}{1.96}$$

$$\sigma_{\beta} \cong \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} \quad \text{or} \quad \sigma_{\beta} \cong \frac{\beta_{high} - \beta_{low}}{3.92}$$

Some studies report only a central effect estimate and *t*-statistic. The *t*-statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient,  $\beta$ , to the standard error of  $\beta$  ( $\sigma_{\beta}$ ). The standard error of  $\beta$  ( $\sigma_{\beta}$ ) can, therefore, be estimated from the *t*-statistic as follows:

$$\sigma_{\beta} = \frac{\beta}{t}$$

## The Logistic Model

In some epidemiological studies, a logistic model is used to estimate the probability of an occurrence of an adverse health effect. Given a pollutant level,  $x$ , and a vector of other explanatory variables,  $Z$ , the logistic model assumes the probability of an occurrence is:

$$y = P(\text{occurrence} | \beta \cdot x, \alpha \cdot Z) = \frac{\exp(\beta \cdot x) \exp(\alpha \cdot Z)}{1 + \exp(\beta \cdot x) \exp(\alpha \cdot Z)},$$

where  $\beta$  is the coefficient of the pollutant concentration,  $x$ , and  $\alpha$  is a vector of coefficients of the variables in the vector  $Z$ .<sup>2</sup>

### Estimating Avoided Cases

The change in the probability of an occurrence ( $\Delta y$ ) corresponding to a change in the level of the pollutant from  $x_b$  to  $x_c$  ( $= \Delta x$ ), all other covariates held constant, may be derived from the original C-R function above:

$$\Delta y = y_b - y_c = y_b \left( 1 - \frac{1}{(1 - y_b) \cdot \exp(\beta \cdot \Delta x) + y_b} \right)$$

<sup>2</sup> Greene (1997, Chapter 19) presents models with discrete dependent variables; in particular, page 874 presents the logit model. See also Judge et al. (1985, p. 763).

Once again, to calculate the expected number of avoided cases of the adverse effect, it is necessary to multiply by the population:<sup>3</sup>

$$Cases\ Avoided = y_b \left( 1 - \frac{1}{(1 - y_b) \cdot \exp(\beta \cdot \Delta x) + y_b} \right) \cdot population$$

### **Estimating the Coefficient ( $\beta$ )**

The estimated pollutant coefficient,  $\beta$ , in the original function is typically not reported in studies that use the logistic model. Instead, the odds ratio (OR) corresponding to a specific change in  $x$  is reported.

The odds of an occurrence is defined as:

$$Odds = \frac{y}{1 - y}$$

It can be shown that:

$$Odds = \frac{y}{1 - y} = \exp(\beta \cdot x) \exp(\alpha \cdot Z)$$

The odds ratio is just the ratio of the odds when the pollutant is at a specified higher level,  $x_{high}$ , to the odds when the pollutant is at a specified lower level,  $x_{low}$ :

$$OR = \frac{\exp(\beta \cdot x_{high}) \exp(\alpha \cdot Z)}{\exp(\beta \cdot x_{low}) \exp(\alpha \cdot Z)} = \frac{\exp(\beta \cdot x_{high})}{\exp(\beta \cdot x_{low})} = \exp(\beta \cdot \Delta x)$$

Often the odds ratio corresponding to a specified change in  $x$ , call it  $\Delta x^*$ , is the only measure of the effect of  $x$  reported from a study using a logistic model (just as the relative risk corresponding to a specified change in  $x$  is often the only measure of the effect of  $x$  reported from a study using a log-linear model). However, it is easy to calculate the underlying pollutant coefficient,  $\beta$ , from the odds ratio as follows:

$$OR|_{\Delta x^*} = \exp(\beta \cdot \Delta x^*) \rightarrow \ln(OR) = \beta \cdot \Delta x^* \rightarrow \beta = \frac{\ln(OR)}{\Delta x^*}$$

Given the pollutant coefficient,  $\beta$ , and the baseline probability of occurrence,  $y_b$ , the change in the probability,  $\Delta y$ , associated with any change in pollutant concentration,  $\Delta x$ , can be derived using the equation for  $\Delta y$  above. The expected number of avoided cases of the adverse effect is then obtained by multiplying by the population.

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<sup>3</sup> Note that because  $\Delta y$  here is a change in probability of occurrence (rather than a change in the rate per 100,000 population), it is necessary to multiply by the population rather than by the population/100,000.

### **Estimating the Standard Error of $\beta$ ( $\sigma_\beta$ )**

The standard error of  $\beta$  ( $\sigma_\beta$ ) is not often directly reported in studies presenting results from logistic regression models. Results are most commonly presented as an odds ratio and 95% confidence interval. The 95% confidence interval is defined as follows:

$$CI_{95\%} = \exp(\beta \cdot \Delta x \pm 1.96 \cdot \sigma_\beta \Delta x)$$

Based on this equation, the standard error of  $\beta$  ( $\sigma_\beta$ ) can be estimated from the odds ratio (OR), upper limit of the 95% confidence interval (UL), and lower limit of the 95% confidence interval (LL), as follows:

$$\sigma_{\beta,high} = \frac{\beta_{high} - \beta}{1.96} = \frac{(\ln(UL)/\Delta x - \ln(OR)/\Delta x)}{1.96} \quad \text{and} \quad \sigma_{\beta,low} = \frac{\beta - \beta_{low}}{1.96} = \frac{(\ln(OR)/\Delta x - \ln(LL)/\Delta x)}{1.96}$$

$$\sigma_\beta \cong \frac{\sigma_{\beta,high} + \sigma_{\beta,low}}{2} \quad \text{or} \quad \sigma_\beta \cong \frac{\beta_{high} - \beta_{low}}{3.92}$$

Some studies report only a central effect estimate and  $t$ -statistic. The  $t$ -statistic describes the strength of the observed pollutant-health effect association. It is defined as the ratio of the coefficient,  $\beta$ , to the standard error of  $\beta$  ( $\sigma_\beta$ ). The standard error of  $\beta$  ( $\sigma_\beta$ ) can, therefore, be estimated from the  $t$ -statistic as follows:

$$\sigma_\beta = \frac{\beta}{t}$$

## Appendix C: Health Impact Functions

A reduction in ambient PM<sub>2.5</sub> levels is associated with reductions in a number of adverse health effects, or “endpoints.” This Appendix discusses the calculation of avoided adverse health effects. The health impact functions in the COBRA model were prepared by Abt Associates in close consultation with EPA and rely on an up-to-date assessment of the published scientific literature to ascertain the relationship between particulate matter and adverse human health effects. We evaluated studies using a variety of selection criteria, including: study location and design, the characteristics of the study population, and whether the study was peer-reviewed (Exhibit C-1).

**Exhibit C-1. Summary of Considerations Used in Selecting Studies**

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies because they control for important individual-level confounding variables that cannot be controlled for in cross-sectional studies.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time.
Study size	Studies examining a relatively large sample are preferred because they generally have more statistical power to detect small magnitude effects. A large sample can be obtained in several ways, either through a large population, or through repeated observations on a smaller population, e.g. through a symptom diary recorded for a panel of asthmatic children.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, population behavior and life style.
Measure of PM	For this analysis, C-R functions based on PM <sub>2.5</sub> are preferred to those based on PM <sub>10</sub> (particulate matter less than 10 microns in aerodynamic diameter) because reductions in emissions from diesel engines are expected to reduce fine particles and not have much impact on coarse particles.
Economically valuable health effects	Some health effects, such as changes in forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are therefore not quantified in this analysis.
Non-overlapping endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category "hospital admissions" includes emergency room visits.

### **Model Selection**

In many epidemiological studies of air pollution and health, researchers estimate and present numerous single pollutant and multi-pollutant models for the same pollutant and health endpoint.

These models may differ from each other in a number of characteristics, including: the functional form of the model, the covariates included in the model, the pollutant exposure metric, the lag structure, and the study population.

For the purposes of estimating health benefits associated with pollutant changes, it is neither realistic nor advantageous to include every model presented in each study. However, it is important that a relatively objective process be used to select from among models. Described below are the criteria that were used as guidance in the selection of a particular model from among several models presented in a study. It is not possible in all cases to select a model using a completely objective and mechanical process. In many cases, professional judgment and an understanding of the study context are necessary as well to select the most appropriate models. Exhibit C-2 summarizes the selection criteria that we used.

**Exhibit C-2. Description of Selection Criteria**

<b>Selection Criteria</b>	<b>Description</b>
Goodness-of-fit statistics	If an appropriate measure of goodness of fit (i.e., how well the model fit the data) is reported for each of several models in a study, then this measure may be used as the basis on which to select a model.
Best captures distributed lag	Select the model that appears to best capture a distributed lag effect, as described below. If multiple single-lag models and/or moving average models are specified, select the model with the largest effect estimate, all else equal.
Best set of control variables	Select the model which includes temporal variables (i.e. season, weather patterns, day of the week) and other known non-pollutant confounders, all else equal. Select the model which uses the most sophisticated methods of capturing the relationship between these variables and the dependent variable (e.g., affords the most flexibility in fitting possible nonlinear trends).
Useful for health effects modeling	The model must be in a form that is useful for health effects modeling (e.g., the pollutant variable should be a continuous variable rather than a categorical variable).
Sample size	Select the model estimated with the larger sample size, all else equal.

### ***Distributed Lag Effect***

The question of lags and the problems of correctly specifying the lag structure in a model has been discussed extensively (U.S. EPA, 2002, Section 8.4.4). In many time-series studies, after the basic model is fit (before considering the pollutant of interest), several different lags are typically fit in separate single-lag models and the most significant lag is chosen. The 2002 draft PM<sub>2.5</sub> CD notes that “while this practice may bias the chance of finding a significant association, without a firm biological reason to establish a fixed pre-determined lag, it appears reasonable” (U.S. EPA, 2002, p. 8-237).

There is recent evidence (Schwartz, 2000) that the relationship between PM<sub>2.5</sub> and health effects may best be described by a distributed lag (i.e., the incidence of the health effect on day n is influenced by PM<sub>2.5</sub> concentrations on day n, day n-1, day n-2 and so on). If this is the case, a model that includes only a single lag (e.g., a 0-day lag or a 1-day lag) is likely to understate the

total impact of PM<sub>2.5</sub>. The 2002 draft PM<sub>2.5</sub> CD makes this point, noting that “if one chooses the most significant single lag day only, and if more than one lag day shows positive (significant or otherwise) associations with mortality, then reporting a RR [relative risk] for only one lag would also underestimate the pollution effects” (U.S. EPA, 2002, p. 8-241). The same may hold true for other pollutants that have been associated with various health effects.

Several studies report similar models with different lag structures. For example, Moolgavkar (2000a) studied the relationship between air pollution and respiratory hospital admissions in three U.S. metropolitan areas. The author reports models with PM<sub>2.5</sub> lagged from zero to five days. Since the lagging of PM<sub>2.5</sub> was the only difference in the models and the relationship is probably best described using a distributed lag model, any of single-lag effect estimates are likely to underestimate the full effect. Therefore, we selected the model with the largest effect estimate.

### **Pooling**

There is often more than one study that has estimated a health impact function for a given pollutant-health endpoint combination. Each study provides an estimate of the pollutant coefficient,  $\beta$ , along with a measure of the uncertainty of the estimate. Because uncertainty decreases as sample size increases, combining data sets is expected to yield more reliable estimates of  $\beta$ , and therefore more reliable estimates of the incidence change predicted using  $\beta$ . Combining data from several comparable studies in order to analyze them together is often referred to as meta-analysis.

For a number of reasons, including data confidentiality, it is often impractical or impossible to combine the original data sets. Combining the results of studies in order to produce better estimates of  $\beta$  provides a second-best but still valuable way to synthesize information. This is referred to as pooling. Pooling  $\beta$ 's requires that all of the studies contributing estimates of  $\beta$  use the same functional form for the health impact function. That is, the  $\beta$ 's must be measuring the same thing.

To be consistent with the recent EPA benefits analyses, COBRA uses a random-/ fixed- effects pooling procedure (see U.S. EPA, 2009, p. 5-18), which is a method for weighting estimates involving using their variances. Variance takes into account both the consistency of data and the sample size used to obtain the estimate, two key factors that influence the reliability of results. The method is based on DerSimonian and Laird (1986).

### **Fixed Effect Weights**

The fixed effects model assumes that there is a single true concentration-response relationship and therefore a single true value for the parameter  $\beta$  that applies everywhere. Differences among  $\beta$ 's reported by different studies are therefore simply the result of sampling error. That is, each reported  $\beta$  is an estimate of the same underlying parameter. The certainty of an estimate is reflected in its variance (the larger the variance, the less certain the estimate). Fixed effects pooling therefore weights each estimate under consideration in proportion to the inverse of its variance:

$$\beta_{fe} = \frac{\sum_{n=1}^N \frac{\beta_n}{v_n}}{\sum_{n=1}^N \frac{1}{v_n}}$$

Where

$N$  – number of studies;

$\beta_n$  – estimate provided by study  $n$ ;

$v_n$  – variance of the estimate provided by study  $n$ ;

$\beta_{fe}$  – pooled fixed effects estimate.

### Random- / Fixed- Effect Weights

An alternative to the fixed effects model is the random effects model, which allows the possibility that the estimates  $\beta_n$  from the different studies may in fact be estimates of different parameters, rather than just different estimates of a single underlying parameter. In studies of the effects of PM<sub>2.5</sub> on hospitalizations for COPD, for example, if the composition of PM<sub>2.5</sub> varies among study locations the underlying relationship between the frequency of hospitalizations for COPD and PM<sub>2.5</sub> may be different from one study location to another. This would violate the assumption of the fixed effects model.

It is possible to test whether it is appropriate to base the pooling on the random effects model (vs. the fixed effects model). A test statistic,  $Q_w$ , the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model  $\beta_{fe}$ , is calculated as:

$$Q_w = \sum_{n=1}^N \frac{(\beta_n - \beta_{fe})^2}{v_n}$$

Under the null hypothesis that there is a single underlying parameter,  $\beta$ , of which all the  $\beta_n$ 's are estimates,  $Q_w$  has a chi-squared distribution with  $N-1$  degrees of freedom. (Recall that  $N$  is the number of studies in the meta-analysis.) If  $Q_w$  is greater than the critical value corresponding to the desired confidence level, the null hypothesis is rejected. That is, in this case the evidence does not support the fixed effects model, and the random effects model is assumed, allowing the possibility that each study is estimating a different  $\beta$ . We use a five percent one-tailed test.

The random effect model-based pooling must take into account not only the within-study variances (used in a meta-analysis based on the fixed effects model) but the between-study variance as well. The between-study variance,  $\eta^2$ , is given by:

$$\eta^2 = \frac{Q_w - (N-1)}{\sum_{n=1}^N \frac{1}{v_n} - \frac{\sum_{n=1}^N \frac{1}{v_n^2}}{\sum_{n=1}^N \frac{1}{v_n}}}$$

(i.e., if  $Q_w < N-1$ ), then  $\eta^2$  is a negative number, and it is not possible to calculate a random effects estimate. In this case, however, the small value of  $Q_w$  would presumably have led to accepting the null hypothesis described above, and the meta-analysis would be based on the fixed effects model. The remaining discussion therefore assumes that  $\eta^2$  is positive.

Given a value for  $\eta^2$ , the random effects estimate is calculated in almost the same way as the fixed effects estimate. However, the pooled estimate now incorporates both the within-study variance ( $v_n$ ) and the between-study variance ( $\eta^2$ ):

$$\beta_{re} = \frac{\sum_{n=1}^N \beta_n}{\sum_{n=1}^N \frac{1}{\eta^2 + v_n}}$$

Where

$N$  – number of studies;

$\beta_n$  – estimate provided by study  $n$ ;

$v_n$  – variance of the estimate provided by study  $n$ ;

$\eta^2$  – within-study variance;

$\beta_{re}$  – pooled random effects estimate.

The weighting scheme used in a pooling based on the random effects model is basically the same as that used if a fixed effects model is assumed, but the variances used in the calculations are different. This is because a fixed effects model assumes that the variability among the estimates from different studies is due only to sampling error (i.e., each study is thought of as representing just another sample from the same underlying population), while the random effects model assumes that there is not only sampling error associated with each study, but that there is also between-study variability – each study is estimating a different underlying  $\beta$ . Therefore, the sum of the within-study variance and the between-study variance yields an overall variance estimate.

### **Thresholds**

Health impact functions have been developed with and without explicit thresholds. A threshold means that air pollution levels below the specified threshold have no adverse health effects. In some prior regulatory impact assessments (e.g., U.S. EPA, 2006) assumed a threshold of 10  $\mu\text{g}/\text{m}^3$  for  $\text{PM}_{2.5}$ . However, EPA's most current understanding of the scientific literature is that there is no threshold in the relationship between  $\text{PM}_{2.5}$  and adverse health impacts. In its recent analysis of proposed  $\text{NO}_2$  national ambient air quality standards, U.S. EPA (2009) used a no-threshold model to calculate  $\text{PM}_{2.5}$  co-benefits down to the lowest modeled  $\text{PM}_{2.5}$  air quality levels.

Following EPA's updated methodology, we also assume there is no threshold for modeling  $\text{PM}_{2.5}$ -related health effects. This is supported by the National Research Council (2002) in its review of methods for estimating the public health benefits of air pollution regulations. They



concluded that there is no evidence for any departure from linearity in the observed range of exposure to PM<sub>10</sub> or PM<sub>2.5</sub>, nor is there any indication of a threshold. They cite the weight of evidence available from both short- and long-term exposure models and the similar effects found in cities with low and high ambient concentrations of PM<sub>2.5</sub>. More recently, Schwartz et al (2008) reached the same conclusion, finding a linear relationship between PM<sub>2.5</sub> and premature mortality with no evidence of a threshold.

In addition, U.S. EPA completed an “expert elicitation” analysis in which it elicited opinions from 12 experts (in epidemiology, toxicology, and medicine) on the nature of this relationship (see: Industrial Economics Incorporated (IEC), 2006). The experts were asked how likely they thought it is that the relationship between PM<sub>2.5</sub> and mortality is causal, and if it is causal, what is the functional form of the C-R relationship, including whether there is a threshold. Eleven of the twelve experts thought that, although each individual may have a threshold, there is insufficient empirical evidence for a threshold for the population, which is the entity of interest in a C-R function. Only one expert did include the possibility of a population threshold, assigning a probability of 50 percent to there being a threshold and, if there is a threshold, an 80 percent chance that it is less than or equal to 5 µg/m<sup>3</sup> (which is below the level of PM<sub>2.5</sub> observed in epidemiological studies), and a 20 percent chance that it is between 5 and 10 µg/m<sup>3</sup>.

## Summary of Health Impact Functions Used in COBRA

In this Appendix, we present the health impact functions used to estimate PM<sub>2.5</sub>-related adverse health effects. Exhibit C-3 summarizes the epidemiological studies in COBRA used to estimate adverse health impacts of PM<sub>2.5</sub>. Each sub-section has an exhibit with a brief description of the health impact function and the underlying parameters. Following each exhibit, we present a brief summary of each study and any information that is unique to that study.

**Exhibit C-3. Epidemiological Studies Used to Estimate Adverse Health Impacts of PM<sub>2.5</sub>**

Endpoint	Author	Age
Mortality, All Cause	Krewski et al. (2009)	30-99
Mortality, All Cause	Lepeule et al. (2012)	25-99
Mortality, All Cause	Woodruff et al. (1997)	Infant
Acute Myocardial Infarction, Nonfatal	Peters et al. (2001)	18-99
Acute Myocardial Infarction, Nonfatal	Pope et al. (2006)	18-99
Acute Myocardial Infarction, Nonfatal	Sullivan et al. (2005)	18-99
Acute Myocardial Infarction, Nonfatal	Zanobetti and Schwartz (2006)	18-99
Acute Myocardial Infarction, Nonfatal	Zanobetti et al. (2009)	18-99
HA, All Cardiovascular (less Myocardial Infarctions)	Bell et al. (2008)	65-99
HA, All Cardiovascular (less Myocardial Infarctions)	Moolgavkar (2000b)	18-64
HA, All Cardiovascular (less Myocardial Infarctions)	Peng et al. (2008)	65-99
HA, All Cardiovascular (less Myocardial Infarctions)	Peng et al. (2009)	65-99
HA, All Cardiovascular (less Myocardial Infarctions)	Zanobetti et al. (2009)	65-99
HA, All Respiratory	Zanobetti et al. (2009)	65-99
HA, All Respiratory	Kloog et al. (2012)	65-99
HA, Asthma	Babin et al. (2007)	0-17
HA, Asthma	Sheppard (2003)	0-17
HA, Chronic Lung Disease	Moolgavkar (2000a)	18-64
Emergency Room Visits, Asthma	Mar et al. (2010)	0-99
Emergency Room Visits, Asthma	Slaughter et al. (2005)	0-99
Emergency Room Visits, Asthma	Glad et al. (2012)	0-99
Acute Bronchitis	Dockery et al. (1996)	8-12
Asthma Exacerbation, Cough	Mar et al. (2004)	6-18
Asthma Exacerbation, Cough	Ostro et al. (2001)	6-18
Asthma Exacerbation, Shortness of Breath	Mar et al. (2004)	6-18
Asthma Exacerbation, Shortness of Breath	Ostro et al. (2001)	6-18
Asthma Exacerbation, Wheeze	Ostro et al. (2001)	6-18
Minor Restricted Activity Days	Ostro and Rothschild (1989)	18-64
Lower Respiratory Symptoms	Schwartz and Neas (2000)	7-14
Upper Respiratory Symptoms	Pope et al. (1991)	9-11
Work Loss Days	Ostro (1987)	18-64

Note that Appendix B mathematically derives the standard types of health impact functions that we encountered in the epidemiological literature, such as, log-linear, logistic and linear, so we simply note here the type of functional form. Appendix D presents a description of the sources for the incidence and prevalence data used in these health impact functions.

## Mortality

Health researchers have consistently linked air pollution, especially PM<sub>2.5</sub>, with excess mortality. Although a number of uncertainties remain to be addressed, a substantial body of published scientific literature recognizes a correlation between elevated PM<sub>2.5</sub> concentrations and increased mortality rates. Based on the scientific evidence, EPA’s Integrated Science Assessment determined a causal relationship between PM<sub>2.5</sub> and premature mortality (<http://www.epa.gov/ncea/isa/>).

Both long- and short-term exposures to ambient levels of particulate matter air pollution have been associated with increased risk of premature mortality. It is clearly an important health endpoint because of the size of the mortality risk estimates, the serious nature of the effect itself, and the high monetary value ascribed to avoiding mortality risk. Because of the importance of this endpoint and the considerable uncertainty among economists and policymakers as to the appropriate way to estimate PM-related mortality risks, this section discusses some of the issues surrounding the estimation of premature mortality associated with PM<sub>2.5</sub>.

Particulate matter has been linked with premature mortality in adults in multiple studies throughout the world (Jerrett et al., 2005; Katsouyanni et al., 2001; Laden et al., 2006; Pope et al., 2002; Samet, Dominici, Curriero, Coursac, & Zeger, 2000) as well as infants (Bobak & Leon, 1999; Conceicao, Miraglia, Kishi, Saldiva, & Singer, 2001; Loomis, Castillejos, Gold, McDonnell, & Borja-Aburto, 1999; Woodruff, Darrow, & Parker, 2008; Woodruff et al., 1997). To estimate premature mortality in adults, we use an epidemiological analysis of the American Cancer Society cohort by Krewski et al. (2009) and analysis of the Six-City cohort by Lepeule et al. (2012). To estimate premature mortality in infants, we used a study by Woodruff et al. (1997).

**Exhibit C-4. Health Impact Functions for Particulate Matter and All-Cause Mortality**

Author	Year	Location	Age	Metric	Beta	Std Err	Functional Form
Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.005827	0.000963	Log-linear
Lepeule et al.	2012	6 Eastern cities	25-99	Annual	0.013103	0.003347	Log-linear
Woodruff et al.	1997	86 cities	0-0	Annual	0.003922	0.001221	Logistic

Note that COBRA does not pool Krewski et al. (2009) and Lepeule et al. (2012) to estimate premature mortality in adults. In recent analysis of proposed NO<sub>2</sub> national ambient air quality standards, U.S. EPA (2009) used Pope et al. (2002)<sup>25</sup> and Laden et al. (2006) to estimate the PM<sub>2.5</sub> mortality-related co-benefits and presented the results separately for each study:

“These are logical choices for anchor points in our presentation because, while both studies are well designed and peer reviewed, there are strengths and weaknesses inherent

<sup>25</sup> Krewski et al. (2009) is an extended and updated analysis of Pope et al. (2002).

in each, which we believe argues for using both studies to generate benefits estimates. Previously, EPA had calculated benefits based on these two empirical studies, but derived the range of benefits, including the minimum and maximum results, from an expert elicitation of the relationship between exposure to PM<sub>2.5</sub> and premature mortality (Roman et al., 2008). Within this assessment, we include the benefits estimates derived from the concentration-response function provided by each of the twelve experts to better characterize the uncertainty in the concentration-response function for mortality and the degree of variability in the expert responses. Because the experts used these cohort studies to inform their concentration-response functions, benefits estimates using these functions generally fall between results using these epidemiology studies (see Figure 5.9). In general, the expert elicitation results support the conclusion that the benefits of PM<sub>2.5</sub> control are very likely to be substantial.” p. 5-25.

### ***Mortality, All Cause (Krewski et al., 2009)***

This cohort study consists of approximately 360,000 participants residing in areas of the country that have adequate monitoring information on levels of PM<sub>2.5</sub> for 1980 and about 500,000 participants in areas with adequate information for 2000. The causes of death that were analyzed included all causes, cardiopulmonary disease (CPD), ischemic heart disease (IHD), lung cancer, and all remaining causes. Data for 44 personal, individual-level covariates, based on participants' answers to a 1982 enrollment questionnaire, were also used for the analyses. The authors also collected data for seven ecologic (neighborhood-level) covariates, each of which represents local factors known or suspected to influence mortality, such as poverty level, level of education, and unemployment (at both zip code and city levels). Long-term average exposure variables were constructed for PM<sub>2.5</sub> from monitoring data for two periods: 1979-1983 and 1999-2000. Similar variables were constructed for long-term exposure to other pollutants of interest from single-year (1980) averages, including total suspended particles, ozone, nitrogen dioxide, and sulfur dioxide. Exposure was averaged for all monitors within a metropolitan statistical area (MSA) and assigned to participants according to their Zip Code area (ZCA) of residence.

The authors chose the standard Cox proportional-hazards model (and a variation to allow for random effects) to calculate hazard ratios for various cause-of-death categories associated with the levels of air pollution exposure in the cohort. They extended the random effects Cox model to accommodate two levels of information for clustering and for ecologic covariates. Three main analyses were conducted: a Nationwide Analysis, Intra-Urban Analyses in the New York City (NYC) and Los Angeles (LA) regions, and an analysis designed to investigate whether critical time windows of exposure to pollutants might have affected mortality in the cohort. Using a multi-pollutant model (O<sub>3</sub>, SO<sub>4</sub>, SO<sub>2</sub>, TSP, and PM<sub>2.5</sub>), the authors reported a relative risk (1.06) for all-cause mortality and the corresponding 95% confidence interval (95% CI: 1.04-1.08) for a 10 µg/m<sup>3</sup> increase in the average of PM<sub>2.5</sub> exposure level for 1999-2000 (Krewski et al., 2009, Commentary Table 4). The results were adjusted for the 44 individual-level covariates and the 7 ecologic covariates at the MSA & DIFF levels.

**Functional Form:** Log-linear

**Coefficient:** 0.005827

**Standard Error:** 0.000963

**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 (Lepeule et al., 2012)***

Lepeule et al. (2012) is an extended and updated analysis of Laden et al. (2006). The authors performed an extended mortality follow-up from 1979-2009 using data from the Harvard Six Cities adult cohort study. They used annual city-specific PM<sub>2.5</sub> concentrations and assigned for each participant until death or censoring. The authors replicated the previously applied Cox regression (as used in Laden et al., 2006), and examined different time lags, the shape of the concentration–response relationship using penalized splines, and changes in the slope of the relation over time. Then they conducted Poisson survival analysis with time-varying effects for smoking, sex, and education. The authors found a significant increase in the overall mean mortality associated with a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>.

The coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.07-1.22) associated with a 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub> (Lepeule et al., 2012, Table 2).

**Functional Form:** Log-linear

**Coefficient:** 0.013103

**Standard Error:** 0.003347

**Incidence Rate:** county-specific annual all-cause mortality rate per person ages 25 and older

**Population:** population of ages 25 and older

### ***Infant Mortality (Woodruff et al., 1997)***

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. (1997) found a significant link between PM<sub>10</sub> 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<sub>10</sub> exposure was significant for all-cause mortality. PM<sub>10</sub> was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

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  $\mu\text{g}/\text{m}^3$  change in PM<sub>10</sub> (Woodruff et al., 1997, Table 3).

**Functional Form:** Logistic

**Coefficient:** 0.003922

**Standard Error:** 0.001221

**Incidence Rate:** county-specific annual post-neonatal<sup>26</sup> infant deaths per infant under the age of one

**Population:** population of infants under one year old

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<sup>26</sup> Post-neonatal refers to infants that are 28 days to 364 days old.

## Non-Fatal Heart Attack

Non-fatal heart attacks have been linked with short-term exposures to PM<sub>2.5</sub> in the U.S. (Peters et al., 2001) and other countries (Poloniecki, Atkinson, de Leon, & Anderson, 1997).<sup>27</sup> We used the C-R functions reported in five studies as shown in Exhibit C-5.

The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the U.S. These studies provide a weight of evidence for this type of effect. Several epidemiological studies (Gold et al., 2000; Liao et al., 1999; Magari et al., 2001) have shown that heart rate variability (an indicator of how much the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM<sub>2.5</sub> levels. Lack of heart rate variability is a risk factor for heart attacks and other coronary heart diseases (Dekker et al., 2000; Liao et al., 1997; Tsuji et al., 1996). As such, the reduction in heart rate variability due to PM<sub>2.5</sub> is consistent with an increased risk of heart attacks.

**Exhibit C-5. Health Impact Functions for Particulate Matter and Non-fatal Heart Attack**

Author	Year	Location	Age	Metric	Beta	Std Error	Functional Form
Peters et al.	2001	Boston, MA	18-99	24-hr avg	0.024121	0.009285	Logistic
Pope et al.	2006	Greater Salt Lake City, Utah	All	24-hr avg	0.00481	0.001992	Logistic
Sullivan et al.	2005	King County, Washington	All	24-hr avg	0.001980	0.002241	Logistic
Zanobetti and Schwartz	2006	Greater Boston area (Middlesex, Norfolk, Suffolk Counties)	All	24-hr avg	0.005300	0.002213	Logistic
Zanobetti et al.	2009	26 U.S. Communities	All	24-hr avg	0.00225	0.000592	Log-linear

COBRA reports two sets of incidence results: (1) incidence results based on C-R function from Peters et al. (2001); (2) pooled incidence based on other four studies using random/fixed effects pooling method.

### ***Acute Myocardial Infarction (Heart Attacks), Nonfatal (Peters et al., 2001)***

Peters et al. (2001) 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<sub>10</sub>, PM<sub>10-2.5</sub>, PM<sub>2.5</sub>, “black carbon”, O<sub>3</sub>, CO, NO<sub>2</sub>, and SO<sub>2</sub> 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<sub>2.5</sub> 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<sub>2.5</sub> concentrations before onset. Significant associations were

<sup>27</sup> Non-fatal heart attacks are considered chronic illness although they are related to short-term exposure because the impact is long-lasting and this is reflected in its valuation (discussed in Appendix F).

observed for PM<sub>10</sub> as well. None of the other particle measures or gaseous pollutants was 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, Table 10).

The coefficient and standard error are calculated from an odds ratio of 1.62 (95% CI 1.13-2.34) for a 20 µg/m<sup>3</sup> increase in twenty-four hour average PM<sub>2.5</sub> (Peters et al., 2001, Table 4, p. 2813).

**Functional Form:** Logistic

**Coefficient:** 0.024121

**Standard Error:** 0.009285

**Incidence Rate:** We use the county-specific daily AMI hospitalization rate (ICD-9 code 410) for the population of individuals aged 18 years and older as the estimate for the incidence rate of nonfatal heart attack, assuming all heart attacks that are not instantly fatal will result in a hospitalization. We did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

**Population:** population of ages 18 and older

**Adjustment:** As some fraction of the admitted individuals die in the hospital, we apply a survival rate of 93% in calculating the avoided cases of AMI in order to avoid double counting (once in the calculation of AMI cases and once in the calculation of PM-related mortality).

### ***Acute Myocardial Infarction (Heart Attacks), Nonfatal (Pope et al., 2006)***

Pope et al. (2006) evaluated the association between short-term exposure to PM<sub>2.5</sub> and acute ischemic heart disease events, including acute nonfatal myocardial infarction, all acute coronary events, and subsequent myocardial infarctions in individuals living in greater Salt Lake City, Utah. In a case-crossover study, these ischemic events were assessed in relation to a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>.

Using a single-pollutant model the coefficient and standard error were estimated from the percent increase (4.81%) and 95% confidence interval (95% CI: 0.98-8.79) for a 10 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> (Pope et al., 2006, Table 3).

**Functional Form:** Logistic

**Coefficient:** 0.00481

**Standard Error:** 0.001992

**Incidence Rate:** We use the county-specific daily AMI hospitalization rate (ICD-9 code 410) for the population of individuals aged 18 years and older as the estimate for the incidence rate of nonfatal heart attack, assuming all heart attacks that are not instantly fatal will result in a hospitalization. We did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

**Population:** The study examined population of all ages. We apply the results to people ages 18 and older. We apply the results to people of ages 18 and older. Since the vast majority of AMI occur among population 65-99, over-counting may not be an issue when applying the risk coefficient to 18+.

**Adjustment:** As some fraction of the admitted individuals die in the hospital, we apply a survival rate of 93% in calculating the avoided cases of AMI in order to avoid double counting (once in the calculation of AMI cases and once in the calculation of PM-related mortality).

### ***Acute Myocardial Infarction (Heart Attacks), Nonfatal (Sullivan et al., 2005)***

Sullivan et al. (2005) studied the relationship between onset time of acute myocardial infarction and the preceding hourly PM<sub>2.5</sub> concentrations in 5,793 confirmed cases of myocardial infarction through King County, Washington. In this case-crossover study from 1988-1994, air pollution exposure levels averaged 1 hour, 2 hours, 4 hours, and 24 hours before onset of myocardial infarction were compared to a set of time-stratified referent exposures from the same day of the week in the month of the case event. The authors estimated that an associated risk of 1.01 (95% CI: 0.98-1.05) for myocardial infarction onset could be attributed to a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> the hour before MI onset. No increased risk was found in all cases with preexisting cardiac diseases with an odds ratio of 1.05 (95% CI: 0.95-1.16). Furthermore, stratification for hypertension, diabetes, and smoking status did not modify the association between PM<sub>2.5</sub> and onset of myocardial infarction.

Using a single-pollutant model, the coefficient and standard error were estimated from the odds ratio (1.02) and 95% confidence interval (95% CI: 0.98-1.07) for a 10 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> lagged 1 day (Sullivan et al., 2005, Table 3).

**Functional Form:** Logistic

**Coefficient:** 0.001980

**Standard Error:** 0.002241

**Incidence Rate:** We use the county-specific daily AMI hospitalization rate (ICD-9 code 410) for the population of individuals aged 18 years and older as the estimate for the incidence rate of nonfatal heart attack, assuming all heart attacks that are not instantly fatal will result in a hospitalization. We did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

**Population:** The study examined population of all ages. We apply the results to people ages 18 and older. We apply the results to people of ages 18 and older. Since the vast majority of AMI occur among population 65-99, over-counting may not be an issue when applying the risk coefficient to 18+.



**Adjustment:** As some fraction of the admitted individuals die in the hospital, we apply a survival rate of 93% in calculating the avoided cases of AMI in order to avoid double counting (once in the calculation of AMI cases and once in the calculation of PM-related mortality).

### ***Acute Myocardial Infarction (Heart Attacks), Nonfatal (Zanobetti & Schwartz, 2006)<sup>28</sup>***

Zanobetti and Schwartz (2006) analyzed hospital admissions through emergency department for myocardial infarction (ICD-9 code 410) and pneumonia (ICD-9 codes 480-487) for associations with fine particulate air pollution, ozone, black carbon, nitrogen dioxide, PM<sub>2.5</sub> not from traffic, and CO in the greater Boston area from 1995-1999. The authors used a case-crossover analysis with control days matched on temperature. Significant associations were detected for NO<sub>2</sub> with a 12.7% increase (95% CI: 5.8-18.0), PM<sub>2.5</sub> with an 8.6% increase (95% CI: 1.2-15.4), and black carbon with an 8.3% increase (95% CI: 0.2-15.8) in emergency myocardial infarction hospitalizations. Similarly, significant associations were identified for PM<sub>2.5</sub> with a 6.5% increase (95% CI: 1.1-11.4) and CO with a 5.5% increase (95% CI: 1.1-9.5) in pneumonia hospitalizations.

Using a single-pollutant model, the coefficient and standard error are estimated from the percent change in risk (8.65%) and 95% confidence interval (95% CI: 1.22-15.38%) for a 16.32 ug/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> for an average of the 0- and 1-day lag (Zanobetti & Schwartz, 2006, Table 4).

**Functional Form:** Logistic

**Coefficient:** 0.005300

**Standard Error:** 0.002213

**Incidence Rate:** We use the county-specific daily AMI hospitalization rate (ICD-9 code 410) for the population of individuals aged 18 years and older as the estimate for the incidence rate of nonfatal heart attack, assuming all heart attacks that are not instantly fatal will result in a hospitalization. We did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

**Population:** The study examined population of ages 65 and older. We apply the results to people of ages 18 and older. Since the vast majority of AMI occur among population 65-99, over-counting may not be an issue when applying the risk coefficient to 18+.

**Adjustment:** As some fraction of the admitted individuals die in the hospital, we apply a survival rate of 93% in calculating the avoided cases of AMI in order to avoid double counting (once in the calculation of AMI cases and once in the calculation of PM-related mortality).

### ***Acute Myocardial Infarction (Heart Attacks), Nonfatal (Zanobetti et al., 2009)***

Zanobetti et al. (2009) examined the relationship between daily PM<sub>2.5</sub> levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure,

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<sup>28</sup> The study looked at hospital admissions of AMI through ER. Under the assumption that all heart attacks will end in hospitalization, we consider the endpoint as heart attack events to be consistent with other studies.

respiratory disease and diabetes among 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific PM<sub>2.5</sub> composition while controlling for seasonal temperature as a substitute for ventilation. Overall, the authors found that PM<sub>2.5</sub> mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. For a 10 ug/m<sup>3</sup> increase in 2-day averaged PM<sub>2.5</sub>, a 1.89% (95% CI: 1.34-2.45) increase in cardiovascular disease admissions, a 2.25% (95% CI: 1.10-3.42) increase in myocardial infarction admissions, a 1.85% (95% CI: 1.19-2.51) increase in congestive heart failure admissions, a 2.74% (95% CI: 1.30-4.20) increase in diabetes admissions, and a 2.07% (95% CI: 1.20-2.95) increase in respiratory admissions were observed. The relationship between PM<sub>2.5</sub> and cardiovascular admissions was significantly modified when the mass of PM<sub>2.5</sub> was high in Br, Cr, Ni, and sodium ions, while mass high in As, Cr, Mn, organic carbon, Ni and sodium ions modified the myocardial infarction relationship and mass high in As, organic carbon, and sulfate ions modified the diabetes admission rates.

Using a single-pollutant model, the coefficient and standard error are estimated from the percent change in risk (2.25%) and 95% confidence interval (95% CI: 1.10-3.42) for a 10 ug/m<sup>3</sup> increase in 2-day averaged PM<sub>2.5</sub> (Zanobetti et al., 2009, Table 3).

**Functional Form:** Log-linear

**Coefficient:** 0.00225

**Standard Error:** 0.000592

**Incidence Rate:** We use the county-specific daily AMI hospitalization rate (ICD-9 code 410) for the population of individuals aged 18 years and older as the estimate for the incidence rate of nonfatal heart attack, assuming all heart attacks that are not instantly fatal will result in a hospitalization. We did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

**Population:** The study examined population of ages 65 and older. We apply the results to people of ages 18 and older. Since the vast majority of AMI occur among population 65-99, over-counting may not be an issue when applying the risk coefficient to 18+.

**Adjustment:** As some fraction of the admitted individuals die in the hospital, we apply a survival rate of 93% in calculating the avoided cases of AMI in order to avoid double counting (once in the calculation of AMI cases and once in the calculation of PM-related mortality).

## Hospitalizations

We include two main types of hospital admissions – respiratory (all respiratory, COPD, and asthma) and cardiovascular (all cardiovascular less myocardial infarctions).

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to PM<sub>2.5</sub> exposure. Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions.

Due to the availability of detailed hospital admission and discharge records, there is an extensive body of literature examining the relationship between hospital admissions and air pollution. Because of this, we pooled some of the hospital admission endpoints, using the results from a number of studies. Specifically, we used the following pooling procedure.

- For respiratory hospital admissions (HA): Babin et al. (2007) and Sheppard (2003) were used to estimate C-R functions for asthma hospitalizations (ICD-9 code: 493) for ages 0-17 in Washington D.C and Seattle, WA, respectively. We pooled the C-R functions from these two studies using the random/fixed effects method. We then pooled results from Zanobetti et al. (2009) and Kloog et al. (2012) using subjective weights pooling method (i.e., 0.5 for each study) to estimate incidence for all-respiratory admissions for the elderly (age 65 and up). We then aggregated incidence estimates from the following three non-overlapping categories: (1) pooled asthma hospitalization (ages 0-17) from above, (2) pooled all-respiratory admissions for the elderly (age 65 and up) from above, and (3) COPD less asthma admissions for ages 18-64 from Moolgavkar (2000a).
- For HA for cardiovascular diseases less myocardial infarctions (ICD-9 codes: 390-409, 411-429): Peng et al. (2008) and Peng et al. (2009) reported C-R functions for people age 65 years and older in 108 U.S. counties and 119 U.S. urban counties, respectively. We assigned equal weights to the estimates from these two studies (i.e., 0.5 for each study) and used the weighted average. We then assigned a weight of 0.33 to the results from each of two other studies that look at population of 65 years and older – Zanobetti et al. (2009) and Bell et al. (2008) – and pooled these results with the pooled results from Peng et al. (2008) and Peng et al. (2009).

**Exhibit C-6. Health Impact Functions for Particulate Matter and Hospital Admissions**

Endpoint	Author	Year	Location	Age	Metric	Beta	Std Error	Functional Form
All Cardiovascular (less AMI) <sup>a</sup>	Zanobetti et al.	2009	26 U.S. communities	65+	24-hr avg	0.00189	0.00028	Log-linear
All Cardiovascular (less AMI) <sup>a</sup>	Peng et al.	2008	108 U.S. counties	65+	24-hr avg	0.00071	0.00013	Log-linear
All Cardiovascular (less AMI) <sup>a</sup>	Peng et al.	2009	119 U.S. urban counties	65+	24-hr avg	0.00068	0.00021	Log-linear
All Cardiovascular (less AMI) <sup>a</sup>	Bell et al.	2008	202 US Counties	65+	24-hr avg	0.0008	0.00011	Log-linear
All Cardiovascular (less AMI) <sup>a</sup>	Moolgavkar	2000b	Los Angeles, CA	18-64	24-hr avg	0.0014	0.00034	Log-linear
HA, All Respiratory <sup>b</sup>	Zanobetti et al.	2009	26 U.S. communities	65+	24-hr avg	0.00207	0.00045	Log-linear
HA, All Respiratory <sup>b</sup>	Kloog et al.	2012	New England area (6 states)	65+	24-hr avg	0.0007	0.00096	Log-linear
HA, Asthma <sup>b</sup>	Babin et al.	2007	Washington, D.C.	0-17	24-hr avg	0.002	0.00434	Log-linear
HA, Asthma <sup>b</sup>	Sheppard	2003	Seattle, WA	0-17	24-hr avg	0.00332	0.00104	Log-linear
HA, COPD <sup>b</sup>	Moolgavkar	2000a	Los Angeles, CA	18-64	24-hr avg	0.0022	0.00073	Log-linear

<sup>a</sup> These studies were pooled to generate pooled incidence estimates for cardiovascular hospital admissions.

<sup>b</sup> These studies were pooled to generate pooled incidence estimates for respiratory hospital admissions.

### ***Hospital Admissions for All Cardiovascular (Bell et al., 2008)***

Bell et al. (2008) evaluated the association between short-term exposure to PM<sub>2.5</sub> and the risk of cardiovascular (ICD-9 codes 410-414, 26-427, 428, 429, 430-438, and 440-449) hospital admissions among Medicare enrollees ≥65 years old varied by season and geographic region in 202 U.S. counties with populations greater than 200,000 from 1999-2005. Three time-series models were used to provide three key variables: consistent PM<sub>2.5</sub> effects across the year, different PM<sub>2.5</sub> effects by season, and smoothly varying PM<sub>2.5</sub> effects throughout the year. A two-stage Bayesian hierarchical model was used to estimate the association between PM<sub>2.5</sub> and hospitalization rates, with the first stage estimating the association within a single county and the second stage combining county-specific estimates to obtain national estimates. The authors found statistically significant evidence of seasonal and regional variation. The strongest association was for the northeast.

We use the national estimate for the all-year reported in Table 2 of Bell et al. (2008). The single pollutant coefficient and standard error are calculated from the estimated 0.8 percent increase in risk and 95% confidence interval (0.59-1.01 percent) for a 10 µg/m<sup>3</sup> increase in same-day (lag 0) daily 24-hour mean PM<sub>2.5</sub> (Bell et al., 2008, Table 2).

Note that Bell et al. (2008) considered a broader range of ICD-9 codes and estimated the risk of both cardiovascular events and cerebro- and peripheral vascular disease. For comparability to

other studies, EPA decided to apply a baseline hospitalization rate for ICD-9 codes 390-409 and 411-429 when using this C-R function in quantifying impacts.

**Functional Form:** Log-linear

**Coefficient:** 0.0008

**Standard Error:** 0.00011

**Incidence Rate:** county-specific daily hospital admission rate for all cardiovascular admissions less AMI per person ages 65+ (ICD-9 codes 390-409, 411-429)

**Population:** population of ages 65+

### ***Hospital Admissions for All Cardiovascular (Peng et al., 2008)***

Peng et al. (2008) examined the risk of hospital admissions for cardiovascular diseases (ICD-9 codes 426-427, 428, 430-438, 410-414, 429, 440-448) in relation to particulate matter (PM<sub>10-2.5</sub> and PM<sub>2.5</sub>). To accomplish this, the authors utilized a database of 108 U.S. counties with daily emergency hospital admission rates for cardiovascular diseases among Medicare enrollees living 9 miles from air, temperature, and dew-point temperature monitors. PM<sub>10-2.5</sub> and PM<sub>2.5</sub> concentrations were calculated by using monitoring data from January 1, 1999 through December 31, 2005. Overall, there were 3.7 million cardiovascular disease-related hospital admissions for the time period assessed. The authors found significant associations of PM<sub>2.5</sub> and PM<sub>10-2.5</sub> with cardiovascular disease admissions.

In a single-pollutant model, the coefficient and standard error are calculated from the estimated percent change in daily admission (0.44%) and 95% posterior interval (95% PI: 0.06-0.82%) for a 10 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> concentrations for the same day (Peng et al., 2008, page 2175).

Note that Peng et al. (2008) considered a broader range of ICD-9 codes and estimated the risk of both cardiovascular events and cerebro- and peripheral vascular disease. For comparability to other studies, EPA decided to apply a baseline hospitalization rate for ICD-9 codes 390-409 and 411-429 when using this C-R function in quantifying impacts.

**Functional Form:** Log-linear

**Coefficient:** 0.00071

**Standard Error:** 0.00013

**Incidence Rate:** county-specific daily hospital admission rate for all cardiovascular admissions less AMI per person ages 65+ (ICD-9 codes 390-409, 411-429)

**Population:** population of ages 65+

### ***Hospital Admissions for All Cardiovascular (Peng et al., 2009)***

Peng et al. (2009) investigated the relationship between hospital admissions for cardiovascular and the chemical components of PM<sub>2.5</sub> across 119 U.S. urban communities for 12 million Medicare enrollees using log-linear Poisson regression models. This was achieved using a national database with daily data from 2000-2006 on emergency hospital admissions of cardiovascular outcomes, ambient levels of PM<sub>2.5</sub> components and weather variables. Bayesian hierarchical statistical models were used to estimate the associations. Three scenarios for PM<sub>2.5</sub>

exposure were assessed which were as follows: 1) for the period 2000-2006 and including only days with available measurements for all 7 PM<sub>2.5</sub> components from the Speciation Trends network (STN); 2) PM<sub>2.5</sub> measured by the STN for the period 2000-2006 and including only days with available measurements for all 7 PM<sub>2.5</sub> components from the STN and 3) PM<sub>2.5</sub> estimated as the sum of the 7 largest components of PM<sub>2.5</sub> mass for the period 2000-2006. Results of percent increases in emergency admissions associated with PM<sub>2.5</sub> at lag 0 under these scenarios were showed in Figure 2 and the results for the components of PM<sub>2.5</sub> from both single and multi-pollutant models were showed in Figure 3.

In a single-pollutant model, the coefficient and standard error are calculated from the estimated percent change in daily admission (0.68%) and 95% posterior interval (95% PI: 0.26-1.10%) for a 10 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> concentrations for the same day (Peng et al., 2009, page 960).

Note that Peng et al. (2008) considered a broader range of ICD-9 codes and estimated the risk of both cardiovascular events and cerebro- and peripheral vascular disease. For comparability to other studies, EPA decided to apply a baseline hospitalization rate for ICD-9 codes 390-409 and 411-429 when using this C-R function in quantifying impacts.

**Functional Form:** Log-linear

**Coefficient:** 0.00068

**Standard Error:** 0.00021

**Incidence Rate:** county-specific daily hospital admission rate for all cardiovascular admissions less AMI per person ages 65+ (ICD-9 codes 390-409, 411-429)

**Population:** population of ages 65+

### ***Hospital Admissions for All Cardiovascular (Zanobetti et al., 2009)***

Zanobetti et al. (2009) examined the relationship between daily PM<sub>2.5</sub> levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure, respiratory disease and diabetes among 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific PM<sub>2.5</sub> composition while controlling for seasonal temperature as a substitute for ventilation. Overall, the authors found that PM<sub>2.5</sub> mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. The relationship between PM<sub>2.5</sub> and cardiovascular admissions was significantly modified when the mass of PM<sub>2.5</sub> was high in Br, Cr, Ni, and sodium ions, while mass high in As, Cr, Mn, organic carbon, Ni and sodium ions modified the myocardial infarction relationship and mass high in As, organic carbon, and sulfate ions modified the diabetes admission rates.

The single-pollutant coefficient and standard error are calculated from the estimated percent change in risk (1.89 percent) and 95% confidence interval (1.34-2.45) for a 10 µg/m<sup>3</sup> increase in 2-day averaged PM<sub>2.5</sub> (Zanobetti et al., 2009, Table 3).

Note that Zanobetti et al. (2009) report results for ICD-9 codes 390-429. In the benefit analysis, avoided nonfatal heart attacks are estimated separately. In order to avoid double counting heart

attack hospitalizations, we have excluded ICD-9 code 410 from the baseline incidence rate used in this function.

**Functional Form:** Log-linear

**Coefficient:** 0.00189

**Standard Error:** 0.00028

**Incidence Rate:** county-specific daily hospital admission rate for all cardiovascular admissions less AMI per person ages 65+ (ICD-9 codes 390-409, 411-429)

**Population:** population of ages 65+

### ***Hospital Admissions for All Cardiovascular (Moolgavkar, 2000b)***

Moolgavkar (2000b) examined the association between air pollution and cardiovascular hospital admissions (ICD-9 390-448) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO<sub>2</sub>, NO<sub>2</sub>, CO, and PM<sub>10</sub> in all three areas. PM<sub>2.5</sub> 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. In a single pollutant model, PM<sub>2.5</sub> was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the PM<sub>2.5</sub> effect dropped out and CO remained significant. For ages 20-64, SO<sub>2</sub> and CO exhibited the strongest effect and any PM<sub>2.5</sub> effect dropped out in co-pollutant models with CO. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM<sub>10</sub> or PM<sub>2.5</sub>. The strongest overall effects were observed for SO<sub>2</sub> and CO.

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.4 and t-statistic of 4.1 for a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> in the zero lag model for ages 18-64 (Moolgavkar, 2000b, Table 4).

Note that Moolgavkar (2000b) reported results that include ICD-9 code 410 (heart attack). In the benefits analysis, avoided nonfatal heart attacks are estimated separately. In order to avoid double counting heart attack hospitalizations, we have excluded ICD-9 code 410 from the baseline incidence rate used in this function.

**Functional Form:** Log-linear

**Coefficient:** 0.0014

**Standard Error:** 0.000341

**Incidence Rate:** county-specific daily hospital admission rate for all cardiovascular admissions per person ages 18 to 64 (ICD-9 codes 390-409, 411-429)

**Population:** population of ages 18 to 64

### ***Hospital Admissions for All Respiratory (Zanobetti et al., 2009)***

Zanobetti et al. (2009) examined the relationship between daily PM<sub>2.5</sub> levels and emergency hospital admissions for cardiovascular causes, myocardial infarction, congestive heart failure, respiratory disease and diabetes among 26 U.S. communities from 2000-2003. The authors used meta-regression to examine how this association was modified by season- and community-specific PM<sub>2.5</sub> composition while controlling for seasonal temperature as a substitute for

ventilation. Overall, the authors found that PM<sub>2.5</sub> mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. The relationship between PM<sub>2.5</sub> and cardiovascular admissions was significantly modified when the mass of PM<sub>2.5</sub> was high in Br, Cr, Ni, and sodium ions, while mass high in As, Cr, Mn, organic carbon, Ni and sodium ions modified the myocardial infarction relationship and mass high in As, organic carbon, and sulfate ions modified the diabetes admission rates.

In a single-pollutant model, the coefficient and standard error are estimated from the percent change in risk (2.07%) and 95% confidence interval (1.2% - 2.95%) for a 10 µg/m<sup>3</sup> increase in 2-day averaged PM<sub>2.5</sub> (Zanobetti et al., 2009, Table 3).

**Functional Form:** Log-linear

**Coefficient:** 0.00207

**Standard Error:** 0.00045

**Incidence Rate:** county-specific daily hospital admission rate for all respiratory admissions per person ages 65+ (ICD-9 codes 460 - 519)

**Population:** population of ages 65+

### ***Hospital Admissions for All Respiratory (Kloog et al., 2012)***

Kloog et al. (2012) investigated both the long and short term effects of PM<sub>2.5</sub> exposure on hospital admissions across New England for all residents aged 65 and older. The authors performed separate Poisson regression analysis for each admission type: all respiratory, cardiovascular disease (CVD), stroke and diabetes. Daily admission counts in each zip code were regressed against long and short-term PM<sub>2.5</sub> exposure, temperature, socio-economic data and a spline of time to control for seasonal trends in baseline risk. They observed associations between both short-term and long-term exposure to PM<sub>2.5</sub> and hospitalization for all of the outcomes examined.

In a single-pollutant model, the coefficient and standard error are estimated from the percent change in risk (0.70%) and 95% confidence interval (0.35% - 0.52%) for a 10 µg/m<sup>3</sup> increase in short-term (same day) PM<sub>2.5</sub> exposure (Kloog et al., 2012, Table 3).

**Functional Form:** Log-linear

**Coefficient:** 0.0007

**Standard Error:** 0.00096

**Incidence Rate:** county-specific daily hospital admission rate for all respiratory admissions per person ages 65+ (ICD-9 codes 460 - 519)

**Population:** population of ages 65+

### ***Hospital Admissions for Asthma (Sheppard, 2003)***

Sheppard et al. (1999) 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<sub>10</sub>, PM<sub>2.5</sub>, coarse PM<sub>10-2.5</sub>, SO<sub>2</sub>, ozone, and CO in a Poisson regression model with control for time trends,



seasonal variations, and temperature-related weather effects.<sup>29</sup> They found asthma hospital admissions associated with PM<sub>10</sub>, PM<sub>2.5</sub>, PM<sub>10-2.5</sub>, CO, and ozone. They did not observe an association for SO<sub>2</sub>. They found PM<sub>2.5</sub> 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<sub>2.5</sub> and CO. Results for other co-pollutant models were not reported.

In response to concerns that the work by Sheppard et al. (1999) may be biased because of concerns about the (S-plus) software used in the original analysis, Sheppard (2003) reanalyzed some of this work; in particular Sheppard reanalyzed the original study's PM<sub>2.5</sub> single pollutant model.

The coefficient and standard error are based on the relative risk (1.04) and 95% confidence interval (1.01-1.06) for a 11.8 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> in the 1-day lag GAM stringent model (Sheppard, 2003, pp 228-299).

**Functional Form:** Log-linear

**Coefficient:** 0.003324

**Standard Error:** 0.001045

**Incidence Rate:** county-specific daily hospital admission rate for asthma admissions per person (ICD-9 code 493)

**Population:** population of ages 0 -17<sup>30</sup>

### ***Hospital Admissions for Asthma (Babin et al., 2007)***

Babin et al. (2007) examined pediatric asthma-related emergency room (ER) visits and hospital admissions (ICD-9 code 493) in Washington, D.C. from 2001-2004 and their short-term associations with ozone, particulate matter, socioeconomic status, and age group. Applying Poisson regression analyses, the authors found significant associations between asthma ER visits and outdoor ozone concentrations for the 5-12 year old age group. The association between PM<sub>2.5</sub> and asthma hospitalization was found statistically insignificant.

The single pollutant coefficient and standard error are calculated from the estimated percent increase in risk (0.2 percent) and 95% confidence interval (-0.6 – 0.1 percent) for a 1 µg/m<sup>3</sup> increase in same-day (lag 0) daily 24-hour mean PM<sub>2.5</sub> based on single-pollutant models (Babin et al., 2007, Table 2).

**Functional Form:** Log-linear

**Coefficient:** 0.002

**Standard Error:** 0.00434

**Incidence Rate:** county-specific daily hospital admission rate for asthma admissions per person (ICD-9 code 493)

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<sup>29</sup> PM<sub>2.5</sub> levels were estimated from light scattering data.

<sup>30</sup> Although Sheppard (2003) reports results for the <65 year old age range, for comparability to other studies, we apply the results to the population of ages 0 to 17.

**Population:** population of ages 0 -17

### ***Hospital Admissions for Chronic Lung Disease (Moolgavkar, 2000a)***

Moolgavkar (2000a) examined the association between air pollution and COPD hospital admissions (ICD-9 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO<sub>2</sub>, NO<sub>2</sub>, CO, and PM<sub>10</sub> in all three areas. PM<sub>2.5</sub> 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<sub>10</sub>. In Los Angeles, marginally significant associations were observed for PM<sub>2.5</sub>, which were generally lower than for the gases. In co-pollutant models with CO, the PM<sub>2.5</sub> effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

The PM<sub>2.5</sub> C-R functions for the 20-64 age group are based on the single-pollutant model. Since the true PM<sub>2.5</sub> effect is most likely best represented by a distributed lag model, any single lag model should underestimate the total PM<sub>2.5</sub> effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

The single pollutant coefficient and standard error are calculated from an estimated percent change of 2.2 and t-statistic of 3.0 for a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> in the two-day lag model (Moolgavkar, 2000a, Table 4).

**Functional Form:** Log-linear

**Coefficient:** 0.0022

**Standard Error:** 0.000733

**Incidence Rate:** county-specific daily hospital admission rate for chronic lung disease admissions per person 18-64 (ICD-9 codes 490-496)

**Population:** population of ages 18 to 64<sup>31</sup>

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<sup>31</sup> Although Moolgavkar (2000a) 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.

## Emergency Room Visits

To estimate the effects of PM<sub>2.5</sub> air pollution reductions on asthma-related ER visits, we use the C-R functions based on Mar et al. (2010), Slaughter et al. (2005) and Glad et al. (2012). COBRA estimates the incidence results for ER visits by pooling these three studies using random/fixed pooling method. Exhibit C-7 below summarizes the attributes of the C-R functions used in COBRA.

**Exhibit C-7. Health Impact Functions for Particulate Matter and Emergency Room Visits**

Author	Year	Location	Age	Metric	Beta	Std Error	Functional Form
Mar et al.	2010	Greater Tacoma, Washington	0-99	24-hr avg	0.0056	0.0021	Log-linear
Slaughter et al.	2005	Spokane, Washington	0-99	24-hr avg	0.0029	0.0027	Log-linear
Glad et al.	2012	Pittsburgh, PA	0-99	24-hr avg	0.0039	0.0028	Logistic

### ***Emergency Room Visits for Asthma (Mar et al., 2010)***

Mar et al. (2010) assessed the effect of particulate matter air pollution, including emissions from diesel generators, on emergency room visits for asthma in the greater Tacoma, Washington area from January 3, 1998 to May 30, 2002 using Poisson regression models. Health data were collected for individuals of all ages from 6 Tacoma hospitals. The authors also assessed the impacts of diesel generator use on emergency room visits for asthma from January 24, 2001 to June 2, 2001. Overall, the researchers found an association between daily PM<sub>2.5</sub> levels and emergency room visits for asthma at lag days 2 and 3, with a relative risk for lag day 2 of 1.04 (95% CI: 1.01-1.07) and a relative risk for lag day 3 of 1.03 (95% CI: 1.0-1.06). No significant association between emergency room visits for asthma and increased use of the diesel generators was observed.

In a single-pollutant model, the PM<sub>2.5</sub> coefficient and standard error are estimated from the relative risk (1.04) and 95% confidence interval (95% CI: 1.01-1.07) for a 7 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> at lag day 2 (Mar et al., 2010, Table 4).

**Functional Form:** Log-linear

**Coefficient:** 0.0056

**Standard Error:** 0.0021

**Incidence Rate:** county-specific daily asthma emergency room rate per person (The study didn't report ICD-9 code but we assume ICD-9 code 493)

**Population:** population of all ages

### ***Emergency Room Visits for Asthma (Slaughter et al., 2005)***

Slaughter et al. (2005) examined the short-term association of particulate matter (PM<sub>1</sub>, PM<sub>2.5</sub>, PM<sub>10</sub>, and PM<sub>10-2.5</sub>) and carbon monoxide with hospital admissions and emergency room visits for respiratory and cardiac outcomes and mortality in Spokane, Washington from January 1995 to June 2001 using a log-linear generalized linear model. The authors found no association between respiratory emergency room visits and any size fraction of PM<sub>2.5</sub>, but there was a

suggestive relationship between fine PM<sub>2.5</sub> and respiratory effects when compared to coarse PM<sub>2.5</sub>. No association between cardiac hospital admissions or mortality and any size fraction of PM<sub>2.5</sub> or CO was observed at the 0- to 3-day lag. CO, on the other hand, was found to be associated with all respiratory emergency room visits and visits for asthma at the 3-day lag.

In a single-pollutant model, the coefficient and standard error are estimated from the relative risk (1.03) and 95% confidence interval (95% CI: 0.98-1.09) for a 10 µg/m<sup>3</sup> increase in daily 24-hour mean PM<sub>2.5</sub> at 1-day lag (Slaughter et al., 2005, Table 4).

**Functional Form:** Log-linear

**Coefficient:** 0.0029

**Standard Error:** 0.0027

**Incidence Rate:** county-specific daily asthma emergency room rate per person (ICD-9 code 493)

**Population:** population of all ages

### ***Emergency Room Visits for Asthma (Glad et al., 2012)***

Glad et al. (2012) investigated the relationship between air pollution and emergency department (ED) visits for asthma in the Pittsburgh, Pennsylvania area between 2002 and 2005 using a case-crossover methodology with a logistical model. The authors found a 2.5% increase in asthma ED visits for each 10 ppb increase in the 1-hour maximum ozone level on day 2 (odds ratio [OR] = 1.025,  $p < 0.05$ ). Particulate matter with an aerodynamic diameter  $\leq 2.5$  µm (PM<sub>2.5</sub>) had an effect both on the total population on day 1 after exposure (1.036,  $p < 0.05$ ), and on African Americans on days 1, 2, and 3. PM<sub>2.5</sub> had no significant effect on Caucasian Americans alone. The disparity in risk estimates by race may reflect differences in residential characteristics, exposure to ambient air pollution, or a differential effect of pollution by race.

In a single-pollutant model, the coefficient and standard error are estimated from the relative risk (1.040) and 95% confidence interval (95% CI: 0.984-1.100) for a 10 µg/m<sup>3</sup> increase 6-day average of daily PM<sub>2.5</sub> (Glad et al., 2012, Table 3).

**Functional Form:** Logistic

**Coefficient:** 0.0039

**Standard Error:** 0.0028

**Incidence Rate:** county-specific daily asthma emergency room rate per person (ICD-9 code 493)

**Population:** population of all ages

## Minor Effects

We include functions to estimate acute bronchitis, lower respiratory symptoms, minor restricted days, and work loss days.

**Exhibit C-8. Health Impact Functions for Particulate Matter and Acute Effects**

Endpoint Name	Author	Year	Location	Age	Metric	Beta	Std Error	Function Form
Minor Restricted Activity Days	Ostro & Rothschild	1989	Nationwide	18-64	24-hr avg	0.007410	0.000700	Log-linear
Acute Bronchitis	Dockery et al.	1996	24 communities	8-12	Annual	0.027212	0.017096	Logistic
Work Loss Days	Ostro	1987	Nationwide	18-64	24-hr avg	0.004600	0.000360	Log-linear
Lower Respiratory Symptoms	Schwartz and Neas	2000	6 U.S. cities	7-14	24-hr avg	0.019012	0.006005	Logistic

### ***Acute Bronchitis (Dockery et al., 1996)***

Dockery et al (1996) examined the relationship between PM<sub>2.5</sub> 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<sub>2.1</sub> and PM<sub>10</sub> were marginally significantly related to bronchitis.<sup>32</sup> They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study 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), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM<sub>15</sub>. Because it is based on a larger sample, the Dockery et al (1996) study is the better study to develop a C-R function linking PM<sub>2.5</sub> with bronchitis.

Bronchitis was counted in the study only if there were “reports of symptoms in the past 12 months” (Dockery et al., 1996, 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<sub>2.5</sub> and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the health impact function based on Dockery et al. is measuring acute bronchitis. The health impact function is based on results of the single pollutant model reported in Table 1.

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<sub>2.1</sub> = 20.7

<sup>32</sup> The original study measured PM<sub>2.1</sub>, however when using the study's results we use PM<sub>2.5</sub>. This makes only a negligible difference, assuming that the adverse effects of PM<sub>2.1</sub> and PM<sub>2.5</sub> are comparable.

$\mu\text{g}/\text{m}^3$ ) versus the least polluted city ( $\text{PM}_{2.1} = 5.8 \mu\text{g}/\text{m}^3$ ) (Dockery et al., 1996, Tables 1 and 4). The original study used  $\text{PM}_{2.1}$ , however, we use the  $\text{PM}_{2.1}$  coefficient and apply it to  $\text{PM}_{2.5}$  data.

**Functional Form:** Logistic

**Coefficient:** 0.027212

**Standard Error:** 0.017096

**Incidence Rate:** annual bronchitis incidence rate per person in 2010 = 0.043 (American Lung Association, 2013, Table 11)

**Population:** population of ages 8-12

### ***Lower Respiratory Symptoms (Schwartz & Neas, 2000)***

Schwartz and Neas (2000) used logistic regression to link lower respiratory symptoms and cough in children with coarse  $\text{PM}_{10}$ ,  $\text{PM}_{2.5}$ , sulfate and  $\text{H}^+$  (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The 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 coefficient and standard error are calculated from the reported odds ratio (1.33) and 95% confidence interval (1.11-1.58) associated with a  $15 \mu\text{g}/\text{m}^3$  change in  $\text{PM}_{2.5}$  (Schwartz & Neas, 2000, Table 2).

**Functional Form:** Logistic

**Coefficient:** 0.01901

**Standard Error:** 0.006005

**Incidence Rate:** daily lower respiratory symptom incidence rate per person = 0.0012 (Schwartz et al., 1994, Table 2).

**Population:** population of ages 7 to 14

### ***Minor Restricted Activity Days (Ostro and Rothschild, 1989)***

Ostro and Rothschild (1989) estimated the impact of  $\text{PM}_{2.5}$  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<sup>33</sup>. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for  $\text{PM}_{2.5}$ , two-week average ozone has highly variable association with RRADs and MRADs. Controlling for ozone, two-week average  $\text{PM}_{2.5}$  was significantly linked to both health endpoints in most years.<sup>34</sup> The health impact function for  $\text{PM}_{2.5}$  is based on this co-pollutant model.

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<sup>33</sup> 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 health impact function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

<sup>34</sup> The study used a two-week average pollution concentration; the health impact function uses a daily average, which is assumed to be a reasonable approximation.

The study is based on a “convenience” sample of non-elderly individuals. Applying the health impact function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM<sub>2.5</sub> as individuals under 65.

Using the results of the two-pollutant model (O<sub>3</sub> and PM<sub>2.5</sub>), 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, Table 4) using the inverse of the variance as the weight:

$$\beta = \frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} = 0.00741$$

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

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

This reduces down to:

$$\sigma_{\beta} = \sqrt{1/\gamma} = 0.00070$$

**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 & Rothschild, 1989, p. 243).

**Population:** adult population ages 18 to 64

### **Work Loss Days (Ostro, 1987)**

Ostro (1987) estimated the impact of PM<sub>2.5</sub> 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.<sup>35</sup> The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average PM<sub>2.5</sub> levels<sup>36</sup> were significantly linked to work-loss days, RADs, and RRADs, however

<sup>35</sup> 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 health impact function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations.

<sup>36</sup> The study used a two-week average pollution concentration; the health impact function uses a daily average, which is assumed to be a reasonable approximation.

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, Table 3) using the inverse of the variance as the weight.

The study is based on a “convenience” sample of non-elderly individuals. Applying the health impact function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM<sub>2.5</sub> as individuals under 65. 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).

The coefficient used in the health impact function is a weighted average of the coefficients in Ostro (1987, Table 3) using the inverse of the variance as the weight:

$$\beta = \frac{\sum_{i=1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^2}} = 0.0046$$

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

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

This eventually reduces down to:

$$\sigma_{\beta} = \sqrt{1/\gamma} = 0.00036$$

**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, Hendershot, & Marano, 1999, Table 41; U.S. Bureau of the Census, 1997, No. 22)

**Population:** adult population ages 18 to 64



## Asthma-Related Effects

We pool the results of studies by Ostro et al. (2001) and Mar et al. (2004) to get an estimate of asthma exacerbation in asthmatics. In addition to the lower respiratory estimate, we include an upper respiratory estimate based on a study by Pope et al. (1991).

**Exhibit C-9. Health Impact Functions for Particulate Matter and Asthma-Related Effects**

Endpoint Name	Author	Year	Location	Age	Metric	Beta	Std Error	Functional Form
Asthma Exacerbation, Cough	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.000985	0.000747	Logistic
Asthma Exacerbation, Shortness of Breath	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.002565	0.001335	Logistic
Asthma Exacerbation, Wheeze	Ostro et al.	2001	Los Angeles, CA	6-18	24-hr avg	0.001942	0.000803	Logistic
Asthma Exacerbation, Cough	Mar et al.	2004	Vancouver, CAN	6-18	24-hr avg	0.01906	0.009828	Logistic
Asthma Exacerbation, Shortness of Breath	Mar et al.	2004	Vancouver, CAN	6-18	24-hr avg	0.01222	0.013849	Logistic
Upper Respiratory Symptoms	Pope et al.	1991	Utah Valley	9-11	24-hr avg	0.0036	0.0015	Logistic

### ***Pooling Ostro et al. (2001) and Mar et al. (2004)***

To characterize asthma exacerbations in children, we use two studies that followed panels of asthmatic children. Ostro et al. (2001) followed a group of 138 African-American children in Los Angeles for 13 weeks, recording daily occurrences of respiratory symptoms associated with asthma exacerbations (e.g., shortness of breath, wheeze, and cough). This study found a statistically significant association between PM<sub>2.5</sub>, measured as a 12-hour average, and the daily prevalence of shortness of breath and wheeze endpoints. Although the association was not statistically significant for cough, the results were still positive and close to significance; consequently, we decided to include this endpoint, along with shortness of breath and wheeze, in generating incidence estimates.

Mar et al. (2004) followed nine asthmatic children for over eight months in Spokane, Washington. Data on respiratory symptoms and medication use were recorded daily by the study's subjects, while air pollution data was collected by the local air agency and Washington State University. The authors found a strong association between cough symptoms and several metrics of particulate matter, including PM<sub>2.5</sub>.

We employed the following pooling approach in combining effect estimates from the two studies to produce a single asthma exacerbation incidence estimate. First, we pooled (with a

fixed/random effects approach) the incidence estimates based on the two studies for “cough” and “shortness of breath” separately. We then assigned an equal weight (i.e., 0.33) to the pooled results for cough, the pooled results for shortness of breath, and the (un-pooled) results for wheeze (from Ostro et al., 2001).

To prevent double-counting, we followed U.S. EPA (2005, p. 4-38) and focused the estimation on asthma exacerbations occurring in children and excluded adults from the calculation. Asthma exacerbations occurring in adults are assumed to be captured in the general population endpoints such as work loss days and MRADs. Consequently, if we had included an adult-specific asthma exacerbation estimate, this would likely have double-counted incidence for this endpoint. However, because the general population endpoints do not cover children (with regard to asthmatic effects), an analysis focused specifically on asthma exacerbations for children (6 to 18 years of age) could be conducted without concern for double-counting.

### ***Asthma Exacerbation: Cough, Wheeze, and Shortness of Breath (Ostro et al., 2001)***

Ostro et al. (2001) 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<sub>10</sub>, PM<sub>2.5</sub>, NO<sub>2</sub>, and O<sub>3</sub> in a logistic regression model with control for age, income, time trends, and temperature-related weather effects.<sup>37</sup> 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 cough prevalence associated with PM<sub>10</sub> and PM<sub>2.5</sub> and cough incidence associated with PM<sub>2.5</sub>, PM<sub>10</sub>, and NO<sub>2</sub>. Ozone was not significantly associated with cough among asthmatics.

Note that the study focused on African-American children ages 8 to 13 years old. We apply the function based on this study to the general population ages 6 to 18 years old.

### **Asthma Exacerbation, Cough**

The coefficient and standard error are based on an odds ratio of 1.03 (95% CI 0.98-1.07) for a 30 µg/m<sup>3</sup> increase in 12-hour average PM<sub>2.5</sub> concentration (Ostro et al., 2001, Table 4, p. 204).

**Functional Form:** Logistic

**Coefficient:** 0.000985

**Standard Error:** 0.000747

**Incidence Rate:** daily cough rate per person (Ostro et al., 2001, p. 202) = 0.145

**Population:** Asthmatic population ages 6 to 17 = 10.70%; asthmatic population age 18 = 7.19%.<sup>38</sup>

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<sup>37</sup> The authors note that there were 26 days in which PM<sub>2.5</sub> concentrations were reported higher than PM<sub>10</sub> 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.

<sup>38</sup> The American Lung Association (2010, Table 7) estimates asthma prevalence for children ages 5-17 at 10.70% and for adults ages 18-44 at 7.19% (based on data from the 2008 National Health Interview Survey).

### **Asthma Exacerbation, Shortness of Breath**

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.00-1.17) for a 30  $\mu\text{g}/\text{m}^3$  increase in 12-hour average  $\text{PM}_{2.5}$  concentration (Ostro et al., 2001, Table 4, p. 204).

**Functional Form:** Logistic

**Coefficient:** 0.002565

**Standard Error:** 0.001335

**Incidence Rate:** daily shortness of breath rate per person (Ostro et al., 2001, p. 202) = 0.074

**Population:** Asthmatic population ages 6 to 17 = 10.70%; asthmatic population age 18 = 7.19%.

### **Asthma Exacerbation, Wheeze**

The coefficient and standard error are based on an odds ratio of 1.06 (95% CI 1.01-1.11) for a 30  $\mu\text{g}/\text{m}^3$  increase in 12-hour average  $\text{PM}_{2.5}$  concentration (Ostro et al., 2001, Table 4, p. 204).

**Functional Form:** Logistic

**Coefficient:** 0.001942

**Standard Error:** 0.000803

**Incidence Rate:** daily wheeze rate per person (Ostro et al., 2001, p. 202) = 0.173

**Population:** Asthmatic population ages 6 to 17 = 10.70%; asthmatic population age 18 = 7.19%.

### ***Asthma Exacerbation, Cough and Shortness of Breath (Mar et al., 2004)***

Mar et al. (2004) studied the effects of various size fractions of particulate matter on respiratory symptoms of adults and children with asthma, monitored over many months. The study was conducted in Spokane, Washington, a semiarid city with diverse sources of particulate matter. Data on respiratory symptoms and medication use were recorded daily by the study's subjects, while air pollution data was collected by the local air agency and Washington State University. Subjects in the study consisted of 16 adults – the majority of whom participated for over a year – and nine children, all of whom were studied for over eight months. Among the children, the authors found a strong association between cough symptoms and several metrics of particulate matter, including  $\text{PM}_{2.5}$ . However, the authors found no association between respiratory symptoms and  $\text{PM}_{2.5}$  of any metric in adults. Mar et al. therefore concluded that the discrepancy in results between children and adults was due either to the way in which air quality was monitored, or a greater sensitivity of children than adults to increased levels of  $\text{PM}_{2.5}$  air pollution.

### **Asthma Exacerbation, Cough**

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.21) and 95% confidence interval (1.00-1.47) for a 10.0  $\mu\text{g}/\text{m}^3$  increase in 1-day lagged concentration of  $\text{PM}_{2.5}$  (Mar et al., 2004, Table 7).

**Functional Form:** Logistic

**Coefficient:** 0.019062

**Standard Error:** 0.009828

**Incidence Rate:** daily cough rate per person (Ostro et al., 2001) = 14.5%

**Population:** The study reported results for population ages 7-12. For comparability to other studies, we apply the results to the population of ages 6 to 18. Asthmatic population ages 6 to 17 = 10.70%; asthmatic population age 18 = 7.19%.

### ***Asthma Exacerbation, Shortness of Breath***

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.13) and 95% confidence interval (0.86-1.48) for a 10.0  $\mu\text{g}/\text{m}^3$  increase in current-day concentration of PM<sub>2.5</sub> (Mar et al., 2004, Table 7).

**Functional Form:** Logistic

**Coefficient:** 0.012222

**Standard Error:** 0.013849

**Incidence Rate:** daily shortness of breath rate per person (Ostro et al., 2001, p.202) = 7.4%

**Population:** The study reported results for population ages 7-12. For comparability to other studies, we apply the results to the population of ages 6 to 18. Asthmatic population ages 6 to 17 = 10.70%; asthmatic population age 18 = 7.19%.

### ***Upper Respiratory Symptoms (Pope, 1991)***

Using logistic regression, Pope et al. (1991) estimated the impact of PM<sub>10</sub> 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<sub>10</sub> 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<sub>2</sub>, and SO<sub>2</sub> 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, 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, Table 5) show PM<sub>10</sub> significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM<sub>10</sub> effect. The results from the school-based sample are used here.

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

**Functional Form:** Logistic

**Coefficient:** 0.0036

**Standard Error:** 0.0015

**Incidence Rate:** daily upper respiratory symptom incidence rate per person = 0.3419 (Pope, 1991, Table 2)

**Population:** Asthmatic population ages 6 to 17 = 10.70% <sup>39</sup> of population ages 9 to 11.

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<sup>39</sup> The American Lung Association (2010, Table 7) estimates asthma prevalence for children 5-17 at 10.70% (based on data from the 2008 National Health Interview Survey).

## Appendix D: Baseline Incidence Rates for Adverse Health Effects

Health impact functions developed from log-linear or logistic models estimate the percent change in an adverse health effect associated with a given pollutant change. In order to estimate the absolute change in incidence using these functions, we need the baseline incidence rate of the adverse health effect. For certain health effects, such as asthma exacerbation, we need a prevalence rate, which estimates the percentage of the general population with a given ailment like asthma. This Appendix describes the data used to estimate baseline incidence rates and prevalence rates for the health effects considered in this analysis.

### Mortality

This section describes the development of county mortality rates for years 2015, 2025 and 2030 for use in COBRA.<sup>40</sup> First, we describe the source of 2012-2014 individual-level mortality data and the calculation of county-level mortality rates. Then we describe how we use national-level Census mortality rate projections to develop county-level mortality rate projections for years 2015, 2025 and 2030.

#### **Mortality Data for 2012-2014**

We obtained individual-level mortality data from 2012-2014 for the whole United States from the Centers for Disease Control (CDC), National Center for Health Statistics (NCHS). The data were compressed into a CD-ROM, which contains death information for each decedent, including residence county FIPS, age at death, month of death, and underlying causes (ICD-10 codes).

Using the detailed mortality data combined with county-level inter-censal population estimates,<sup>41</sup> we generated age-, cause-, and county-specific mortality rates using the following formula:

$$RR_{i,jj,kk} = \frac{DD_{i,jj,kk}(2012) + DD_{i,jj,kk}(2013) + DD_{i,jj,kk}(2014)}{PP_{ii,kk}(2012) + PP_{ii,kk}(2013) + PP_{ii,kk}(2014)}$$

where  $R_{i,j,k}$  is the mortality rate for age group  $i$ , cause  $j$ , and county  $k$ ;  $D$  is the death count; and  $P$  is the population.

For county-age group cells with fewer than 10 deaths, CDC WONDER suppresses the exact death count. For these observations, a mortality rate cannot be calculated. For each combination

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<sup>40</sup> We use projected 2020 mortality rates for analysis year 2017 in COBRA because the Census Bureau national mortality rates are projected only every 5 years and are not available for 2017.

<sup>41</sup> The detailed mortality data obtained from CDC do not include population. The county-level inter-censal population estimates are based on US Census of Population and Housing 2010 and forecasts developed by Woods & Poole (2011).

of age group and mortality cause, we used the following procedure to deal with suppressed counts.

For each combination of state, age group and mortality cause, we grouped counties with unsuppressed mortality figures and summed their reported death counts. We then subtracted these unsuppressed deaths from the state-level age- and cause-specific death count, which includes suppressed deaths. We divided the resulting state-wide death count in suppressed counties by the age-specific populations in those counties. This calculation resulted in an age- and cause- specific average mortality rate for suppressed counties.

In some instances, age- and cause-specific death counts were suppressed at both the county and state level. In these cases, we substituted national-level age- and cause- specific mortality rates for the respective missing county mortality rates.

Following CDC Wonder (<http://wonder.cdc.gov>), we treated mortality rates as “unreliable” when the death count is less than 20.<sup>42</sup> For each combination of age group and mortality cause, we used the following procedure to deal with the problem of “unreliable” rates:

- For a given state, we grouped the counties where the death count (i.e., the numerator on the right-hand side of the above equation) was less than 20 and summed those death counts across those counties. If the sum of deaths was greater than or equal to 20, we then summed the populations in those counties, and calculated a single rate for the “state collection of counties” by dividing the sum of deaths by the sum of populations in those counties. This rate was then applied to each of those counties.<sup>43</sup>
- If the sum of deaths calculated in the above step was still less than 20, the counties in the “state collection of counties” were not assigned the single rate from the above step. Instead, we proceeded to the regional level (see Exhibit D-1 for region definition). In each region, we identified all counties whose death counts were less than 20 (excluding any such counties that were assigned a rate in the previous step). We summed the death counts in those counties. If the sum of deaths was greater than or equal to 20, we then summed the populations in those counties, and calculated a single rate for the “regional collection of counties” by dividing the sum of deaths by the sum of populations in those counties. This rate was then applied to each of those counties in the “regional collection of counties.”<sup>44</sup>

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<sup>42</sup> Among all the calculated age-, cause-, and county-specific mortality rates, there were about 67% “unreliable” rates.

<sup>43</sup> After this adjustment, there were 17% unreliable rates left.

<sup>44</sup> After this regional adjustment, there were 7% unreliable rates left.

**Exhibit D-1. Regional Definitions from U.S. Census**

Region	States Included
Northeast	Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Pennsylvania
Midwest	Ohio, Indiana, Illinois, Michigan, Wisconsin, Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas
South	Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida, Kentucky, Tennessee, Alabama, Mississippi, Arkansas, Louisiana, Oklahoma, Texas
West	Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada, Washington, Oregon, California, Alaska, Hawaii

- If the sum of deaths calculated in the previous (regional) step was still less than 20, the counties in the “regional collection of counties” were not assigned the single rate from the above step. Instead, we proceeded to the national level, identifying all counties in the nation whose death counts were less than 20 (excluding any such counties that were assigned a rate in the previous steps). We summed the death counts in those counties and divided by the sum of the populations in those counties to derive a single rate for the “national collection of counties.” This rate was then applied to each of those counties in the “national collection of counties.”<sup>45</sup> In these cases where national adjustment still did not yield a death count greater than 20, we simply calculated a single rate for the “national collection of counties, even though it was “unreliable,” and assigned it to those counties in the “national collection of counties.”

Exhibit D-2 shows the resulting national average all-cause mortality rates.

**Exhibit D-2. National All-Cause Mortality Rates (per 100 people per year) by Age Group**

Mortality Category	Infant *	1--17	18--24	25--34	35--44	45--54	55--64	65--74	75--84	85+
Mortality, All Cause	0.593 96	0.019 51	0.078 04	0.106 65	0.172 64	0.405 42	0.861 62	1.796 70	4.628 37	13.580 34

\* We estimate post-neonatal mortality (deaths after the first month) for infants because the health impact function (see Appendix F) estimates post-neonatal mortality.

***Mortality Rate Projections to 2015, 2025 and 2030***

To estimate age- and county-specific mortality rates in years 2015, 2025 and 2030, we calculated adjustment factors, based on a series of Census Bureau projected national mortality rates (for all-

<sup>45</sup> Even after this national adjustment, there were about 1% unreliable rates left. In these cases, we simply calculated a single rate for the “national collection of counties”, even though it was “unreliable,” and assigned it to those counties in the “national collection of counties.”



cause mortality), to adjust the age- and county-specific mortality rates calculated using 2012-2014 data as described above. We used the following procedure:

- For each age group, we obtained the series of projected national mortality rates from 2013 to 2050 (see the 20135 rate in Exhibit D-3) based on Census Bureau projected life tables.<sup>46</sup>
- We then calculated, separately for each age group, the ratio of Census Bureau national mortality rate in years 2015, 2025, and 2030 to the 2013 rate. These ratios are shown in Exhibit D-4.
- Finally, to estimate mortality rates in years 2015, 2025 and 2030 that are both age group-specific and county-specific, we multiplied the county- and age-group-specific mortality rates for 2012-2014 by the appropriate ratio calculated in the previous step. For example, to estimate the projected mortality rate in 2015, 2025 and 2030 among ages 18-24 in Wayne County, MI, we multiplied the mortality rate for ages 18-24 in Wayne County in 2012-2014 by the ratio of Census Bureau projected national mortality rate in 2015, 2025 and 2030 for ages 18-24 to Census Bureau national mortality rate in 2013 for ages 18-24.

**Exhibit D-3. All-Cause Mortality Rate (per 100 people per year), by Source, Year, and Age Group**

Source & Year	Infant*	1--17	18--24	25--34	35--44	45--54	55--64	65--74	75--84	85+
Calculated CDC 2012-2014	0.594*	0.020	0.078	0.107	0.173	0.405	0.862	1.797	4.628	13.580
Census Bureau 2013**	0.654	0.029	0.088	0.102	0.183	0.387	0.930	2.292	5.409	13.091

\* The Census Bureau estimate is for all deaths in the first year of life. COBRA uses post-neonatal mortality (deaths after the first month, i.e., 0.23 per 100 people) because the health impact function (see Appendix F) estimates postneonatal mortality. For comparison purpose, we also calculated the rate for all deaths in the first year, which is 0.684 per 100 people).

**Exhibit D-4. Ratio of 2025 and 2020 All-Cause Mortality Rate to 2013 Estimated All-Cause Mortality Rate, by Age Group**

Year	Infant	1--17	18--24	25--34	35--44	45--54	55--64	65--74	75--84	85+
2025	0.85	0.81	0.74	0.80	0.75	0.77	0.85	0.91	0.93	0.97
2020	0.94	0.94	0.98	1.04	0.97	0.98	1.02	1.03	1.03	1.00

<sup>46</sup> For a detailed description of the model, the assumptions, and the data used to create Census Bureau projections, see the working paper, "Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100, Working Paper #38.", which is available on <http://www.census.gov/population/www/documentation/twps0038/twps0038.html> (Hollman, et al. 2000) .

## Hospitalizations

Hospitalization rates were calculated using data from the Healthcare Cost and Utilization Project (HCUP). HCUP is a family of health care databases developed through a Federal-State-Industry partnership and sponsored by the Agency for Healthcare Research and Quality (AHRQ).<sup>47</sup> HCUP products include the State Inpatient Databases (SID), the State Emergency Department Databases (SEDD), the Nationwide Inpatient Sample (NIS), and the Nationwide Emergency Department Sample (NEDS). HCUP databases can be obtained from the following data services:

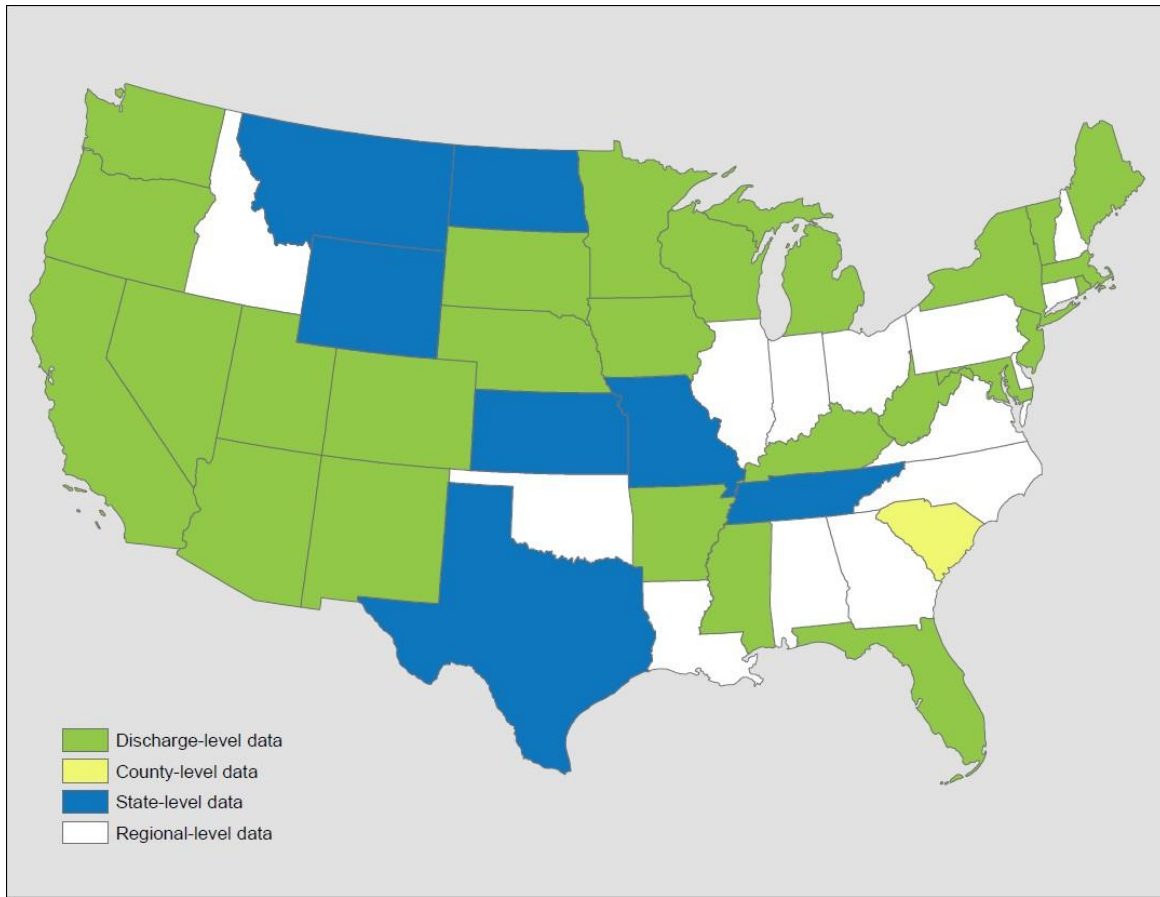
- The HCUP Central Distributor: Many of the HCUP databases are available for purchase through the HCUP Central Distributor. The databases include detailed information for individual discharges, such as primary diagnosis (in ICD-9 codes), patient's age and residence county.
- HCUP State Partners: Some HCUP participating states do not release their data to the Central Distributor; however, the data may be obtained through contacting the State Partners. Some State Partners (e.g., CA, TX, and NY) provided discharge-level data; others (e.g., OH) provided summarized data.
- HCUPnet: This is a free, on-line query system based on data from HCUP. It provides access to summary statistics at the state, regional and national levels.

Exhibit D-5 shows the level of hospitalization data (e.g., discharge-level or state-level) for each state. Note that for some states neither discharge-level nor state-level data were available. In such cases, we used regional statistics from HCUPnet to estimate hospitalization rates for those states. The data year for states using HCUPnet data is 2014. For discharge-level data, the data year for most states is 2014; however, some states provided data for 2011 (CA, MS); 2012 (ME); and 2013 (AR, MA, MD, NV, SD, UT). We assume hospitalization rates are reasonably constant from 2011-2014 and consider all as 2014 rates.

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<sup>47</sup> More information about HCUP can be found at <http://www.hcup-us.ahrq.gov/>.

**Exhibit D-5. Hospitalization Data from HCUP**



The procedures for calculating hospitalization rates are summarized as follows:<sup>48</sup>

- For states with discharge-level data:
  - We calculated age-, health endpoint-, and county-specific hospitalization counts.<sup>49</sup>
  - The above calculation excluded hospitalizations with missing patient age or county FIPS, which may lead to underestimation of rates. Therefore we scaled up the previously calculated age-, endpoint-, and county-specific counts using an adjustment factor obtained as follows:

<sup>48</sup> The data year for most states is 2007; the exception is MA, for which the data year is 2006. We assume hospitalization rates are reasonably constant from 2006-2007 and consider all as 2007 rates.

<sup>49</sup> Ohio was the only state that, while not providing discharge-level data, did provide county-level data for each age group-endpoint combination.

- We first counted the number of discharges for a specific endpoint in the state including those discharges with missing age or county FIPS.
    - We then counted the number of discharges for the endpoint in the state excluding those records with missing age or county FIPS.
    - The adjustment factor is the ratio of the two counts.
  - For California and West Virginia, patient county was unavailable for all observations. For these two states, we used hospital county in place of patient county.
  - We calculated hospitalization rates for each county by dividing the adjusted county-level hospitalization counts by the Census estimated county-level population for the corresponding year (2011 – 2014). Following CDC Wonder, we treated rates as “unreliable” when the hospitalization count was less than 20, using the same procedure we used for mortality rates above.
- For states with summarized state statistics (from HCUPnet) we calculated the state-, age-, endpoint- specific hospitalization rates and applied them to each county in the state. We used the previously described procedure to adjust the “unreliable” rates.
- For states without discharge-level or state-level data:
  - We obtained the endpoint-specific hospitalization counts in each region from HCUPnet/NIS (we refer to this count for the *i*th endpoint in the *j*th region as “TOTAL<sub>*ij*</sub>”).
  - For those states in the *j*th region that do have discharge-level or state-level data, we summed the hospital admissions by endpoint (we refer to this count for the *i*th endpoint in the *j*th region as “SUB *ij*”).
  - We then estimated the hospitalization count for states without discharge or state data for the *i*th endpoint in the *j*th region as TOTAL<sub>*ij*</sub> – SUB *ij*. Note that while this count is endpoint- and region- specific, it is not age-specific. We obtained the distribution of hospital admission counts across age groups based on the National Hospital Discharge Survey (NHDS) and assumed the same distribution for the HCUP hospitalizations. We then applied this distribution to the estimated hospital counts (i.e., TOTAL<sub>*ij*</sub> – SUB *ij*) to obtain endpoint-, region-, and age-specific counts.
  - Using the corresponding age- and region-specific populations, we calculated age-specific hospitalization rates for the *i*th endpoint in the *j*th region and applied them to those counties in the region that didn’t have discharge-level or state-level data.

Exhibit D-6 shows the resulting average national hospitalization rates by health endpoint and age group.

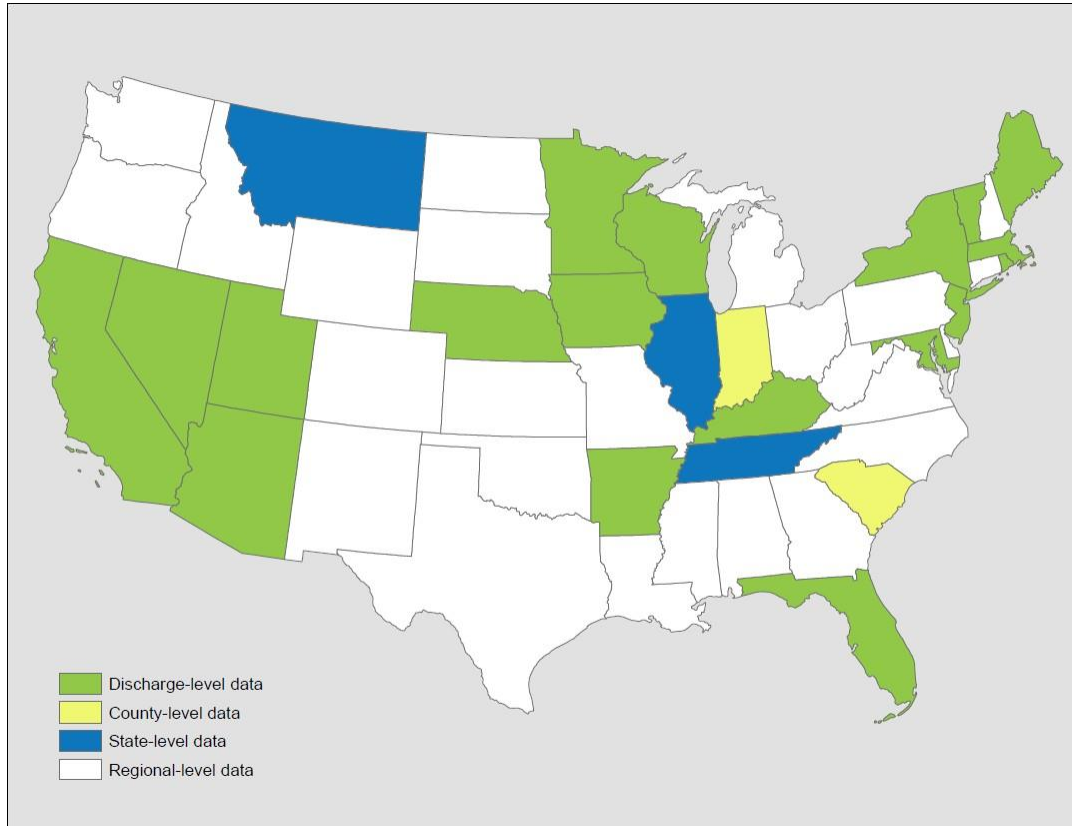
**Exhibit D-6. National Hospitalization Rates, by Health Endpoint and Age Group**

Hospitalization Category	ICD-9 Codes	Hospitalization Rate by Age Group (admissions per 100 people per year)								
		Age 0-1	2-17	18-24	25-34	45-54	55-64	65-74	75-84	85+
<b>Respiratory</b>										
all respiratory	460-519	2.387	0.363	0.166	0.212	0.340	0.737	1.297	2.292	4.151
asthma	493	0.217	0.147	0.036	0.048	0.076	0.123	0.136	0.157	0.218
chronic lung disease	490-496	0.226	0.151	0.041	0.056	0.105	0.281	0.496	0.837	1.276
<b>Cardiovascular</b>										
all cardiovascular	390-429	0.044	0.017	0.061	0.138	0.377	0.914	1.747	3.131	5.886

## Emergency Room Visits for Asthma

The data source for emergency department/room (ED or ER) visits is also HCUP, i.e., SID, SEDD, and NEDS. The types of data providers are also the same as those described above for hospitalizations. Exhibit D-7 shows the emergency department data in each state.

**Exhibit D- 7. Emergency Department Data from HCUP**



The calculation of ER visit rates is also similar to the calculation of hospitalization rates, except for the following differences:

- The SEDD databases include only those ER visits that ended with discharge. To identify the ER visits that ended in hospitalization, we used a variable called “admission source” in the SID databases. Admission source identified as “emergency room” indicates that the hospital admission came from the ER – i.e., the ER visit ended in hospitalization. For each combination of age group, endpoint and county, we summed the ER visits that ended with discharge and those that resulted in hospitalization.

- The data year varies across the states from 2011 to 2014 (see Exhibit D-7); we assumed that ER visit rates are reasonably constant across these three years and consider them as 2014 rates.
- Instead of using HCUPnet/NIS and NHDS in the last step as described for hospitalizations, we used HCUPnet/NEDS and the National Ambulatory Medical Care Survey (NAMCS) to calculate ER visit rates for states without discharge level or state level data.

Exhibit D-8 shows the resulting average national rates of asthma emergency room visits by age group.

**Exhibit D-8. National Emergency Room Visit Rates for Asthma, by Age Group**

ER Category	ICD-9 Codes	ER Visit Rate (visits per 100 people per year)								
		Age 0-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Asthma	493	0.959	0.601	0.556	0.538	0.552	0.408	0.331	0.368	0.350

## Nonfatal Heart Attacks

The relationship between short-term particulate matter exposure and heart attacks was quantified in case-crossover analyses by Peters et al (2001), Pope et al. (2006), and Sullivan et al. (2005). The study population was selected from heart attack survivors in a medical clinic. Therefore, the applicable population to apply to the C-R function is all individuals surviving a heart attack in a given year. Several data sources are available to estimate the number of heart attacks per year. For example, several cohort studies have reported estimates of heart attack incidence rates in the specific populations under study. However, these rates depend on the specific characteristics of the populations under study and may not be the best data to extrapolate nationally. The American Heart Association reports approximately 785,000 new heart attacks per year (Roger et al., 2012). Exclusion of heart attack deaths reported by CDC Wonder yields approximately 575,000 nonfatal cases per year.

An alternative approach to the estimation of heart attack rates is to use data from the HCUP, assuming that all heart attacks that are not instantly fatal will result in a hospitalization. According to the HCUPnet, in 2014 there were approximately 608,795 hospitalizations due to heart attacks (acute myocardial infarction: ICD-9 code of 410, primary diagnosis).<sup>50</sup> We used county-level hospitalization rates over estimates extrapolated from cohort studies because the former is part of a nationally representative survey with a larger sample size, which is intended to provide reliable national estimates. The hospitalization section above describes the detailed

<sup>50</sup> Source: Online query on HCUPnet website (AHRQ 2012), accessed 1-13-2012  
<http://hcupnet.ahrq.gov/HCUPnet.app/HCUPnet.jsp?Id=53F290DC050F1296&Form=SelLAY&GoTo=MAINSEL&JS=Y>

procedure for developing the incidence rates for hospitalization of AMI. As additional information is provided regarding the American Heart Association methodology, we will evaluate the usefulness of this estimate of heart attack incidence.

Rosamond et al. (1999) reported 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, therefore, applied a factor of 0.93 to the count of hospitalizations to estimate the number of nonfatal heart attacks per year. Note that we did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

Exhibit D-9 presents the national nonfatal heart attack incidence rates around year 2007 by age group (Note: county-level rates around year 2007 are used in COBRA).

**Exhibit D-9. Nonfatal Heart Attack Rates by Age Group**

ER Category	ICD-9 Codes	Nonfatal Heart Rate by Age Group (admissions per 100 people per year)								
		Age 0-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Acute Myocardial Infarction, Nonfatal	410	0.000	0.000	0.002	0.010	0.068	0.202	0.380	0.575	0.921

### Other Acute Effects

For many of the minor effect studies, baseline rates from a single study are often the only source of information, and we assume that these rates hold for locations in the U.S. The use of study-specific estimates is likely to increase the uncertainty around the estimate because they are often estimated from a single location using a relatively small sample. These endpoints include: acute bronchitis, upper respiratory symptoms, and lower respiratory symptoms. Exhibit D-10 presents a summary of these baseline rates.



**Exhibit D-10. Selected Acute Incidence (Cases / Person-Year)**

Endpoint	Age	Parameter	Rate	Source
Acute Bronchitis	8-12	Incidence	0.043	(American Lung Association, 2002, Table 11)
Lower Respiratory Symptoms (LRS)	7-14	Incidence	430.483	(Schwartz et al., 1994, Table 2)
Minor Restricted Activity Days (MRAD)	18-64	Incidence	7.8	(Ostro & Rothschild, 1989, p. 243)
Work Loss Day (WLD)	18-64	Incidence	2.172	(Adams, Hendershot, & Marano, 1999, Table 41; U.S. Bureau of the Census, 1997)

### **Acute Bronchitis**

The annual rate of acute bronchitis for children ages 5 to 17 was obtained from the American Lung Association (2002). The authors reported an annual incidence rate per person of 0.043, derived from the 1996 National Health Interview Survey.

### **Lower Respiratory Symptoms**

Lower respiratory symptoms (LRS) are defined as two or more of the following: cough, chest pain, phlegm, and wheeze. The proposed yearly incidence rate for 100 people, 43.8, is based on the percentiles in Schwartz et al (1994, Table 2). The authors did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated per person per day values are 10th = 0 percent, 25th = 0 percent, 50th = 0 percent, 75th = 0.29 percent, and 90th = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence per person per day is zero up to the 75th percentile, a constant 0.29 percent between the 75th and 90th percentiles, and a constant 0.34 percent between the 90th and 100th percentiles. Alternatively, assuming a linear slope between the 50th and 75th, 75th and 90th, and 90th to 100th percentiles, the estimated mean incidence rate per person per day is 0.12 percent.<sup>51</sup> We used the latter approach in this analysis, and then multiplied by 100 and by 365 to calculate the incidence rate per 100 people per year.

### **Minor Restricted Activity Days (MRAD)**

Ostro and Rothschild (1989, p. 243) provide an estimate of the annual incidence rate of MRADs (7.8). We multiplied this estimate by 100 to get an annual rate per 100 people.

### **Work Loss Days**

The yearly work-loss-day incidence rate per 100 people is based on estimates from the 1996 National Health Interview Survey (Adams et al., 1999, Table 41). They reported a total annual work loss days of 352 million for individuals ages 18 to 65. The total population of individuals of this age group in 1996 (162 million) was obtained from (U.S. Bureau of the Census, 1997).

<sup>51</sup> For example, the 62.5<sup>th</sup> percentile would have an estimated incidence rate per person per day of 0.145 percent.

The average annual rate of work loss days per individual (2.17) was multiplied by 100 to obtain the average yearly work-loss-day rate of 217 per 100 people.

## Asthma-Related Health Effects

Several studies have examined the impact of air pollution on asthma development or exacerbation in the asthmatic population. Many of the baseline incidence rates used in the C-R functions are based on study-specific estimates. The baseline rates for the various endpoints are described below and summarized in Exhibit D-11.

**Exhibit D-11. Asthma-Related Health Effects Rates**

Endpoint	Age	Parameter <sup>a</sup>	Rate	Source
Asthma Exacerbation, Cough	6-18	Incidence	24.46	
		Prevalence	14.50%	
Asthma Exacerbation, Shortness of Breath	6-18	Incidence	13.51	(Ostro, Lipsett, Mann, Braxton-Owens, & White, 2001, p. 202) <sup>b</sup>
		Prevalence	7.40%	
Asthma Exacerbation, Wheeze	6-18	Incidence	27.74	
		Prevalence	17.3%	
Asthma	6-17	Prevalence	10.70%	(American Lung Association, 2010, Table 7) <sup>c</sup>
	18	Prevalence	7.19%	
Upper Respiratory Symptoms (URS)	9-11	Incidence	124.79	(Pope, Dockery, Spengler, & Raizenne, 1991, Table 2)

<sup>a</sup> The incidence rate is the number of cases per person per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

<sup>b</sup> the rates in the study were for African American children of ages 8-13. We apply it to children aged 6-18 to match what was used in the selected epidemiological studies.

<sup>c</sup> The American Lung Association (2010, Table 7) estimates asthma prevalence for children 5-17 at 10.70% and for adults ages 18-44 at 7.19% (based on data from the 2008 National Health Interview Survey).

## Appendix E: Population Forecasts

To estimate the change in population exposure to air pollution, we use projections based on US Census of Population and Housing 2010 and forecasting models developed by Woods & Poole (2015). The Woods and Poole (WP) database contains county-level projections of population by age, sex, ethnicity, and race out to 2050. Projections in each county are determined simultaneously with every other county in the United States to take into account patterns of economic growth and migration. The sum of growth in county-level populations is constrained to equal a previously determined national population growth, based on Bureau of Census estimates. The projection years used for COBRA are 2016, 2023, and 2028.

According to WP, linking county-level growth projections together and constraining to a national-level total growth avoids potential errors introduced by forecasting each county independently. County projections are developed in a four-stage process. First, national-level variables such as income, employment, and populations are forecasted. Second, employment projections are made for 172 economic areas defined by the Bureau of Economic Analysis, using an “export-base” approach, which relies on linking industrial sector production of non-locally consumed production items, such as outputs from mining, agriculture, and manufacturing with the national economy. The export-based approach requires estimation of demand equations or calculation of historical growth rates for output and employment by sector. Third, population is projected for each economic area based on net migration rates derived from employment opportunities and following a cohort component method based on fertility and mortality in each area. Fourth, employment and population projections are repeated for counties, using the economic region totals as bounds. The age, sex, ethnicity, and race distributions for each region or county are determined by aging the population by single year of age by sex and race for each year through 2050 based on historical rates of mortality, fertility, and migration.

## Appendix F: Economic Value of Health Effects

This Appendix presents the mean estimate of the unit values used in this analysis. Exhibit F-1 lists these unit values for 2028, Exhibit F-2 lists these unit values for 2023 and Exhibit F-3 lists these unit values for 2016.

**Exhibit F-1. Unit Values for Economic Valuation of Health Endpoints in 2028 (2017 \$)**

Health Endpoint	Age Range	Unit Value (2028 Income Level)	
		3% DR	7% DR
Mortality <sup>a</sup>	25 - 99	\$10,040,738	\$8,943,125
Infant Mortality <sup>b</sup>	0 - 0	\$11,191,541	\$11,191,541
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	0 - 24	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	25 - 44	\$52,999	\$49,415
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	45 - 54	\$59,550	\$55,281
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	55 - 64	\$156,951	\$142,483
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	65 - 99	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	0 - 24	\$192,048	\$192,048
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	25 - 44	\$205,873	\$204,425
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	45 - 54	\$212,424	\$210,291
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	55 - 64	\$309,825	\$297,494
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	65 - 99	\$192,048	\$192,048
HA, All Cardiovascular (less AMI)	18 - 64	\$47,652	\$47,652
HA, All Cardiovascular (less AMI)	65 - 99	\$44,727	\$44,727
HA, All Respiratory	65 - 99	\$37,569	\$37,569
HA, Asthma	0 - 17	\$17,707	\$17,707
HA, Chronic Lung Disease	18-64	\$23,362	\$23,362
Asthma ER Visit (Smith et al. 1997)	0 - 99	\$547	\$547
Asthma ER Visit (Stanford et al. 1999)	0 - 99	\$457	\$457
Acute Bronchitis	8 - 12	\$556	\$556
Lower Resp. Symptoms	7 - 14	\$24	\$24
Upper Resp. Symptoms	9 - 11	\$39	\$39
MRAD	18 - 64	\$79	\$79
Work Loss Days	18 - 64	\$178	\$178
Asthma Exacerbation (Cough, Shortness of Breath, or Wheeze)	6 - 18	\$67	\$67

NOTE: <sup>a</sup> Mortality value after adjustment for 20-year lag.

<sup>b</sup> Infant mortality value is not adjusted for 20-year lag.

<sup>c</sup> Based on Russell (1998)

<sup>d</sup> Based on Wittels (1990)

**Exhibit F-2. Unit Values for Economic Valuation of Health Endpoints in 2023 (2017 \$)**

<b>Health Endpoint</b>	<b>Age Range</b>	<b>Unit Value (2023 Income Level)</b>	
		<b>3% DR</b>	<b>7% DR</b>
Mortality <sup>a</sup>	25 - 99	\$9,748,682	\$8,682,996
Infant Mortality <sup>b</sup>	0 - 0	\$10,866,012	\$10,866,012
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	0 - 24	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	25 - 44	\$52,999	\$49,415
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	45 - 54	\$59,550	\$55,281
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	55 - 64	\$156,951	\$142,483
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	65 - 99	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	0 - 24	\$192,048	\$192,048
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	25 - 44	\$205,873	\$204,425
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	45 - 54	\$212,424	\$210,291
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	55 - 64	\$309,825	\$297,494
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	65 - 99	\$192,048	\$192,048
HA, All Cardiovascular (less AMI)	18 - 64	\$47,581	\$47,581
HA, All Cardiovascular (less AMI)	65 - 99	\$44,642	\$44,642
HA, All Respiratory	65 - 99	\$37,463	\$37,463
HA, Asthma	0 - 17	\$17,655	\$17,655
HA, Chronic Lung Disease	18-64	\$23,294	\$23,294
Asthma ER Visits (Smith et al. 1997)	0 - 99	\$547	\$547
Asthma ER Visits (Stanford et al. 1999)	0 - 99	\$457	\$457
Acute Bronchitis	8 - 12	\$550	\$550
Lower Resp. Symptoms	7 - 14	\$24	\$24
Upper Resp. Symptoms	9 - 11	\$38	\$38
MRAD	18 - 64	\$78	\$78
Work Loss Days	18 - 64	\$178	\$178
Asthma Exacerbation (Cough, Shortness of Breath, or Wheeze)	6 - 18	\$66	\$66

NOTE: <sup>a</sup> Mortality value after adjustment for 20-year lag.

<sup>b</sup> Infant mortality value is not adjusted for 20-year lag.

<sup>c</sup> Based on Russell (1998)

<sup>d</sup> Based on Wittels (1990)

**Exhibit F-3. Unit Values for Economic Valuation of Health Endpoints in 2016 (2017 \$)**

Health Endpoint	Age Range	Unit Value (2017 Income Level)	
		3% DR	7% DR
Mortality <sup>a</sup>	25 - 99	\$9,480,981	\$8,444,559
Infant Mortality <sup>b</sup>	0 - 0	\$10,567,629	\$10,567,629
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	0 - 24	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	25 - 44	\$52,999	\$49,415
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	45 - 54	\$59,550	\$55,281
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	55 - 64	\$156,951	\$142,483
Acute Myocardial Infarction, Nonfatal <sup>c</sup>	65 - 99	\$39,174	\$37,038
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	0 - 24	\$192,048	\$192,048
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	25 - 44	\$205,873	\$204,425
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	45 - 54	\$212,424	\$210,291
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	55 - 64	\$309,825	\$297,494
Acute Myocardial Infarction, Nonfatal <sup>d</sup>	65 - 99	\$192,048	\$192,048
HA, All Cardiovascular (less AMI)	18 - 64	\$47,480	\$47,480
HA, All Cardiovascular (less AMI)	65 - 99	\$44,524	\$44,524
HA, All Respiratory	65 - 99	\$37,316	\$37,316
HA, Asthma	0 - 17	\$17,582	\$17,582
HA, Chronic Lung Disease	18-64	\$23,200	\$23,200
Asthma ER Visits (Smith et al. 1997)	0 - 99	\$547	\$547
Asthma ER Visits (Stanford et al. 1999)	0 - 99	\$457	\$457
Acute Bronchitis	8 - 12	\$550	\$550
Lower Resp. Symptoms	7 - 14	\$24	\$24
Upper Resp. Symptoms	9 - 11	\$38	\$38
MRAD	18 - 64	\$77	\$77
Work Loss Days	18 - 64	\$178	\$178
Asthma Exacerbation (Cough, Shortness of Breath, or Wheeze)	6 - 18	\$65	\$65

NOTE: <sup>a</sup> Mortality value after adjustment for 20-year lag.

<sup>b</sup> Infant mortality value is not adjusted for 20-year lag.

<sup>c</sup> Based on Russell (1998)

<sup>d</sup> Based on Wittels (1990)

## **Selecting Unit Values for Monetizing Health Endpoints**

The appropriate economic value for a change in a health effect depends on whether the health effect is viewed ex ante (before the effect has occurred) or ex post (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health effects by a small amount for a large population. The appropriate economic measure is therefore ex ante WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk.

For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is \$100, then the WTP for an avoided statistical premature mortality amounts to \$1 million ( $\$100/0.0001$  change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect. For example, for the valuation of hospital admissions EPA used the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These COI estimates generally understate the true value of reductions in risk of a health effect, because, while they reflect the direct expenditures related to treatment, they omit the value of avoiding the pain and suffering from the health effect itself.

### *Updating Values for Inflation*

The studies based on which the unit values were developed report estimates for a range of years prior to 2017. To allow for the effect of inflation, we have adjusted these values to reflect prices in 2017\$. Because some functions are based on willingness to pay to avoid illness, while others are based on cost of illness and/or lost wages, three different inflation indices are used. These are the All Goods Index, the Medical Cost Index, and the Wage Index, respectively. Exhibit F-4 summarizes the types of inflation indices and their sources used to adjust different types of unit values in COBRA.

**Exhibit F-4. Type of Inflation Index Used for Adjust Unit Values for Health Effects Endpoints**

Index	Source	Health Effects Endpoints
All Goods Index	Bureau of Labor Statistics' (BLS) Consumer Price Index (CPI)	Acute Bronchitis Asthma Exacerbation Lower Respiratory Symptoms Mortality Minor Restricted Activity Days Upper Respiratory Symptoms
Medical Cost Index	BLS/CPI	Acute Myocardial Infarction Emergency Room Visits Hospital Admissions
Wage Index	BLS Employment Cost Index for private industry workers, 2001-2015	Acute Myocardial Infarction Hospital Admissions

### ***Growth in Unit Values Reflecting Growth in National Income***

The unit value estimates reflect expected growth in real income over time. This is consistent with economic theory, which argues that WTP for most goods (such as health risk reductions) will increase if real incomes increase. There is substantial empirical evidence that the income elasticity of WTP for health risk reductions is positive, although there is uncertainty about its exact value (and it may vary by health effect). Although one might assume that the income elasticity of WTP is unit elastic (e.g., a 10 percent higher real income level implies a 10 percent higher WTP to reduce health risks), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefits estimates in two ways: through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and through differences in income between study populations and the affected populations at a particular time. Following the analysis in the 2006 PM<sub>2.5</sub> NAAQS regulatory impact assessment (U.S. EPA, 2006), we have focused on the former.

The income adjustment in COBRA follows the approach used by EPA (2005, p. 4-17), who adjusted the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, EPA applied estimates derived from cross-sectional studies.<sup>52</sup> The available income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and changes in WTP. As a result, EPA (2005, p. 4-18) used different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality (see Exhibit F-5).

<sup>52</sup> Details of the procedure can be found in Kleckner and Neumann 1999.



#### Exhibit F-5. Elasticity Values Used to Account for National Income Growth

Benefit Category	Central Elasticity Estimate
Minor Health Effect	0.15
Severe & Chronic Health Effects	0.45
Premature Mortality	0.40

In addition to elasticity estimates, projections of populations and real gross domestic product (GDP) are needed to adjust benefits to reflect real per capita income growth. COBRA uses population and GDP projections developed by EPA, which are described in EPA (2005, p. 4-17). To estimate national population growth rates for the years between 1990 and 1999, EPA used national population estimates U.S. Census Bureau (Hollman, Mulder, & Kallan, 2000). These population estimates are based on an application of a cohort-component model to 1990 U.S. Census data projections (U.S. Bureau of the Census, 2000). For the years between 2000 and 2010, EPA applied growth rates based on the U.S. Census Bureau projections to the U.S. Census estimate of national population in 2000. EPA used projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010, and projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's (2000) for the years 2010 to 2025.

Using the method outlined in Kleckner and Neumann (1999) and the population and income data described above, EPA (2005, p. 4-18) calculated WTP adjustment factors for each of the elasticity estimates. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) are adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor.

Note that because of a lack of data on the dependence of COI on income, and a lack of data on projected growth in average wages, no adjustments are made to benefits estimates based on the COI approach or to work loss days benefits estimates. This lack of adjustment would tend to result in an under-prediction of benefits in future years, because it is likely that increases in real U.S. income would also result in increased COI (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher).

#### **Valuation Pooling**

In some cases there are multiple valuations available for a health effect, with no one valuation clearly superior to another. In such cases we pooled valuations in COBRA.

- Smith et al. (1997) and Stanford et al. (1999) both evaluate asthma ER visits using COI. We assign equal weight to each study (i.e., 0.5) and COBRA will then use the weighted average to value ER visit.
- To value Acute Myocardial Infarction, we pool Russell (1998) and Wittels (1990) by assigning equal weight (i.e., 0.5) to each.

- To value respiratory hospitalization, we sum across non-overlapping respiratory hospitalization effects, i.e., Asthma HA (age 0-17), Chronic Lung Disease HA (age 18-64), All Respiratory HA (age 65-99).
- Similarly, we sum across non-overlapping cardiovascular hospitalization effects, i.e., we sum the value for cardiovascular less AMI hospitalization for ages 18-64 and that for ages 65+.

## Valuing Premature Mortality

To estimate the monetary value of risk change in premature death, we used the “value of statistical lives” saved (VSL) approach, which is a summary measure for the value of small changes in mortality risk for a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable monetary value of preventing premature mortality. Based on 26 published studies,<sup>53</sup> the mean value of avoiding one statistical death is estimated to be roughly \$9.5 million in 2016, \$9.7 million in 2023, and \$10 million in 2028 (all values are in 2017\$ at the 2016, 2023, and 2028 income levels, respectively).

There are a number of uncertainties in this estimate. The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. An ideal benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual’s WTP to improve one’s own chances of survival plus WTP to improve other individuals’ survival rates.

The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward and how individuals value these changes. Each individual’s survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual’s survival. This probability shift will differ across individuals because survival curves depend on such characteristics as age, health state, and the current age to which the individual is likely to survive.

There are other potentially important factors that go beyond the scope of this discussion. For additional details, EPA (2005, p. 4-57) has an in-depth discussion of the uncertainties underlying mortality valuation.

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<sup>53</sup> These 26 studies have been identified in the Section 812 Reports to Congress as “applicable to policy analysis.” This represents an intermediate value from a variety of estimates, and it is a value EPA has frequently used in Regulatory Impact Analyses (RIAs) as well as in the Section 812 Retrospective and Prospective Analyses of the Clean Air Act.

### ***Present Discounted Value of Avoiding Future Mortality***

The delay, or lag, between changes in PM<sub>2.5</sub> exposures and changes in mortality rates is not precisely known. The current scientific literature on adverse health effects, such as those associated with PM<sub>2.5</sub> (e.g., smoking-related disease), and the difference in the effect size estimated in chronic exposure studies versus daily mortality studies, suggests that it is likely that not all cases of avoided premature mortality associated with a given incremental reduction in PM<sub>2.5</sub> exposure would occur in the same year as the exposure reduction.

Current EPA benefits analyses (U.S. EPA, 2006, p. 5-21) assume a 20-year lag structure, with 30 percent of premature deaths occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in PM<sub>2.5</sub>, and 20 percent occurring evenly over years 6 to 20 after the reduction in PM<sub>2.5</sub>. It should be noted that the selection of a 20-year lag structure is not directly supported by any PM<sub>2.5</sub>-specific literature. Rather, it is intended to be a best guess at the appropriate time distribution of avoided cases of PM<sub>2.5</sub>-related mortality. As noted by EPA, the distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Finally, it is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths.

Specifying the lag is important because people are generally willing to pay more for something now than for the same thing later. They would for example, be willing to pay more for a reduction in the risk of premature death in the same year as exposure is reduced than for that same risk reduction to be received the following year. This time preference for receiving benefits now rather than later is expressed by discounting benefits received later. There is an ongoing discussion within the federal government about the choice of a discount rate in this context: a 3% discount rate is recommended by EPA, while a 7% is recommended by OMB. Therefore, the users now have the ability to specify the discount rate—3% or 7%—for a COBRA session. Following EPA's Guidelines for Preparing Economic Analyses (U.S. EPA, 2010a), COBRA users are recommended to calculate monetized health benefits using both discount rates and to evaluate whether (and to what extent) the overall outcome of their analysis is affected by the choice of discount rate.

Following EPA (2006, p. 5-21), COBRA assumes that some of the incidences of premature mortality related to PM<sub>2.5</sub> exposures occur in a distributed fashion over the 20 years following exposure. To take this into account in the valuation of reductions in premature mortality, we applied an annual 3 percent discount rate to the value of premature mortality occurring in future years. Note that this lag adjustment does not apply to infant mortality, because Woodruff et al. (1997) estimate the number of infant deaths occurring in the same year as the emissions change.

### ***Valuing Non-Fatal Myocardial Infarction***

We are not able to identify a suitable WTP value for reductions in the risk of non-fatal heart attacks. Instead, we have used a cost-of-illness unit value with two components: the direct medical costs and the opportunity cost (lost earnings) associated with the illness event. Because

the costs associated with a heart attack extend beyond the initial event itself, we considered costs incurred over several years. For opportunity costs, we used values derived from Cropper and Krupnick (Cropper & Sussman, 1990), originally used in the 812 Retrospective Analysis of the Clean Air Act (U.S. EPA, 1997). For the direct medical costs, we found three possible sources in the literature.

Wittels et al. (1990) estimated expected total medical costs of myocardial infarction over five years to be \$51,211 (in 1986\$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Using the CPI, the Wittels et al. estimate is \$192,048 in year 2017\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes and prices (using “knowledgeable cardiologists” as consultants).

The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors noted that the average length of hospitalization for acute myocardial infarction has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay may have decreased from 1983 to the present. The average length of stay for ICD code 410 (myocardial infarction) in 2009 is 4.9 days (Agency for Healthcare Research and Quality, 2010). However, this may include patients who died in the hospital (not included among our non-fatal cases), whose length of stay was therefore substantially shorter than it would be if they hadn’t died.

Eisenstein et al. (2001) estimated 10-year costs of \$44,663, in 1997\$, or \$87,102 in 2017\$ for myocardial infarction patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal myocardial infarction of \$15,540 (in 1995\$), and \$1,051 annually thereafter. Converting to year 2017\$, that would be \$39,174 (3% discount rate) and \$37,038 (7% discount rate) for a 5-year period.

As seen in Exhibit F-6, the three different studies provided significantly different values. We have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used a simple average of the two estimates for medical costs that similarly cover a 5-year period (i.e., assign a subjective weight of 0.5 to each estimate). We added this to the 5-year opportunity cost estimate. Exhibit F-7 gives the resulting estimates.

**Exhibit F-6 Summary of Studies Valuing Reduced Incidences of Myocardial Infarction**

Study	Direct Medical Costs (2017 \$, 3% DR)	Direct Medical Costs (2017 \$, 7% DR)	Over an x-year period, for x =
Wittels et al., 1990 <sup>a</sup>	\$192,048	\$192,048	5
Russell et al., 1998	\$39,174	\$37,038	5
Eisenstein et al., 2001	\$87,102	\$87,102	10

<sup>a</sup> Wittels et al. did not appear to discount costs incurred in future years.

**Exhibit F-7. Estimated Costs Over a 5-Year Period of a Non-Fatal Myocardial Infarction**

Age Group	Opportunity Cost (2017 \$, 3% DR) <sup>a</sup>	Opportunity Cost (2017 \$, 7% DR) <sup>a</sup>	Medical Cost (2017 \$, 3% DR) <sup>b</sup>	Medical Cost (2017 \$, 7% DR) <sup>b</sup>	Total Cost (2017 \$, 3% DR)	Total Cost (2017 \$, 7% DR)
0 - 24	\$0	\$0	\$115,611	\$114,543	\$115,611	\$114,543
25-44	\$13,273	\$11,883	\$115,611	\$114,543	\$128,884	\$126,426
45 - 54	\$19,564	\$17,516	\$115,611	\$114,543	\$135,175	\$132,059
55 - 65	\$113,085	\$101,245	\$115,611	\$114,543	\$228,696	\$215,788
> 65	\$0	\$0	\$115,611	\$114,543	\$115,611	\$114,543

<sup>a</sup> From Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings, adjusted from 1977\$ to 2017\$ using CPI.

<sup>b</sup> An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998). Note that Wittels et al. appears not to have used discounting in deriving a 5-year cost; Russell et al. estimated first-year direct medical costs and annual costs thereafter.

## Valuing Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), as well as WTP of the affected individual, as well as of that of relatives, friends, and associated caregivers, to avoid the pain and suffering.<sup>54</sup>

Because medical expenditures are to a significant extent shared by society, via medical insurance, Medicare, etc., the medical expenditures actually incurred by the individual are likely to be less than the total medical cost to society. The total value to society of an individual's avoidance of hospital admission, then, might be thought of as having two components: (1) the cost of illness (COI) to society, including the total medical costs plus the value of the lost productivity, as well as (2) the WTP of the individual, as well as that of others, to avoid the pain and suffering resulting from the illness.

In the absence of estimates of social WTP to avoid hospital admissions for specific illnesses (components 1 plus 2 above), estimates of total COI (component 1) are typically used as conservative (lower bound) estimates. Because these estimates do not include the value of avoiding the pain and suffering resulting from the illness (component 2), they are biased downward. Some analyses adjust COI estimates upward by multiplying by an estimate of the ratio of WTP to COI, to better approximate total WTP. Other analyses have avoided making this adjustment because of the possibility of over-adjusting – that is, possibly replacing a known

<sup>54</sup> Some people take action to avert the negative impacts of pollution. While the costs of successful averting behavior should be added to the sum of the health-endpoint-specific costs when estimating the total costs of pollution, these costs are not associated with any single health endpoint. It is possible that in some cases the averting action was not successful, in which case it might be argued that the cost of the averting behavior should be added to the other costs listed (for example, it might be the case that an individual incurs the costs of averting behavior and in addition incurs the costs of the illness that the averting behavior was intended to avoid). Because averting behavior is generally not taken to avoid a particular health problem (such as a hospital admission for respiratory illness), but instead is taken to avoid the entire collection of adverse effects of pollution, it does not seem reasonable to ascribe the entire costs of averting behavior to any single health endpoint. However, omission of these averting behavior costs will tend to bias the estimates downward.

downward bias with an upward bias. The COI values used in this benefits analysis will not be adjusted to better reflect the total WTP.

Following the method used in the §812 analysis (U.S. EPA, 1999), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. The median annual wages for 2017 were obtained from the U.S. Census Bureau, 2017 American Community Survey, 1-year estimates, Table B20017: “Median earnings in the past 12 months (in 2017 inflation-adjusted dollars) by sex by work experience in the past 12 months for the population 16 years and over with earnings in the past 12 months”. The median daily wage was calculated as the simple average of full-time male earnings (\$51,284) and full-time female earnings (\$41,453), divided by  $52 \times 5$ , resulting in an estimate of \$178.

For all hospital admissions endpoints available in this analysis, estimates of hospital charges and lengths of hospital stays were based on discharge statistics provided by the Agency for Healthcare Research and Quality’s Healthcare Utilization Project National Inpatient Sample (NIS) database (2007). The NIS is the largest inpatient care database in the United States, and it is the only national hospital database containing charge information on all patients. It contains data from a very large nationally representative sample of about eight million hospital discharges, and therefore provides the best estimates of mean hospital charges and mean lengths of stay available, with negligible standard errors. The sampling frame for the 2007 NIS is a sample of hospitals that comprises approximately 90 percent of all hospital discharges in the United States. Since the NIS is based on discharge samples, the discharge-level weight was used to weight discharges in order to produce national estimates. The principle diagnoses (based on ICD-9 codes) were used to define the health endpoints.

Since most pollution-related hospital admissions are likely unscheduled, the unit values of avoided hospital admissions used in COBRA are based solely on unscheduled hospitalizations. The total COI for an ICD-code-specific hospital stay lasting  $n$  days is estimated as the mean hospital charge plus  $n$  times the daily lost wage. The hospital admissions for which unit values are available in COBRA are given in Exhibit F-1.

Because of distortions in the market for medical services, the hospital charge may exceed “the cost of a hospital stay.” We use the example of a hospital visit to illustrate the problem. Suppose a patient is admitted to the hospital to be treated for an asthma episode. The patient’s stay in the hospital (including the treatments received) costs the hospital a certain amount. This is the hospital cost – i.e., the short-term expenditures of the hospital to provide the medical services that were provided to the patient during his hospital stay. The hospital then charges the payer a certain amount – the hospital charge. If the hospital wants to make a profit, is trying to cover costs that are not associated with any one particular patient admission (e.g., uninsured patient services), and/or has capital expenses (building expansion or renovation) or other long term costs, it may charge an amount that exceeds the patient-specific short term costs of providing services. The payer (e.g., the health maintenance organization or other health insurer) pays the

hospital a certain amount – the payment – for the services provided to the patient. The less incentive the payer has to keep costs down, the closer the payment will be to the charge. If, however, the payer has an incentive to keep costs down, the payment may be substantially less than the charge; it may still, however, exceed the short-term cost for services to the individual patient.

Although the hospital charge may exceed the short-term cost to the hospital of providing the medical services required during a patient’s hospital stay, cost of illness estimates based on hospital charges are still likely to understate the total social WTP to avoid the hospitalization in the first place, because the omitted WTP to avoid the pain and suffering is likely to be quite large.

### **Valuing Emergency Room Visits for Asthma**

To value asthma emergency room (ER) visits, we used a simple average of two estimates from the literature. The first estimate comes from Smith et al.(1997), who reported that there were approximately 1.2 million asthma-related ER visits made in 1987, at a total cost of \$186.5 million, in 1987\$. The average cost per visit was therefore \$155 in 1987\$, or \$547 in 2017\$. The second is from Stanford et al. (1999), who examined data from asthmatics from 1996-1997, and reported an average cost of \$457 (2017\$). We use a simple average of the two estimates, which yields a unit value of about \$502 (2017\$).

In comparing their study to Smith et al. (1997), Stanford et al. (1999) noted that the data used by Smith et al., “may not reflect changes in treatment patterns during the 1990s.” In addition, its costs are the costs to the hospital (or ER) for treating asthma rather than charges or payments by the patient and/or third party payer. Costs to the ER are probably a better measure of the value of the medical resources used up on an asthma ER visit.

### **Valuing Acute Symptoms and Illness Not Requiring Hospitalization**

Several acute symptoms and illnesses have been associated with air pollution, including acute bronchitis in children, upper and lower respiratory symptoms, and exacerbation of asthma (as indicated by one of several symptoms whose occurrence in an asthmatic generally suggests the onset of an asthma episode). In addition, several more general health endpoints which are associated with one or more of these acute symptoms and illnesses, such as minor restricted activity days and work loss days, have also been associated with air pollution.

### ***Valuing Acute Bronchitis in Children***

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore must be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

In previous benefits analyses, such as in the §812 Prospective analysis (U.S. EPA, 1999), acute bronchitis was valued at \$59.31 (in 2000 \$ and at 1990 income level). This is the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. For a more complete description of the derivation of this estimate, see Abt Associates (2000, p. 4-30).

A unit value of \$59.31 assumes that an episode of acute bronchitis lasts only one day. However, this is generally not the case. More typically, it can last for 6 or 7 days. We therefore made a simple adjustment, multiplying the original unit value of \$59.31 by 6. The unit value thus derived and used was \$356 in 2000 \$ and at 1990 income level ( $=\$59.31 \times 6$ ), \$544 in 2017\$.

### ***Valuing Upper Respiratory Symptoms (URS) in Children***

Willingness to pay to avoid a day of upper respiratory symptoms is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. (1991) as part of the complex of upper respiratory symptoms. Three contingent valuation studies have estimated WTP to avoid various morbidity symptoms that are either within the complex defined by Pope et al. (1991), or are similar to those symptoms. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Exhibit F-8.

The three individual symptoms listed in Exhibit F-8 that were identified as most closely matching those listed by Pope, et al. (1991) for upper respiratory symptoms are cough, head/sinus congestion, and eye irritation, corresponding to “wet cough,” “runny or stuffy nose,” and “burning, aching or red eyes,” respectively. A day of upper respiratory symptoms could consist of any one of the seven possible “symptom complexes” consisting of at least one of these three symptoms. We assumed that each of these seven complexes is equally likely.<sup>55</sup> The point estimate of WTP is just an average of the seven estimates of WTP for the different complexes.

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<sup>55</sup> With empirical evidence, we could presumably improve the accuracy of the probabilities of occurrence of each type of URS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable “default” assumption.



**Exhibit F-8. Median WTP Estimates and Derived Midrange Estimates (2017 \$)<sup>a</sup>**

Symptom <sup>b</sup>	Dickie et al. (1987)	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	7.11	30.84	-	18.87
Head/sinus congestion	8.30	33.22	15.46	18.87
Coughing	2.38	26.11	9.39	13.21
Eye irritation	-	29.64	-	29.64
Headache	0.00	47.46	0.00	18.87
Shortness of breath	8.33	-	19.93	9.42
Pain upon deep inhalation (PDI)	4.75	-	-	8.33
Wheeze	5.19	-	-	4.75
Coughing up phlegm	5.19	-	-	5.19
Chest tightness	11.88	-	-	11.88

<sup>a</sup> Values were inflated to 2017 \$ using CPI.

<sup>b</sup> All estimates are WTP to avoid one day of symptom. Midrange estimates were derived by IEc (1993).

<sup>c</sup> 10% trimmed mean.

### **Valuing Lower Respiratory Symptoms (LRS) in Children**

Schwartz et al. (1994, p. 1235) defined lower respiratory symptoms as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. To value this combination of symptoms, we used the same method as we did for upper respiratory symptoms. We chose those individual health effects that seem most consistent with lower respiratory symptoms, we derived all of the possible combinations of these symptoms, and then we valued these combinations.

The symptoms for which WTP estimates are available that reasonably match lower respiratory symptoms are: cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of lower respiratory symptoms could consist of any one of the 11 combinations of at least two of these four symptoms.<sup>56</sup> We assumed that each of the eleven types of lower respiratory

<sup>56</sup> Because cough is a symptom in some of the upper respiratory symptom clusters as well as some of the lower respiratory symptom clusters, there is the possibility of a very small amount of double counting – if the same individual were to have an occurrence of upper respiratory symptoms which included cough and an occurrence of lower respiratory symptoms which included cough *both on exactly the same day*. Because this is probably a very small probability occurrence, the degree of double counting is likely to be very minor. Moreover, because upper respiratory symptoms is applied only to asthmatics ages 9-11 (a very small population), the amount of potential double counting should be truly negligible.

symptoms is equally likely,<sup>57</sup> and the mean WTP is the average of the WTPs over all combinations.

### **Valuing Work Loss Days (WLDs)**

Willingness to pay to avoid the loss of one day of work was estimated by dividing median annual earnings (\$46,369 in 2017\$) by  $(52 \times 5)$ , which resulted a unit value of \$178 (2017\$). The median annual earnings for males and females were obtained from U.S. Census Bureau, 2017 American Community Survey, Table B20017: “Median earnings in the past 12 months (in 2010 inflation-adjusted dollars) by sex by work experience in the past 12 months for the population 16 years and over with earnings in the past 12 months.” Overall earnings were calculated as the simple average of full-time male earnings and full-time female earnings. Valuing the loss of a day’s work at the wages lost is consistent with economic theory, which assumes that an individual is paid exactly the value of his labor.

The use of the median rather than the mean, however, requires some comment. If all individuals in society were equally likely to be affected by air pollution to the extent that they lose a day of work because of it, then the appropriate measure of the value of a work loss day would be the mean daily wage. It is highly likely, however, that the loss of work days due to pollution exposure does not occur with equal probability among all individuals, but instead is more likely to occur among lower income individuals than among high income individuals. It is probable, for example, that individuals who are vulnerable enough to the negative effects of air pollution to lose a day of work as a result of exposure tend to be those with generally poorer health care. Individuals with poorer health care have, on average, lower incomes.

To estimate the average lost wages of individuals who lose a day of work because of exposure to PM<sub>2.5</sub> pollution, then, would require a weighted average of all daily wages, with higher weights on the low end of the wage scale and lower weights on the high end of the wage scale. Because the appropriate weights are not known, however, the median wage was used rather than the mean wage. The median is more likely to approximate the correct value than the mean because means are highly susceptible to the influence of large values in the tail of a distribution (in this case, the small percentage of very large incomes in the United States), whereas the median is not susceptible to these large values.

### **Valuing Minor Restricted Activity Days (MRADs)**

No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEC (1993) has derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is \$38.37 (1990\$), or after adjusting for inflation and income growth \$79 in 2028 (2028 income level and 2017\$), \$78 in 2023 (2023 income level and 2017\$), and \$77 in 2016 (2016 income level and 2017\$). Although Ostro and Rothschild (1989)

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<sup>57</sup> As with URS, if we had empirical evidence we could improve the accuracy of the probabilities of occurrence of each type of LRS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable “default” assumption.

estimated the relationship between  $PM_{2.5}$  and MRADs, rather than MRRADs (a component of MRADs), it is likely that most of the MRADs associated with exposure to  $PM_{2.5}$  are in fact MRRADs. For the purpose of valuing this health endpoint, then, we assumed that MRADs associated with  $PM_{2.5}$  exposure may be more specifically defined as MRRADs, and therefore used the estimate of mean WTP to avoid a MRRAD.

Any estimate of mean WTP to avoid a MRRAD (or any other type of restricted activity day other than WLD) will be somewhat arbitrary because the endpoint itself is not precisely defined. Many different combinations of symptoms could presumably result in some minor or less minor restriction in activity. Krupnick and Kopp (1988) argued that mild symptoms will not be sufficient to result in a MRRAD, so that WTP to avoid a MRRAD should exceed WTP to avoid any single mild symptom. A single severe symptom or a combination of symptoms could, however, be sufficient to restrict activity. Therefore WTP to avoid a MRRAD should, these authors argue, not necessarily exceed WTP to avoid a single severe symptom or a combination of symptoms. The “severity” of a symptom, however, is similarly not precisely defined; moreover, one level of severity of a symptom could induce restriction of activity for one individual while not doing so for another. The same is true for any particular combination of symptoms.

### ***Valuing Asthma Exacerbations***

Rowe and Chestnut (1986) surveyed asthmatics to estimate WTP for avoidance of a “bad asthma day,” as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut study. Using the mean of average WTP estimates for the four severity definitions of a “bad asthma day,” the asthma exacerbation could be valued at \$66.91 (2028 income level and 2017\$), \$66.11 (2023 income level and 2017\$), and \$65.43 (2016 income level and 2017\$) per incidence in 2028, 2023, and 2016, respectively.

## Appendix G: Instructions for Running COBRA Using Batch Files

Batch files can be used to submit emissions data representing multiple years or scenarios, so that the scenarios will run in succession without additional input from the user. This feature enables analysis of multiple years or scenarios in a single modeling step.

Running COBRA using batch files requires that users have some baseline understanding of what batch files are and how they function. Below, we describe how to (1) format emissions files for use in COBRA, (2) write a Windows command to conduct a single COBRA run, and (3) create and run a batch script for multiple COBRA runs.

### (1) Formatting emissions files for use in COBRA

COBRA requires that imported baseline emission files be in a specific format. To create properly formatted custom baseline and scenario emissions files, create a CSV file with the headings shown in Exhibit H-1. Each row of the file should correspond to a different source in a different county.

**Exhibit H-1. Format of Baseline and Scenario Emissions Files**

Column Heading	Description of Column Headings
typeindx <sup>1</sup>	Stack height associated with the emission
sourceindx <sup>2</sup>	Source index, which COBRA uses in its source receptor model
stid	FIPS state ID (e.g., the state FIPS code for Pennsylvania is 42)
cyid	FIPS county ID (e.g., the county FIPS code for Swarthmore, PA is 045)
TIER1 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 1 level
TIER2 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 2 level
TIER3 <sup>3</sup>	Emissions category to which emissions source corresponds, at tier 3 level
NO2	NO <sub>x</sub> emissions from each source in the baseline
SO2	SO <sub>2</sub> emissions from each source in the baseline
NH3	NH <sub>3</sub> emissions from each source in the baseline
PM25	Primary PM <sub>2.5</sub> emissions from each source in the baseline
VOC	VOC emissions from each source in the baseline

Notes:

<sup>1</sup> A table of typeindx and name is saved on your computer after installing COBRA in the default location, C:/Program Files/COBRA/input files/data dictionary/typeindx – stack heights.csv.

<sup>2</sup> A table of sourceindx and FIPS is saved on your computer after installing COBRA in the default location, C:/Program Files/COBRA/input files/data dictionary/SOURCEINDEX to FIPS crosswalk.csv

<sup>3</sup> A table of tier definitions and tier numbers is saved on your computer after installing COBRA in the default location, C:/Program Files/COBRA/input files/data dictionary/EmissionsTier Definitions.csv

### (2) Writing a Windows COBRA command to conduct a single COBRA run

After formatting the baseline emission and scenario emission files, advanced users can run COBRA from the Windows command prompt using the following syntax:

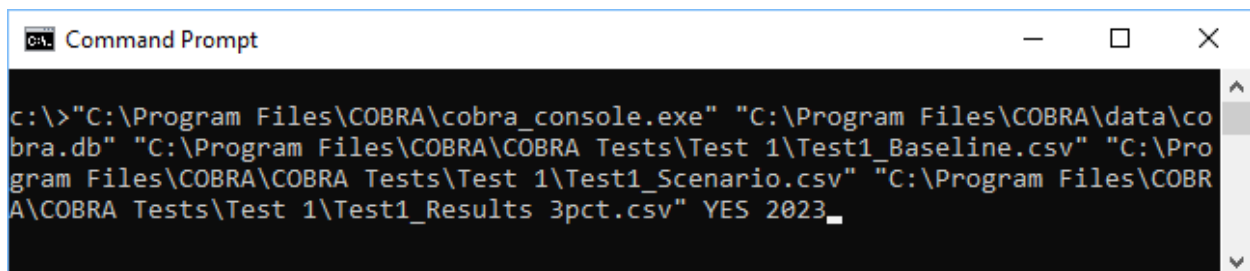
```
"C:\Program Files\COBRA\cobra_console.exe"  
"C:\Program Files\COBRA\data\cobra.db" A B C D E
```

where:

- A** is the file path for the emissions baseline in a CSV file
- B** is the file path for the emissions control scenario in a CSV file, formatted in the same way as the emissions baseline
- C** is the file path for where you would like the results CSV to be saved
- D** is YES or NO, where YES runs COBRA with a 3% discount rate and NO runs COBRA with a 7% discount rate
- E** is 2016, 2023, or 2028, which indicate the year for which population, incidence, and valuation files will be used

Below, we illustrate how one could use this syntax to run COBRA for a single scenario, named Test 1, where **A** = "C:\Program Files\COBRA\cobra\_console.exe", **B** = "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1\_Scenario.csv", **C** = "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1\_Results3pct.csv", and the user wants to run COBRA with a 7% discount rate for year 2023.

```
"C:\Program Files\COBRA\cobra_console.exe"  
"C:\Program Files\COBRA\data\cobra.db" "C:\Program  
Files\COBRA\COBRA Tests\Test 1\Test1_Baseline.csv"  
"C:\Program Files\COBRA\COBRA Tests\Test  
1\Test1_Scenario.csv" "C:\Program Files\COBRA\COBRA  
Tests\Test 1\Test1_Results3pct.csv" YES 2023
```



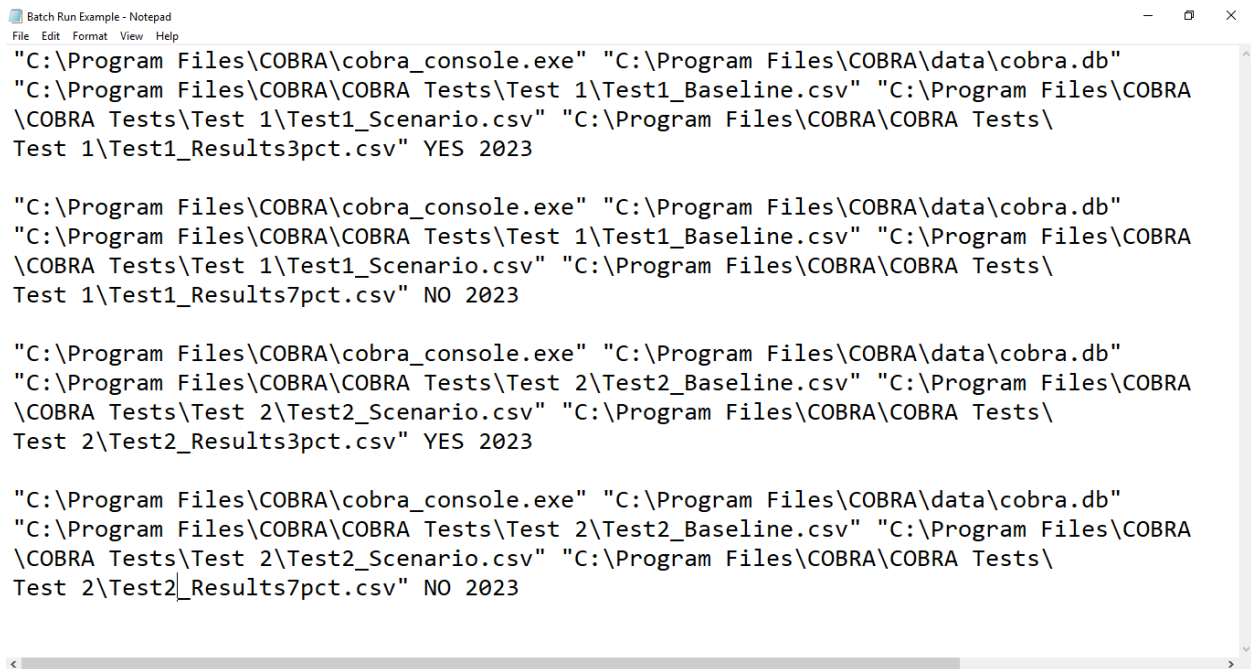
The screenshot shows a Windows Command Prompt window titled "Command Prompt". The command entered is: `c:\>"C:\Program Files\COBRA\cobra_console.exe" "C:\Program Files\COBRA\data\cobra.db" "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1_Baseline.csv" "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1_Scenario.csv" "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1_Results 3pct.csv" YES 2023_`. The cursor is at the end of the command.

### (3) Creating a batch script for multiple COBRA runs

Users can run multiple scenarios in succession without any additional input from the user by creating batch files that contain Windows commands for multiple COBRA runs. Below, we demonstrate how a user could use a batch file to run COBRA four times (1) "Test 1" with a 3% discount rate, (2) "Test 1" with a 7% discount rate, (3) "Test 2" with a 3% discount rate, and (4) "Test 2" with a 7% discount rate, all for year 2023. First, the user would create the properly formatted baseline and scenario emissions files, as described above. For this example, we assume that the following file paths are used:

- "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1\_Baseline.csv" = file path for the emissions baseline for Test 1
- "C:\Program Files\COBRA\COBRA Tests\Test 1\Test1\_Scenario.csv" = file path for the emissions scenario for Test 1
- "C:\Program Files\COBRA\COBRA Tests\Test 1\Test2\_Baseline.csv" = file path for the emissions baseline for Test 2
- "C:\Program Files\COBRA\COBRA Tests\Test 1\Test2\_Scenario.csv" = file path for the emissions scenario for Test 2

Then, the user would open a simple text editor (e.g. Notepad) and write the series of Windows COBRA commands that describe which baseline emissions, scenario emissions, and discount rate to use for each run, using the syntax described in (2).



```

Batch Run Example - Notepad
File Edit Format View Help
"C:\Program Files\COBRA\cobra_console.exe" "C:\Program Files\COBRA\data\cobra.db"
"C:\Program Files\COBRA\COBRA Tests\Test 1\Test1_Baseline.csv" "C:\Program Files\COBRA
\COBRA Tests\Test 1\Test1_Scenario.csv" "C:\Program Files\COBRA\COBRA Tests\
Test 1\Test1_Results3pct.csv" YES 2023

"C:\Program Files\COBRA\cobra_console.exe" "C:\Program Files\COBRA\data\cobra.db"
"C:\Program Files\COBRA\COBRA Tests\Test 1\Test1_Baseline.csv" "C:\Program Files\COBRA
\COBRA Tests\Test 1\Test1_Scenario.csv" "C:\Program Files\COBRA\COBRA Tests\
Test 1\Test1_Results7pct.csv" NO 2023

"C:\Program Files\COBRA\cobra_console.exe" "C:\Program Files\COBRA\data\cobra.db"
"C:\Program Files\COBRA\COBRA Tests\Test 2\Test2_Baseline.csv" "C:\Program Files\COBRA
\COBRA Tests\Test 2\Test2_Scenario.csv" "C:\Program Files\COBRA\COBRA Tests\
Test 2\Test2_Results3pct.csv" YES 2023

"C:\Program Files\COBRA\cobra_console.exe" "C:\Program Files\COBRA\data\cobra.db"
"C:\Program Files\COBRA\COBRA Tests\Test 2\Test2_Baseline.csv" "C:\Program Files\COBRA
\COBRA Tests\Test 2\Test2_Scenario.csv" "C:\Program Files\COBRA\COBRA Tests\
Test 2\Test2_Results7pct.csv" NO 2023

```

Then, the user would save this file a batch file (.bat). To run the batch file, the user would double click the appropriate “.bat” file and the COBRA commands will run automatically in the command prompt.

## Appendix H: References

- Abt Associates Inc. (2000). Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results. Bethesda, MD: Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- Adams, P. F., Hendershot, G. E., & Marano, M. A. (1999). Current Estimates from the National Health Interview Survey, 1996. *Vital Health Stat, 10(200)*, 1-212.
- Agency for Healthcare Research and Quality. Healthcare Cost and Utilization Project (HCUP), from <http://www.hcup-us.ahrq.gov/>
- Agency for Healthcare Research and Quality. (2007). Healthcare Cost and Utilization Project. National Inpatient Sample (NIS). Rockville, Maryland.
- Agency for Healthcare Research and Quality. (2012). HCUPnet, Healthcare Cost and Utilization Project, from Agency for Healthcare Research and Quality, Rockville, MD <http://hcupnet.ahrq.gov/>
- American Lung Association. (2002, September 2002). Trends in Morbidity and Mortality: Pneumonia, Influenza, and Acute Respiratory Conditions.
- American Lung Association. (2010). Trends in Asthma Morbidity and Mortality: American Lung Association Epidemiology and Statistics Unit Research and Program Services Division.
- American Lung Association. (2013, March 2013). Trends in COPD (Chronic Bronchitis and Emphysema): Morbidity and Mortality, from <http://www.lung.org/assets/documents/research/copd-trend-report.pdf>.
- Babin, S. M., Burkom, H. S., Holtry, R. S., Taberner, N. R., Stokes, L. D., Davies-Cole, J. O., . . . Lee, D. H. (2007). Pediatric patient asthma-related emergency department visits and admissions in Washington, DC, from 2001-2004, and associations with air quality, socio-economic status and age group. *Environ Health, 6*, 9.
- Bell, M. L., & Dominici, F. (2008). Effect Modification by Community Characteristics on the Short-term Effects of Ozone Exposure and Mortality in 98 U.S. Communities. *American Journal of Epidemiology, 167(8)*, 986-997.
- Bell, M. L., Ebisu, K., Peng, R. D., Walker, J., Samet, J., Zeger, S. L., & Dominici, F. (2008). Seasonal and Regional Short-term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999–2005. *American Journal of Epidemiology, 168(11)*, 1301-1310.
- Bobak, M., & Leon, D. A. (1999). The effect of air pollution on infant mortality appears specific for respiratory causes in the postneonatal period [see comments]. *Epidemiology, 10(6)*, 666-670.
- Bureau of Labor Statistics. Consumer Price Index, from <http://www.bls.gov/cpi/>
- Bureau of Labor Statistics. (2012, January 31, 2012). Employment Cost Index: Wages & Salaries: Private Industry Workers, from <http://research.stlouisfed.org/fred2/data/ECIWAG.txt>

- Conceicao, G. M., Miraglia, S. G., Kishi, H. S., Saldiva, P. H., & Singer, J. M. (2001). Air pollution and child mortality: a time-series study in Sao Paulo, Brazil. *Environ Health Perspect*, 109(Suppl 3), 347-350.
- Coursey, J. S., Schwab, D. J., Tsai, J. J., & Dragoset, R. A. (2011). Atomic Weights and Isotopic Compositions with Relative Atomic Masses, from <http://physics.nist.gov/PhysRefData/Compositions>
- Cropper, M. L., & Krupnick, A. J. (1990). The Social Costs of Chronic Heart and Lung Disease. Washington, DC: Resources for the Future.
- Cropper, M. L., & Sussman, F. G. (1990). Valuing Future Risks to Life. *Journal of Environmental Economics and Management*, 19, 160-174.
- Dekker, J. M., Crow, R. S., Folsom, A. R., Hannan, P. J., Liao, D., Swenne, C. A., & Schouten, E. G. (2000). Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. Atherosclerosis Risk In Communities. *Circulation*, 102(11), 1239-1244.
- DerSimonian, R., & Laird, N. (1986). Meta-Analysis in Clinical Trials. *Controlled Clinical Trials*, 7, 177-188.
- Dickie, M., & Gerking, S. (1987). Reconciling Averting Behavior and Contingent Valuation Benefit Estimates of Reducing Symptoms of Ozone Exposure (draft), as cited in Neumann, J.E., M. Dickie, and R.E. Unsworth. 1994.: Prepared by Industrial Economics. Prepared for Jim DeMocker, U.S. EPA, Office of Air and Radiation.
- Dockery, D. W., Cunningham, J., Damokosh, A. I., Neas, L. M., Spengler, J. D., Koutrakis, P., Speizer, F. E. (1996). Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. *Environmental Health Perspectives*, 104(5), 500-505.
- Dockery, D. W., Speizer, F. E., Stram, D. O., Ware, J. H., Spengler, J. D., & Ferris, B. G., Jr. (1989). Effects of Inhalable Particles on Respiratory Health of Children. *Am Rev Respir Dis*, 139, 587-594.
- E.H. Pechan & Associates Inc. (1994). Development of the OPPE Particulate Program Implementation Evaluation System. Springfield, VA: Prepared for Christopher Knopes, U.S. Environmental Protection Agency, Office of Policy, Planning and Evaluation.
- E.H. Pechan & Associates Inc. (1997). Integrated Ozone and Particulate Matter and Regional Haze Costs Analysis: Methodology and Results. Prepared for Innovative Strategies and Economics Group, Office of Air Quality Planning and Standards, US. Environmental Protection Agency. Research Triangle Park, NC.
- Eisenstein, E. L., Shaw, L. K., Anstrom, K. J., Nelson, C. L., Hakim, Z., Hasselblad, V., & Mark, D. B. (2001). Assessing the clinical and economic burden of coronary artery disease: 1986-1998. *Med Care*, 39(8), 824-835.



- GeoLytics Inc. (2002). CensusCD 2000 Long Form Profile Release 1.0.
- Glad, J.A., L.L. Brink, E.O. Talbott, P.C. Lee, X. Xu, M. Saul, and J. Rager. 2012. The Relationship of Ambient Ozone and PM2.5 Levels and Asthma Emergency Department Visits: Possible Influence of Gender and Ethnicity. *Archives of Environmental & Occupational Health*. Vol 62 (2): 103-108.
- Gold, D. R., Litonjua, A., Schwartz, J., Lovett, E., Larson, A., Nearing, B., and R. Verrier. (2000). Ambient pollution and heart rate variability. *Circulation*, 101(11), 1267-1273.
- Grabow, M. L., Spak, S. N., Holloway, T., Stone, B., Mednick, A. C., & Patz, J. A. (2012). Air quality and exercise-related health benefits from reduced car travel in the midwestern United States. *Environ Health Perspect*, 120(1), 68-76. doi: 10.1289/ehp.1103440
- Greene, W. H. (1997). *Econometric Analysis*. Upper Saddle River, NJ: Prentice Hall.
- Hiemstra, P. (2012). Package ‘automap’: Automatic interpolation package. Version 1.0-12. For R version 2.14.1 (2011-12-22) Copyright (C) 2011 The R Foundation for Statistical Computing ISBN 3-900051-07-0.
- Hollman, F. W., Mulder, T. J., & Kallan, J. E. (2000). Methodology and Assumptions for the Population Projections of the United States: 1999 to 2100. Washington, D.C.: Population Projections Branch, Population Division, U.S. Census Bureau, Department of Commerce.
- Industrial Economics Incorporated (IEc). (1993). Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review.
- Industrial Economics Incorporated (IEc). (1994). Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document: Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by J.E. Neumann, M.T. Dickie, and R.E. Unsworth.
- Industrial Economics Incorporated (IEc). (2006). Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between PM2.5 Exposure and Mortality, Final Report. Cambridge, MA: Prepared for U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by IEc.
- Jerrett, M., Burnett, R. T., Ma, R., Pope, C. A., 3rd, Krewski, D., Newbold, K. B., . . . Thun, M. J. (2005). Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiology*, 16(6), 727-736.
- Judge, G. G., Griffiths, W. E., Hill, R. C., Lutkepohl, H., & Lee, T.-C. (1985). *The Theory and Practice of Econometrics* (2nd ed.). New York: John Wiley and Sons.
- Katsouyanni, K., Touloumi, G., Samoli, E., Gryparis, A., Le Tertre, A., Monopolis, Y., . . . Schwartz, J. (2001). Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. *Epidemiology*, 12(5), 521-531.

- Kleckner, N., & Neumann, J. (1999). Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income: Memorandum to Jim Democker, U.S. EPA/OPAR.
- Kloog, I., B.A. Coull, A. Zanobetti, P. Koutrakis, J.D. Schwartz. 2012. Acute and Chronic Effects of Particles on Hospital Admissions in New-England. PLoS ONE. Vol 7 (4): 1-8.
- Krewski, D., Jerrett, M., Burnett, R. T., Ma, R., Hughes, E., Shi, Y., . . . Tempalski, B. (2009). Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. *Res Rep Health Eff Inst*(140), 5-114; discussion 115-136.
- Krupnick, A. J., & Kopp, R. J. (1988). The Health and Agricultural Benefits of Reductions in Ambient Ozone in the United States. Washington, DC: Resources for the Future.
- Laden, F., Schwartz, J., Speizer, F. E., & Dockery, D. W. (2006). Reduction in Fine Particulate Air Pollution and Mortality: Extended follow-up of the Harvard Six Cities Study. *Am J Respir Crit Care Med*, 173(6), 667-672.
- Latimer, D. A. (1993, June 13-18). *Development of Regional Haze Screening Models*. Paper presented at the 86th Annual Meeting and Exhibition, Denver, CO.
- Lepeule J, Laden F, Dockery D, Schwartz J. Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009. *Environ Health Perspect*, 120(7), 965-970.
- Levy, J. I., Wilson, A. M., Evans, J. S., & Spengler, J. D. (2003). Estimation of Primary and Secondary Particulate Matter Intake Fractions for Power Plants in Georgia. *Environ Sci Technol*, 37, 5528-5536.
- Liao, D., Cai, J., Rosamond, W. D., Barnes, R. W., Hutchinson, R. G., Whitsel, E. A., . . . Heiss, G. (1997). Cardiac autonomic function and incident coronary heart disease: a population-based case-cohort study. The ARIC Study. Atherosclerosis Risk in Communities Study. *Am J Epidemiol*, 145(8), 696-706.
- Liao, D., Creason, J., Shy, C., Williams, R., Watts, R., & Zweidinger, R. (1999). Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspect*, 107(7), 521-525.
- Loehman, E. T., Berg, S. V., Arroyo, A. A., Hedinger, R. A., Schwartz, J. M., Shaw, M. E., . . . Green, A. E. S. (1979). Distributional Analysis of Regional Benefits and Cost of Air Quality Control. *Journal of Environmental Economics and Management*, 6, 222-243.
- Loomis, D., Castillejos, M., Gold, D. R., McDonnell, W., & Borja-Aburto, V. H. (1999). Air pollution and infant mortality in Mexico City. *Epidemiology*, 10(2), 118-123.
- Magari, S. R., Hauser, R., Schwartz, J., Williams, P. L., Smith, T. J., & Christiani, D. C. (2001). Association of heart rate variability with occupational and environmental exposure to particulate air pollution. *Circulation*, 104(9), 986-991.

- Mar, T. F., Koenig, J. Q., & Primomo, J. (2010). Associations between asthma emergency visits and particulate matter sources, including diesel emissions from stationary generators in Tacoma, Washington. *Inhal Toxicol*, 22(6), 445-448.
- Mar, T. F., Larson, T. V., Stier, R. A., Claiborn, C., & Koenig, J. Q. (2004). An analysis of the association between respiratory symptoms in subjects with asthma and daily air pollution in Spokane, Washington. *Inhal Toxicol*, 16(13), 809-815.
- Moolgavkar, S. H. (2000a). Air Pollution and Hospital Admissions for Chronic Obstructive Pulmonary Disease in Three Metropolitan Areas in the United States. *Inhalation Toxicology*, 12(Supplement 4), 75-90.
- Moolgavkar, S. H. (2000b). Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. *J Air Waste Manag Assoc*, 50(7), 1199-1206.
- Moolgavkar, S. H. (2003). Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. In: Revised Analyses of Time-Series Studies of Air Pollution and Health (pp. 183-198). Boston, MA: Health Effects Institute.
- National Climatic Data Center. NAMER-WINDTEMP (North America wind and temperature) tapes. Asheville, NC.
- National Research Council. (2002). *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, D.C.: The National Academies Press.
- Ostro, B., Lipsett, M., Mann, J., Braxton-Owens, H., & White, M. (2001). Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology*, 12(2), 200-208.
- Ostro, B. D. (1987). Air Pollution and Morbidity Revisited: A Specification Test. *Journal of Environmental Economics and Management*, 14, 87-98.
- Ostro, B. D. (1989). Estimating the Risks of Smoking, Air Pollution, and Passive Smoke On Acute Respiratory Conditions. *Risk Anal*, 9(2), 189-196.
- Ostro, B. D., & Rothschild, S. (1989). Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. *Environ Res*, 50(2), 238-247.
- Peng, R. D., Bell, M. L., Geyh, A. S., McDermott, A., Zeger, S. L., Samet, J. M., & Dominici, F. (2009). Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. *Environ Health Perspect*, 117(6), 957-963.
- Peng, R. D., Chang, H. H., Bell, M. L., McDermott, A., Zeger, S. L., Samet, J. M., & Dominici, F. (2008). Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. *Jama*, 299(18), 2172-2179.
- Peters, A., Dockery, D. W., Muller, J. E., & Mittleman, M. A. (2001). Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*, 103(23), 2810-2815.

- Poloniecki, J. D., Atkinson, R. W., de Leon, A. P., & Anderson, H. R. (1997). Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med*, 54(8), 535-540.
- Pope, C. A. (1991). Respiratory hospital admissions associated with PM10 pollution in Utah, Salt Lake, and Cache Valleys. *Arch Environ Health*, 46(2), 90-97.
- Pope, C. A., 3rd, Burnett, R. T., Thun, M. J., Calle, E. E., Krewski, D., Ito, K., & Thurston, G. D. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Jama*, 287(9), 1132-1141.
- Pope, C. A., Dockery, D. W., Spengler, J. D., & Raizenne, M. E. (1991). Respiratory Health and Pm10 Pollution - a Daily Time Series Analysis. *American Review of Respiratory Disease*, 144(3), 668-674.
- Pope, C. A., 3rd, Muhlestein, J. B., May, H. T., Renlund, D. G., Anderson, J. L., & Horne, B. D. (2006). Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. *Circulation*, 114(23), 2443-2448.
- Popovic, J. R. (2001). 1999 National Hospital Discharge Survey: annual summary with detailed diagnosis and procedure data. *Vital Health Stat* 13(151), i-v, 1-206.
- Roman, H. A., Walker, K. D., Walsh, T. L., Conner, L., Richmond, H. M., Hubbell, B. J., & Kinney, P. L. (2008). Expert judgment assessment of the mortality impact of changes in ambient fine particulate matter in the U.S. *Environ Sci Technol*, 42(7), 2268-2274.
- Rosamond, W., Broda, G., Kawalec, E., Rywik, S., Pajak, A., Cooper, L., & Chambless, L. (1999). Comparison of medical care and survival of hospitalized patients with acute myocardial infarction in Poland and the United States. *Am J Cardiol*, 83(8), 1180-1185.
- Rowe, R. D., & Chestnut, L. G. (1986). Oxidants and Asthmatics in Los Angeles: A Benefits Analysis -- Executive Summary. Washington, DC: Prepared for U.S. Environmental Protection Agency, Office of Policy Analysis. Prepared by Energy and Resource Consultants, Inc.
- Russell, M. W., Huse, D. M., Drowns, S., Hamel, E. C., & Hartz, S. C. (1998). Direct medical costs of coronary artery disease in the United States. *Am J Cardiol*, 81(9), 1110-1115.
- Samet, J. M., Dominici, F., Curriero, F. C., Coursac, I., & Zeger, S. L. (2000). Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994. *N Engl J Med*, 343(24), 1742-1749.
- Schwartz, J. (2000). The distributed lag between air pollution and daily deaths. *Epidemiology*, 11(3), 320-326.
- Schwartz, J., Coull, B., Laden, F., & Ryan, L. (2008). The effect of dose and timing of dose on the association between airborne particles and survival. *Environ Health Perspect*, 116(1), 64-69.
- Schwartz, J., Dockery, D. W., Neas, L. M., Wypij, D., Ware, J. H., Spengler, J. D., . . . Ferris, B. G. (1994). Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children.

- Am J Respir Crit Care Med*, 150(5), 1234-1242.
- Schwartz, J., & Neas, L. M. (2000). Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology*, 11(1), 6-10.
- Sheppard, L. (2003). Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. In: Revised Analyses of Time-Series Studies of Air Pollution and Health (pp. 227-230). Boston, MA: Health Effects Institute.
- Sheppard, L., Levy, D., Norris, G., Larson, T. V., & Koenig, J. Q. (1999). Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology*, 10(1), 23-30.
- Slaughter, J. C., Kim, E., Sheppard, L., Sullivan, J. H., Larson, T. V., & Claiborn, C. (2005). Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. *J Expo Anal Environ Epidemiol*, 15(2), 153-159.
- Smith, D. H., Malone, D. C., Lawson, K. A., Okamoto, L. J., Battista, C., & Saunders, W. B. (1997). A national estimate of the economic costs of asthma. *Am J Respir Crit Care Med*, 156(3 Pt 1), 787-793.
- Standard and Poor's. (2000). The U.S. Economy: The 25 Year Focus.
- Stanford, R., McLaughlin, T., & Okamoto, L. J. (1999). The cost of asthma in the emergency department and hospital. *Am J Respir Crit Care Med*, 160(1), 211-215.
- Sullivan, J., Sheppard, L., Schreuder, A., Ishikawa, N., Siscovick, D., & Kaufman, J. (2005). Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. *Epidemiology*, 16(1), 41-48.
- Tolley, G. S., & et al. (1986). Valuation of Reductions in Human Health Symptoms and Risks: Prepared for U.S. Environmental Protection Agency.
- Tsuji, H., Larson, M. G., Venditti, F. J., Jr., Manders, E. S., Evans, J. C., Feldman, C. L., & Levy, D. (1996). Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. *Circulation*, 94(11), 2850-2855.
- Turner, D. B. (1970). Workbook of Atmospheric Dispersion Estimates, AP-26. Research Triangle Park, NC: U.S. EPA, Office of Air Programs.
- U.S. Bureau of the Census. (1997). *Statistical Abstract of the United States: 1997* (117 ed.). Washington, DC.
- U.S. Bureau of the Census. (2000). Population Projections for States by Age, Sex, Race, and Hispanic Origin: 1999 to 2100. Washington, DC: Population Projections Program, Population Division, U.S. Census Bureau.
- U.S. Bureau of the Census. (2002). *Statistical Abstract of the United States: 2001*. Washington DC.

- U.S. Bureau of the Census. (2008). *Statistical Abstract of the United States: 2009*. Washington, DC.
- U.S. EPA. (1995). User's Guide for the Industrial Source Complex (ISC3) Dispersion Models. EPA-454/B-95-003a. Research Triangle Park, NC.
- U.S. EPA. (1997). *The Benefits and Costs of the Clean Air Act: 1970 to 1990*. Washington, DC: U.S. EPA, Office of Air and Radiation, Office of Policy, Planning and Evaluation.
- U.S. EPA. (1999). *The Benefits and Costs of the Clean Air Act: 1990 to 2010: EPA Report to Congress*. Washington, DC: U.S. EPA, Office of Air and Radiation, Office of Policy.
- U.S. EPA. (2002). *Air Quality Criteria for Particulate Matter, Third External Review Draft*. Research Triangle Park, NC: National Center for Environmental Assessment, Office of Research and Development.
- U.S. EPA. (2003a). *Biogenic Emissions Inventory System (BEIS) Modeling: BEIS Version 3.12*.
- U.S. EPA. (2003b). *Technical Support Document for the Clear Skies Act 2003 Air Quality Modeling Analyses*. Research Triangle Park, NC: Office of Air Quality Planning and Standards, Emissions Analysis and Monitoring Division.
- U.S. EPA. (2005). *Regulatory Impact Analysis for the Final Clean Air Interstate Rule*. Research Triangle Park, NC: Office of Air Quality Planning and Standards (OAQPS), Emission, Monitoring, and Analysis Division and Clean Air Markets Division.
- U.S. EPA. (2006). *Final Regulatory Impact Analysis: PM<sub>2.5</sub> NAAQS*. Research Triangle Park, NC: Office of Air and Radiation, Office of Air Quality Planning and Standards.
- U.S. EPA. (2009). *Proposed NO<sub>2</sub> NAAQS Regulatory Impact Analysis (RIA)*. Research Triangle Park, NC.: Office of Air and Radiation, Office of Air Quality Planning and Standards Retrieved from [https://www3.epa.gov/ttnecas1/docs/ria/naaqs-no2\\_ria\\_proposal\\_2009-07.pdf](https://www3.epa.gov/ttnecas1/docs/ria/naaqs-no2_ria_proposal_2009-07.pdf).
- U.S. EPA. (2010a). *Guidelines for Preparing Economic Analyses*. Washington, DC: National Center for Environmental Economics, Office of Policy Economics and Innovation.
- U.S. EPA. (2011a). *Assessing the Multiple Benefits of Clean Energy: A Resource for States*. (EPA-430-R-11-014). Washington, DC: Retrieved from [https://www.epa.gov/sites/production/files/2015-08/documents/epa\\_assessing\\_benefits.pdf](https://www.epa.gov/sites/production/files/2015-08/documents/epa_assessing_benefits.pdf).
- U.S. EPA. (2011b). *Emissions Modeling for the Final Mercury and Air Toxics Standards Technical Support Document*. Washington, DC.
- U.S. EPA. (2011c). *Energy Efficiency in Local Government Operations*. (EPA-430-R-09-033). Washington, DC: Retrieved from [https://www.epa.gov/sites/production/files/2015-08/documents/ee\\_municipal\\_operations.pdf](https://www.epa.gov/sites/production/files/2015-08/documents/ee_municipal_operations.pdf)
- U.S. EPA. (2011d). *2011 Daily Particulates Monitoring Data*. Retrieved from [http://aqsdrl.epa.gov/aqsweb/aqstmp/airdata/download\\_files.html](http://aqsdrl.epa.gov/aqsweb/aqstmp/airdata/download_files.html)

- U.S. EPA. (2012a). 2008 National Emissions Inventory, version 2: Technical Support Document. Research Triangle Park, NC: Retrieved from [https://www.epa.gov/sites/production/files/2015-07/documents/2008\\_neiv3\\_tsd\\_draft.pdf](https://www.epa.gov/sites/production/files/2015-07/documents/2008_neiv3_tsd_draft.pdf).
- U.S. EPA (2012f.) Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. Research Triangle Park, NC. [https://www3.epa.gov/ttn/ecas/docs/ria/naaqs-pm\\_ria\\_final\\_2012-12.pdf](https://www3.epa.gov/ttn/ecas/docs/ria/naaqs-pm_ria_final_2012-12.pdf)
- U.S. EPA. (2012c, July 2012). Roadmap for Incorporating Energy Efficiency/Renewable Energy Policies and Programs into State and Tribal Implementation Plans. (EPA-456/D-12-001j). Research Triangle Park, NC: Retrieved from [https://www.epa.gov/sites/production/files/2016-05/documents/appendix\\_0.pdf](https://www.epa.gov/sites/production/files/2016-05/documents/appendix_0.pdf).
- U.S. EPA. (2012c, March 2, 2012). Preferred/Recommended Models, from [http://www.epa.gov/scram001/dispersion\\_prefrec.htm](http://www.epa.gov/scram001/dispersion_prefrec.htm) U.S. EPA. (2015, March 2015). U.S. EPA default PM<sub>2.5</sub> configuration and pooling setup (.zip) file. Office of Air Quality Planning and Standards, Risk and Benefits Group, Research Triangle Park, NC: Accessible at [https://www.epa.gov/sites/production/files/2014-12/pm25\\_cfgx\\_and\\_apvx.zip](https://www.epa.gov/sites/production/files/2014-12/pm25_cfgx_and_apvx.zip).
- U.S. EPA (2018.) Quantifying the Multiple Benefits of Energy Efficiency and Renewable Energy: A Guide for State and Local Governments. State and Local Energy and Environment Program. Washington, DC. <https://www.epa.gov/statelocalenergy/quantifying-multiple-benefits-energy-efficiency-and-renewable-energy-guide-state>
- U.S. EPA. (2020, January 2020). The Emissions & Generation Resource Integrated Database, Technical Support Document for eGRID with Year 2018 Data. Retrieved May 7, 2020, from <https://www.epa.gov/energy/emissions-generation-resource-integrated-database-egrid>
- Wittels, E. H., Hay, J. W., & Gotto, A. M., Jr. (1990). Medical costs of coronary artery disease in the United States. *Am J Cardiol*, 65(7), 432-440.
- Woodruff, T. J., Darrow, L. A., & Parker, J. D. (2008). Air pollution and postneonatal infant mortality in the United States, 1999-2002. *Environ Health Perspect*, 116(1), 110-115. doi: 10.1289/ehp.10370
- Woodruff, T. J., Grillo, J., & Schoendorf, K. C. (1997). The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives*, 105(6), 608-612.
- Woods & Poole Economics Inc. (2015). 2012 Complete U.S. Demographic Database Files. Washington, DC.
- Yamartino, R. J. (1985). Atmospheric Pollutant Deposition Modeling. In D. D. Houghton (Ed.), *Handbook of Applied Meteorology*. New York: John Wiley & Sons.
- Zanobetti, A., Franklin, M., & Schwartz, J. (2009). Fine particulate air pollution and its components in association with cause-specific emergency admissions. *Environmental Health* 8, 58-60.

Zanobetti, A., & Schwartz, J. (2006). Air pollution and emergency admissions in Boston, MA. *J Epidemiol Community Health*, 60(10), 890-895. doi: 60/10/890 [pii] 10.1136/jech.2005.039834 [doi]