# BenMAP

# Environmental Benefits Mapping and Analysis Program

# User's Manual

**December 2022**

**Updated for BenMAP Version 0.01**

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# Chapter 1

# BenMAP

# Quick Start Guide

# Chapter 1 Table of Contents

This chapter of the User Manual will introduce you to U.S. EPA's web-based BenMAP tool and guide you through the process of setting up and running an analysis of the health benefits associated with an improvement in air quality.

# 1.1    How to Acesss BenMAP

**Returning Users:** If you have already registered, navigate to https://benmap.epa.gov, and login using your username and password.

**New Users:** In order to use Benmap, you first need to complete a registration process, described below.

## 1.1.1 New User Registration

The registration steps differ for EPA and non-EPA users.

**EPA Users**

1.  Navigate to https://benmap.epa.gov

    a.  If you are on the EPA network, you will be automatically logged in.

    b.  If you are accessing BenMAP via the Internet, you will be greeted with a login page and should use your PIV card to authenticate. If you are an EPA account user without a PIV card, you should access the tool via VDI desktop or follow the login.gov route described below.

2.  If you are not already a member of the BenMAP Users group, you will see a page directing you to https://waa.epa.gov to request access.

    a.  Click Community Access and then Request Community Access.

    b.  Select the BenMAP Users group.

    c.   Click Submit at the bottom of the page.

3.   Once you receive an email indicating that you have been added to the BenMAP Users group, return to https://benmap.epa.gov and proceed with Section 1.2 of this Quick Start Guide.

**Non-EPA Users**

1.   Navigate to https://waa.epa.gov

2.   Click the Login button on the login.gov tab.
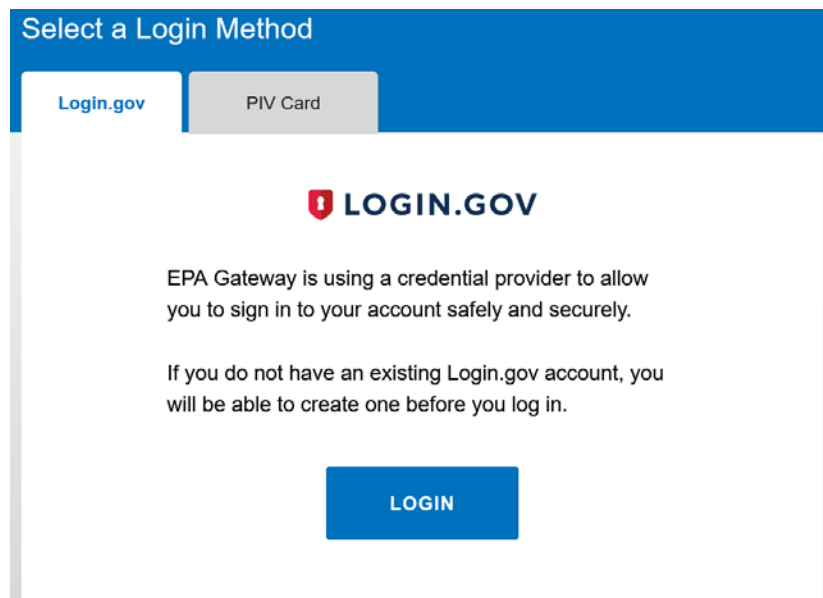
- *If you already have a Login.gov account*, enter your username and password and click Login. On the following web page, select the **Community Access** menu and select **Request Web Community Access**.

- *If you do not already have a Login.gov account*, click **Create an Account** and complete the process to configure and activate your account. Once the account is active, you will be asked to complete the Request Web Community Access form. If you are not automatically directed to the form, select the **Community Access** menu and select **Request Web Community Access**.

3. Complete all the fields on the Request Web Community Access form (see screenshot on next page). Your EPA contact is Neal Fann, fann.neal@epa.gov, 919-541-0209. At the bottom of the page, select the BenMAP Users community from the dropdown list.

**EPA** United States
Environmental Protection
Agency

### Web Application Access Registration

Thank you for registering for EPA Web Applicaiton Access with your login.gov credentials. Please complete this form to gain access to EPA Web Community or Application.

**ALL FIELDS ARE REQUIRED**

EPA Contact Name:

EPA Contact's Email Address:          e.g. emailId@epa.gov

EPA Contact's Phone Number:

**Your Information:**

First Name:

Last Name:

Email Address:

Street Address:

City:

Country:                              ⌄

State/Province/Region:

Postal Code:

Phone Number:

**Select the Community or Application for which you are requesting access:**

Select One                            ⌄
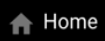
☐ I accept the EPA Privacy & Security Notice. **Click here to read.**

**Submit Registration**    **Reset**

4.  After submitting the form, you will receive one email acknowledging receipt of your request and then, after a period of time, another notifying you that your request has been approved.

5.  Once approved, proceed to https://benmap.epa.gov, log in using login.gov, and proceed with Section 1.2 of this Quick Start Guide.

## 1.2   Home Screen

Across the top of the screen is a black banner with four icons: **Home**, **Data Center**, **Help**, and **Feedback**. These features are described below.
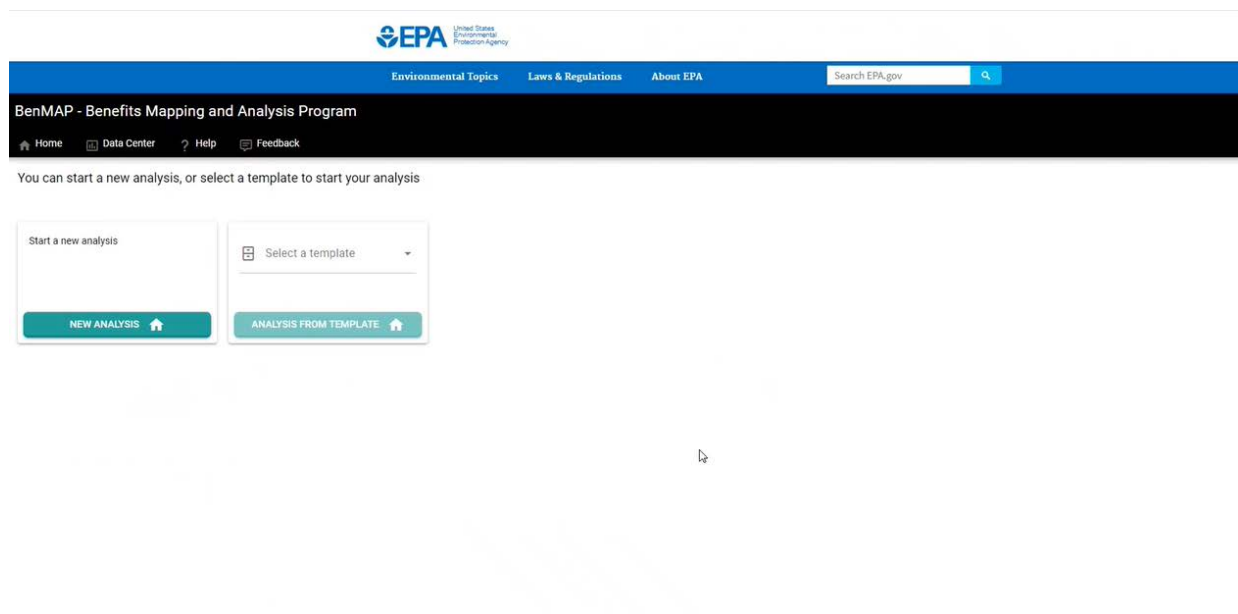


**Home.** ⌂ Home This is the BenMAP welcome screen. To begin a new analysis, click **New Analysis** NEW ANALYSIS ⌂ . If you have previously conducted analyses in BenMAP, you can also choose the **Analysis from Template** ANALYSIS FROM TEMPLATE ⌂ option, which allows you to re-run or modify a previous analysis.

**Data Center.** ⊞ Data Center The data center contains records of your past analyses and the custom file uploads that you created or that have been shared with you by others.

To view your active, pending, or completed tasks, click **Manage Tasks**

MANAGE TASKS ⊞ . If you have any active or pending tasks, the panels on this screen will identify your task, its status, and progress. Completed tasks are shown beneath the list of active/pending tasks.

To review the air quality layers saved in the tool's database and add or delete any custom datasets, click **Review Air Quality Layers** REVIEW AIR QUALITY LAYERS ≣ . Select a pollutant from the drop-down menu, and the default air quality surfaces will be displayed. Clicking on a specific air quality layer will display the air quality values in the table in the bottom portion of this screen.

**Help.** ? Help The Help screen links to https://www.epa.gov/benmap/benmap-cloud, a webpage that includes this user documentation for BenMAP and other more detailed references supporting the health benefits quantification and valuation approaches employed in the tool.

**Feedback.** ⊟ Feedback Clicking this icon directs you to a form where you can provide feedback on the BenMAP tool: https://www.epa.gov/benmap/forms/contact-us-about-benmap.

## 1.3  Conducting an Analysis

The following steps guide you through a basic BenMAP health impacts analysis. From the **Home** screen, click **New Analysis** to start estimating health benefits. You will be directed to a new screen with a progress bar at the top that shows all the steps of a BenMAP analysis: steps

shown in gray have yet to completed; steps shown in blue with a check mark ✅ have been completed; and a step shown in blue with a pencil icon ✏️ indicates the current step.
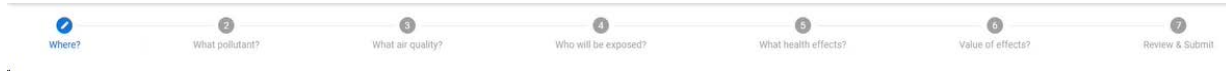


## 1.4   Step 1: Where?

The first step is to select where you want to perform your analysis. BenMAP analyses can be conducted at a variety of scales, ranging from global to national to city or neighborhood scale. The selection you make here will establish the geographical boundaries of your analysis; later you will select the scale at which benefits will be estimated within those boundaries. Currently, **U.S. National** is the only option available in this step.



Select **U.S. National** and click **Continue** to move on to the next step.

## 1.5   Step 2: What pollutant?

In this step, you select the pollutant of concern; this is the pollutant for which BenMAP will estimate **health effects**. The tool currently supports health impact calculations for **ozone** and **PM₂.₅**. Only one pollutant may be analyzed per run. Choosing a pollutant will determine which air quality surfaces and **health impact functions** you will be able to select later. The USEPA website provides more information on [particulate matter pollution](#) and [ground-level ozone pollution](#) and associated **health effects**.



Select a pollutant and click **Continue** to move on to the next step, or **Back** to edit previous selections.

## 1.6   Step 3: What air quality?

To estimate population exposure to air pollution, BenMAP combines air quality data assigned to a spatial grid (i.e., an "air quality surface") with spatially gridded population data. In this step, you will select a pair of air quality surfaces for BenMAP to compare. The first of these surfaces represents **pollutant concentrations** that represent the state of the world before an air quality-related policy or action has been implemented (**Pre-policy**); the second represents concentrations after implementing the policy or action (**Post-policy**). BenMAP calculates the

difference in pollutant concentrations between these surfaces. These differences are used to calculate health impacts.  You may select an existing air quality surface from the BenMAP database or create new surfaces from your own air quality data. BenMAP can generate new surfaces from .csv files of air quality data (instructions for preparing your own .csv file are [here](here)). BenMAP currently accepts files of air quality concentrations generated from computer models that simulate the transport and transformation of pollutants. (Future versions will also accept air quality data from air quality monitor measurements).

BenMAP assigns air quality data to a spatial structure, where each cell contains an air quality concentration for your selected pollutant. These grids are characterized either by regularly shaped and sized cells (like those typically used by **air quality models**), or by irregular polygons representing political designations such as counties, provinces, cities, or nations. BenMAP assumes that the ambient **pollutant concentrations** assigned to a cell represent the exposure experienced by people living in that cell. The tool uses these values to estimate average pollutant concentrations that can be fed into formulas for estimating health impacts.

Currently, the tool accepts air quality inputs assigned to a 12km grid, however future versions of the model will accept data at the U.S. National, State, or County level as well.

## If you want to use a pre-loaded air quality surface:

**Select your pre-policy air quality data.** BenMAP currently contains 4 pre-loaded **ozone** surfaces and 12 pre-loaded **PM$_{2.5}$** surfaces. BenMAP will show you the surfaces that correspond to the pollutant you selected in Step 2.  First, select the scenario you would like to represent the **pre-policy** (i.e., baseline) conditions of your analysis. Metadata for the selected scenario are displayed below your selection and show information such as the count of **grid cells** and mean **pollutant concentration**.

Step 1. Select your pre-policy air quality data

◉ RCU_PM2.5_Annual_Baseline

○ RCU_PM2.5_Annual_Final

Metric, Seasonal Metric
D24HourMean, QuarterlyMean ▾

| Input file characteristic | Value |
|---|---|
| Number of grid cells | 97416 |
| Mean value | 4.28 |
| 2.5th and 97.5th value | 0 - 8.58 |

**Select your post-policy air quality data.** Next, select the scenario you would like to represent the air quality conditions following policy implementation. Metadata for the selected **post-policy scenario** are displayed below your selection.

Step 2. Select your post-policy air quality data
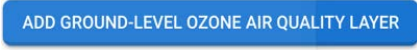
○ RCU_PM2.5_Annual_Baseline

◉ RCU_PM2.5_Annual_Final

Metric, Seasonal Metric
D24HourMean, QuarterlyMean

| Input file characteristic | Value |
|---|---|
| Number of grid cells | 97416 |
| Mean value | 4.28 |
| 2.5th and 97.5th value | 0 - 8.58 |

## If you want to use your own data:

To add a custom air quality layer, click the button at the top of the screen that says **Add Air Quality Layer** `ADD GROUND-LEVEL OZONE AIR QUALITY LAYER`. A window will pop up. In this window, you will need to provide a name for your custom air quality layer and select the **grid definition** it uses. Then, click the **+** icon at the top right to upload the air quality surface from your computer. For details on how to format custom air quality data files so that BenMAP recognizes them, see Section 5.2 of the user manual.
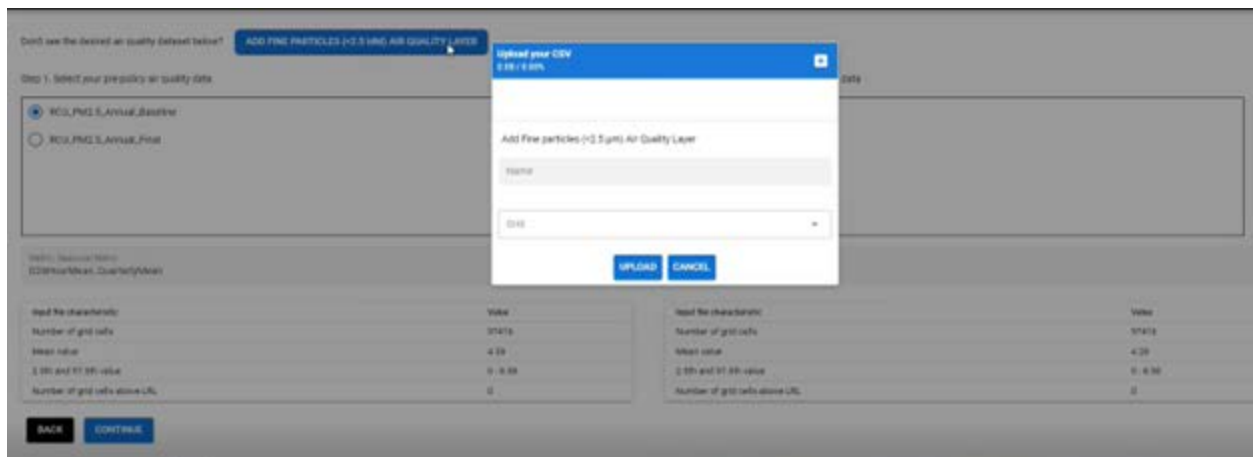


After you've uploaded your custom air quality layer, BenMAP will validate the surface to ensure it is formatted correctly. If your air quality layer passes the validation step and is successfully uploaded, the following pop-up message will appear. Click **OK** to dismiss.
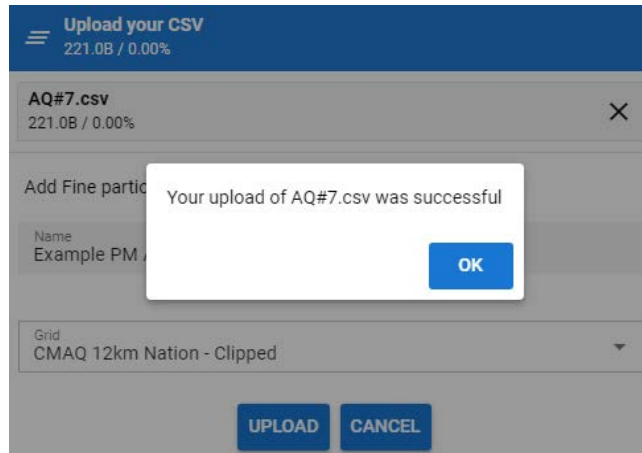
If there is an error with your custom air quality surface, it will not pass the validation step and an error message will appear. A pop-up with possible errors is shown below.
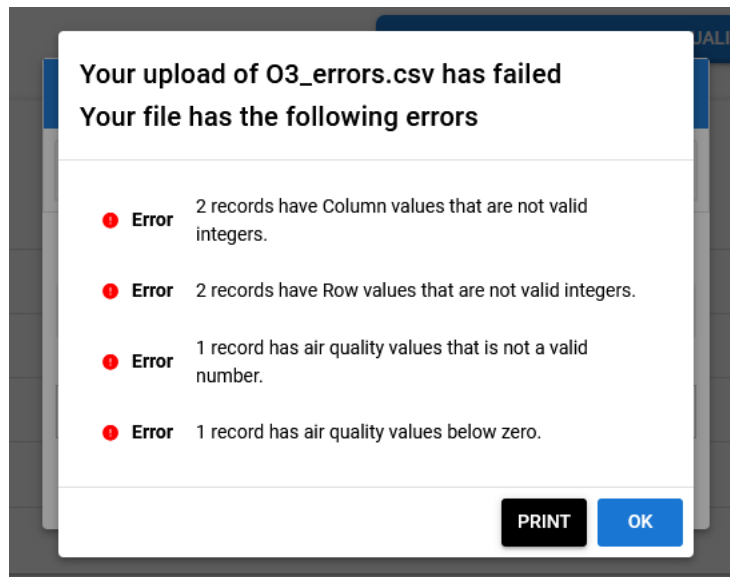


Other potential errors not shown here include using an unavailable pollutant or using an air quality surface name that's already taken. Click **OK** and address the listed error(s) in the input file you created before reuploading.

Once you've selected or uploaded your **pre-policy** and **post-policy** air quality scenarios, click **Continue** to move on to the next step, or **Back** to edit previous selections.

## 1.7 Step 4: Who will be exposed?

BenMAP uses population data to understand how many people are exposed at each modeled or observed concentration of air pollution and can stratify exposures spatially and demographically. This information is critical for estimating numbers of avoided premature deaths or cases of illness associated with a change in air pollution. Population data are associated with a **grid definition**, which specifies the geographic areas for which the data is available (e.g., County or 12-km **grid cells**). The pre-loaded population data for each grid cell are stratified by year and by age, race, ethnicity, and gender. The latter information helps you understand how pollution exposures may be distributed across subpopulations.

You must currently select from pre-loaded U.S. population files in BenMAP. (Future versions will allow you to import their own population data.)

Step 1. What population dataset do you want to use?

Population
US CMAQ 12km Nation

Step 2. In what year will people be exposed?

Year
2015

Step 3. What baseline health dataset do you want to use?

Incidence
County, race-stratified

BACK    CONTINUE

## Population details

**Step 1. What population dataset do you want to use?** Select your population dataset from the drop-down menu. Currently, the only option in BenMAP is **US CMAQ 12km Nation population**.

**Step 2. In what year will people be exposed?** Select a year for your population from the drop-down menu. Currently, the tool supports population years from 2000 through 2055.

## Baseline Health Incidence

In the third step on this screen, you will need to select data describing the baseline health status of your selected population. BenMAP's baseline health datasets describe rates of disease and death in the U.S. population and include data on both incidence (the rate of new cases in the population per person per time) and in some cases **prevalence** (the fraction of the population with a specified disease or diagnosis at a given time). Because these data are meant to capture baseline conditions, these values reflect cases resulting from all stressors or risk factors, including air pollution. These data are specific to the set of **health effects** in the BenMAP database.

**Step 3. What baseline health dataset do you want to use?** Select a baseline health dataset from the drop-down menu. Currently, the dataset options are:

| | |
|---|---|
| County | This dataset contains baseline incidence and prevalence data for fatal and nonfatal health effects at U.S. county scale. More information on this dataset can be found here. |
| County, race-stratified | This dataset contains baseline mortality incidence data stratified by race at the U.S. county scale. More information on this dataset can be found here. |
| County, ethnicity-stratified | This dataset contains baseline mortality incidence data stratified by both race and ethnicity at the U.S. county scale. More information on this dataset can be found here. |
| National | This dataset contains baseline incidence and prevalence data for various nonfatal health effects that are currently only available at the U.S. national level. |

Your answer to the question above indicates your **preferred data**. If baseline health data are available at the spatial scale you selected for the **health effects** you choose, BenMAP will use those data. If baseline health data are not available at the indicated spatial scale, BenMAP will select the next best available data. For example, baseline data for prevalence of asthma symptoms is currently available at the national level only. If you select **County** in this step and choose to evaluate the change in asthma symptoms associated with a change in air quality, BenMAP will automatically select **National** incidence for this **health effect**. You can check BenMAP's incidence data selections when you get to Step 6.

Click **Continue** to move on to the next step, or **Back** to edit previous selections.

## 1.8   Step 5: What health effects?

**Health impact functions (HIFs)** estimate the change in the number of adverse **health effects** associated with a change in exposure to air pollution. The inputs to a health impact function include: the change in air quality concentration for a pollutant, the size of the exposed population, the **baseline incidence rate** of the adverse health effect, and an effect coefficient derived from epidemiological studies. In the steps above you selected the pollutant, population and baseline incidence rates. Below you will select the health effects to estimate using these data.

BenMAP currently allows you to select broad sets of **health effects** to evaluate, specific to your chosen pollutant. (Future versions of the tool will allow you either to specify individual health effects or to specify a custom set of both health effects and the functions used to quantify those effects.)

Estimate a standard set of health effects ⓘ

☐ Premature Death - Primary
☐ Premature Death - All
☑ Chronic Effects - All
☐ Acute Effects - Primary
☐ Acute Effects - All

BACK   CONTINUE

**Estimate a standard set of health effects.** Check the box next to each set of **health effects** you would like to analyze. Available **health impact functions** are grouped into standard **sets** based on the type of health impact and duration of disease.

| Premature Death – Primary | Contains functions that estimate changes in premature death attributable to the air pollutant. These are the studies judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
|---|---|
| Premature Death – All | Contains an expanded set of functions for estimating premature deaths, including functions used in EPA's sensitivity analyses in RIAs, and functions specific to at-risk groups. |
| Chronic Effects – All | Contains functions that estimate longer-term health conditions, e.g., new cases of asthma. These are the studies judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
| Acute Effects – Primary | Contains functions that estimate short-term health effects, e.g., respiratory emergency department (ED) visits. These are the studies judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
| Acute Effects – All | Contains expanded set of functions that estimate short term health effects, including functions used in EPA's sensitivity analyses in RIAs, and functions specific to at-risk groups. |

BenMAP will show you the **health effects** and studies corresponding to your selections above in the next step. Click **Continue** to move on to the next step, or **Back** to edit previous selections.

## 1.9   Step 6: Value of effects?

In addition to quantifying population changes in health, BenMAP lets you assign monetary values to these changes. You can match each **health effect** with a **valuation function** derived from peer-reviewed economic literature and/or extensive health-care databases of

medical costs. Monetizing human health benefits helps express the economic value of improved health to society and facilitates easier comparisons of a policy's health benefits to its implementation costs.

After you select a set of **health effects**, you will be directed to a screen that lists all the individual health functions you have chosen. Here, you can review the **health effect sets**, individual health effects, and the key meta-data for each **health impact function**.



The valuation step is recommended, but optional. If you would like to value your selected **health effects**, click on the pencil icon ✏ at the right of each row. A pop-up window will appear with the same metadata for the **health impact function** of interest, as well as a drop-down menu you can use to select a **valuation function**. Open the drop-down menu and scroll to the valuation function or functions that match the health effect and age range of the health impact function. Click on a valuation function to select it. You may click multiple rows to select multiple valuation functions. To delete any valuation functions you do not wish to include, click the ⊗ next to the function in the pop-up window.

Select all the valuation functions you wish to run and click **Save** to exit the pop-up window and return to the main screen with the full list of health effects/functions. Repeat this process for all health effects that you wish to value.

Once you have selected all valuation functions, click **Continue** to review and submit your analysis, or **Back** to edit previous selections.

If you choose to skip valuation, leave the **Valuation** column blank, scroll to the bottom of the screen, and click **Continue** to review and submit your analysis.

## 1.10  Step 7: Review & Submit

On the **Review and Submit** screen, you will be able to review all of your selections for this analysis: the pollutant, pre- and post-policy air quality surfaces, population dataset, year of analysis, baseline health dataset, **health effect set(s)**, and number of **health impact functions**. Confirm that these are correct or click **Back** to edit your previous selections.

Review And Submit

| Pollutant | Fine particles (<2.5 µm) |
| --- | --- |
| Pre-Policy | RCU_PM2.5_Annual_Baseline |
| Post-Policy | RCU_PM2.5_Annual_Final |
| Population Dataset | US CMAQ 12km Nation |
| Year | 2015 |
| Incidence | County |
| Health Effects | Chronic Effects - All |
| Health Impact Functions | 37 |

Enter a **Task Name** in the text box below the analysis summary. You can use this name to identify the analysis later. If you want to save your analysis configuration as a **template**, enter a **Template Name** as well, and click **Save Template**. Saving a template allows you to preserve the details of your analytical setup and re-use it later.

Click **Submit Task** and BenMAP will assign your task to the Task Queue. A window will pop up that says "Your task [TASK NAME] has been submitted". Click **OK** to close this window and let the analysis run in the background or click **View your Task** to be directed to the **Manage Tasks** window (also available through the **Data Center** if you want to check on your task later).

## 1.11 View and Export Results

Once you have submitted your task, it is assigned to the Task Queue in the **Manage Tasks** window. If there were no pending tasks when your task was submitted, BenMAP will begin running your task momentarily; otherwise, it will begin running once either the tasks ahead of it are completed, or sufficient computing resources become available.

View your active or completed tasks by clicking **View your Task** via the **Review and Submit** window or through the **Manage Tasks** window in the **Data Center**. If your task is still pending, you can see information on its status in the top portion of Manage Tasks window. Also note that each analysis that involves both quantification and valuation will be represented by two tasks: one for quantification and a second for the valuation step. The valuation step will only begin once the corresponding quantification task is completed.
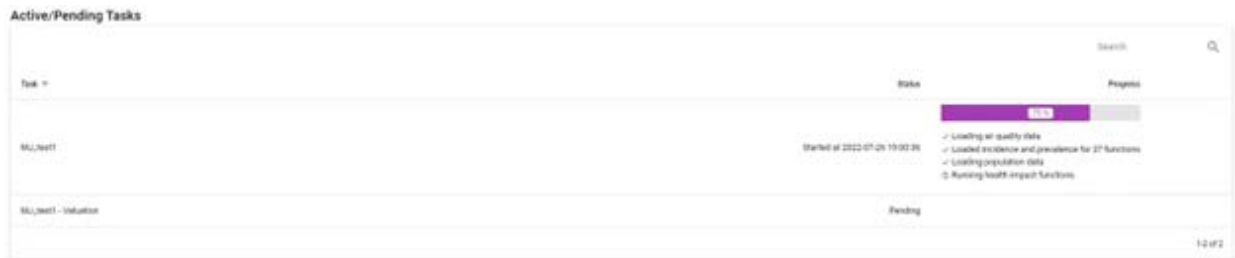


The bottom panel of the **Manage Tasks** window shows all completed tasks; each completed task should have a blue drop-down button  under the **Action** column at the far right. Click on this drop-down to access a menu that lets you either or **View/Export Results** to access the results of the analysis or to **Delete** a task.



If you click **View/Export Results,** a screen will load displaying the **Task Name** at the top left, along with the date and time of the run. The main panel of this screen displays the results of your analysis by **health effect**, age, and study, aggregated to the U.S. National level. This panel also provides the national, population-weighted average change in air quality, the number of avoided health effects, the number of exposed people, and the **baseline incidence** among the population for each health effect. You can select which columns are displayed by clicking the **Columns** drop-down menu and selecting any additional characteristics, such as

race, ethnicity, and gender, that you would like to display. If all columns cannot be shown on screen scroll bars will appear to help you navigate.



Although BenMAP displays your results at the national level, your "raw," unaggregated results have been saved to the BenMAP database, and you can access these data, **aggregate** your results to a variety of spatial scales, and export the results as a .csv file. Click the DOWNLOAD button at the top right. A window will pop up with the task name, as well as a list of available aggregation levels to select. Check the box next to any grid levels you wish to aggregate to – the options currently supported are **CMAQ 12km Nation**, **CMAQ 12km Nation – Clipped**, **County**, **State,** and **Nation.**

| | |
|---|---|
| CMAQ 12km Nation | This shapefile grid definition contains grid cells that are roughly 12 kilometers on each side, for use with air quality modeling data. |
| CMAQ 12km Nation – Clipped | This shapefile grid definition contains grid cells that are roughly 12 kilometers on each side, clipped to the national boundary of the contiguous U.S. |
| County | This shapefile grid definition contains county borders, for use with county-based population and baseline health data. |
| State | This shapefile grid definition contains state borders, for use in generating results aggregated to the state level. |

| Nation | This shapefile grid definition contains an outline of the contiguous United States, defining an overall area of interest. |
|--------|---|



Click **Export** to download your results. The results file(s), in .csv format, and the task log (a text file containing all the selections you made to generate your results) will be available in your browser's download page. The results .csv files can be opened in programs such as Microsoft Excel and Google Sheets.

# Chapter 2

# Introduction to Health Benefits Analysis

In this chapter, find…

- An overview of the tool.
- An overview of health benefits analysis.
- Frequently asked questions.

# Chapter 2 Table of Contents

# 2.1 Introduction to Health Benefits Assessment

The environmental Benefits Mapping and Analysis Program (BenMAP) is a powerful yet easy-to-use program that estimates the number of cases and economic value of health impacts resulting from changes in air pollution concentrations. The open-source BenMAP web tool replaces the desktop version of the program ([BenMAP-CE](#)) that the U.S. Environmental Protection Agency (U.S. EPA) first developed in 2003 to analyze national-scale air quality policies. Previous analyses include health benefits assessments for the National Ambient Air Quality Standards (NAAQS) for Particulate Matter (2006, 2012) and Ozone (2008, 2010) as well as the Revised Cross-State Air Pollution Rule (2021).

U.S. EPA and its partners designed BenMAP to serve the analytical needs of a range of users, including scientists, policy analysts, and decision makers. Most users apply the BenMAP tool to answer one of two types of questions:

1. What are the human health and economic benefits associated with a policy changing air quality?
2. What is the human health burden attributable to total air pollution levels?

### 2.1.1 Overview of BenMAP & Benefits Assessment

The BenMAP program estimates the human health impacts and economic value of air quality changes. That is — BenMAP calculates the human health benefits associated with air quality changes. Such analyses are a critical component of air quality policy assessments. As such, a variety of federal, state and local air pollution officials have used BenMAP to inform air quality management decisions.[1]

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[1] For a list of peer-reviewed articles that used the BenMAP tool, see: www.epa.gov/benmap

BenMAP estimates benefits from improvements in human health such as reductions in the risk of premature death, heart attacks, and other adverse **health effects**. Non-health benefits of reducing air pollution (i.e., visibility and ecosystem effects) are not quantified in the current version of BenMAP. After estimating the changes in the incidence of adverse health effects, BenMAP calculates the monetary benefits associated with those reductions. We provide a high-level overview of this process below. Additional details on the health effect quantification and valuation steps can be found in Chapters 3 and 4, respectively.

**How does BenMAP estimate human health effects?**

First, BenMAP determines the change in ambient air pollution using user-specified air quality data. Because BenMAP does not model air quality changes, you must input these data into BenMAP. Next, BenMAP applies a **health impact function** or a **concentration-response (C-R) function** to pollution concentration changes in order to calculate the corresponding **health effect** changes. HIFs are derived from **epidemiology** studies that calculate effect estimates which relate a change in **pollutant concentration** to a health impact, alternatively called a change in the incidence of health outcomes. Effect estimates are combined with the change in pollutant concentration, population, and **baseline incidence** to create a HIF.  Equation 2-1 shows a typical functional form in which $\Delta Y$ is the change in incidence or health effect, $Y_0$ is the baseline incidence, $\beta$ is the effect estimate, $\Delta$conc is the change in pollutant concentration, and $pop$ is the exposed population. You can specify their preferred HIFs in BenMAP.  This calculation is done for each location in the area of study.  Figure 2-1 illustrates a potential calculation, showing data over a specific area of study which are combined in an HIF to estimate a health impact.

<div align="center">

**Equation 2-1**

$$\Delta Y = Y_0 \left( 1 - e^{-\beta \Delta \text{conc}} \right) * pop$$

</div>

**Figure 2-1.    Calculating a change in health outcomes**
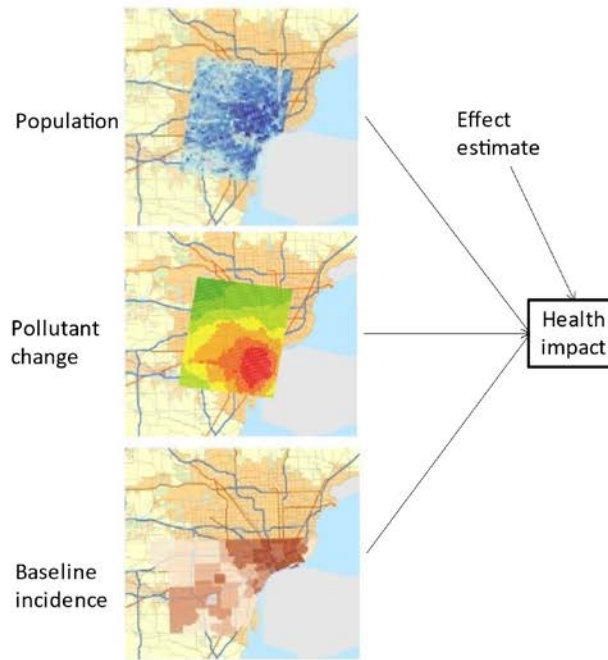


- **Population**. The exposed population is the number of people affected by the air pollution change. The Census Bureau is a good source for this information. In addition, private companies may collect this information and offer it for sale.
- **Pollutant change**. The air quality change is the difference between the starting air pollution level (i.e., the **pre-policy scenario**) and the air pollution level after some change, such as a new regulation (i.e., the **post-policy scenario**).
- **Baseline Incidence**. The baseline Incidence is the probability that a person will suffer a specified adverse **health effect** in a given population over a given period of time. This value should represent the incidence of health outcomes in the population before changes in air quality are considered. Baseline incidences and other health data are typically collected by the government. The World Health Organization is another good

source for baseline incidence data.[2] Additionally, the Global Burden of Disease (BGD) query site can be used to find baseline incidence data.[3]

- **Effect estimate**. The effect estimate approximates the percentage change in the risk of an adverse **health effect** due to a one unit change in ambient air pollution (typically 1 μg/m³ or 1 ppm). Epidemiological studies are a good source for effect estimates.

**How does BenMAP estimate the economic value of human health effects?**

BenMAP calculates the economic value of avoided human **health effects** by multiplying the quantity of these effects by an estimate of the economic value per case (see Figure 2-2 as well as Chapter 4 for details):

Economic Value = Health Impact * Statistical Unit Value of Health Impact

**Figure 2-2.    Estimating the Economic Value of Human Health Effects**



| An air quality policy reduces the number of hospital admissions by 100 | The economic value of each avoided admission is $5,000 in the year 2010 | The economic value is the number of cases multiplied by the value of each admission |
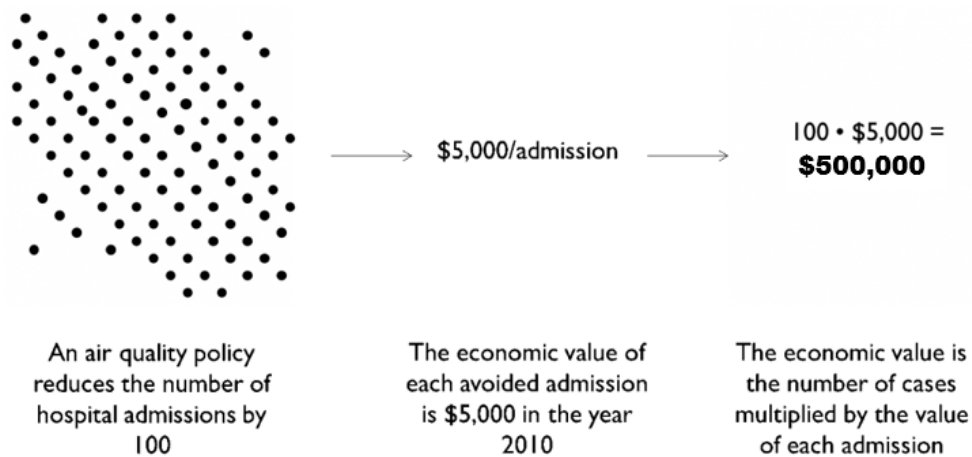
There are several different economic valuation metrics that can be used when calculating the value of the **health effect**. For example, the value of an avoided premature mortality is

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[2] The World Health Organization is a good source for international health data, see: http://www.who.int.
[3] The GBD query site is available at: https://vizhub.healthdata.org/gbd-results/.

generally calculated using the **Value of Statistical Life (VSL)** – the average monetary value that a group of people are willing to pay to slightly reduce the risk of premature death in the population. For other **health effects**, the costs of the illness may be the only valuation data available. **Cost of illness (COI)** measures may include some or all of the direct medical costs, indirect medical costs, lost wages, caregiving costs, and other incurred costs due to the illness.  COI measures do not measure economic utility. The BenMAP database includes several different functions for VSL and valuation functions for other health effects for you to choose, or you can use the U.S. EPA's approach for quantifying and valuing air pollution effects.[4]

Please note that BenMAP does not have air quality modeling capabilities; you must provide externally created air quality data.

**What types of data can you use within BenMAP?**

Table 2-1 lists the 10 types of possible input datasets that you can use within BenMAP and indicates which types of data are needed to perform certain analyses. These components are built into the web tool; the current version of the tool also supports user-uploaded **air quality model** data. Future versions will support additional custom user data inputs.

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[4] See https://www.epa.gov/benmap/benmap-community-edition.

**Table 2-1.     BenMAP Data Elements**

| Dataset Type | Required to Estimate Health Impacts | Required to Quantify Economic Impacts |
|---|:---:|:---:|
| Grid Definitions | ✓ | ✓ |
| Pollutants | ✓ | ✓ |
| Modeled Air Quality Data | ✓ | ✓ |
| Incidence/Prevalence Rates | ✓ | ✓ |
| Population Datasets | ✓ | ✓ |
| Health Impact Functions | ✓ | ✓ |
| Variable Datasets[*] | | ✓ |
| Inflation Datasets | | ✓ |
| Valuation Functions | | ✓ |
| Income Growth Adjustments | | ✓ |

*This includes data tables of socio-economic and demographic data used to support valuation of health impacts as well as the assessment of exposure and impacts stratified by indicators of poverty, education, and other factors.

Analysts can use BenMAP to:

- Compare benefits associated with various regulatory programs;

- Characterize the distribution of health impacts among population subgroups;

- Estimate health impacts and economic values of existing air pollution concentrations;

- Estimate the health benefits of alternative ambient air quality standards; and

- Perform sensitivity analyses of health or valuation functions, or of other inputs.

| Example Applications |
| --- |

Analysts have used BenMAP to investigate a variety of policy questions such as:

- What is the current health burden from $PM_{2.5}$ levels in Addis Ababa?

- How large are the economic benefits of reduced maternal exposure to fine particulate matter?

- What are the future health impacts of wildfire smoke under alternative climate scenarios?

- What are the Environmental Justice implications of alternative air quality strategies in Detroit, MI?

- How large are tree and forest effects on air quality and human health?

- What are the health benefits from vehicular pollution control strategies?

## 2.2  Browser and Computer Requirements

- We recommend using BenMAP with the latest release of any of the following desktop browsers, with cookies and JavaScript enabled (mobile browsers not supported at this time):
    - Google Chrome
    - Firefox
    - Safari
    - Microsoft Edge
- Microsoft Excel or other spreadsheet program (to read exported .csv files and prepare air quality input files)

## 2.3   Contacts for Comments, Questions, and Bug Reporting

If you have comments and questions, or to report a bug, please send EPA a message at the BenMAP website: https://www.epa.gov/benmap/forms/contact-us-about-benmap, or email benmap@epa.gov.

## 2.4   Frequently Asked Questions

**Is BenMAP free? Is there a Terms of Use agreement? Are there any restrictions on using BenMAP?**

BenMAP is free. There is no Terms of Use agreement, though new users must register with EPA to obtain access. The only restriction is that individuals are limited to storing the output of 10 BenMAP runs at any time on the system. You may download results to store locally and delete them from the web database to free up space to conduct additional runs.

**How do I know which version of BenMAP I am using? How do I know if I have the most current version of BenMAP?**

You are automatically using the most up-to-date version of BenMAP at https://benmap.epa.gov. The current version number is shown in the upper right portion of the screen, beneath your username. The version number for any given run is also indicated at the beginning of the Task Log exported with every result download.

**Why do I get different results than someone else?**

There are many possible reasons why your results might differ from someone else's results. One good place to start is reviewing the Task Log. With the Task Log you can examine the assumptions and selections that you have made to generate your results and compare your

selections with those made in another analysis. You can also check and compare BenMAP version numbers and database numbers, which are reported at the beginning of each Task Log.

**Does BenMAP estimate effects of air pollution that are not related to human health (i.e., ecological effects)?**

No. BenMAP does not currently have impact functions to estimate other than human health effects. In principle, it would be possible to estimate ecological effects, as BenMAP is designed to combine different types of geographically variable data. To do so, you would need to develop and load data and impact functions appropriate to estimating ecological effects of interest.

**Where can I find the source code for BenMAP?**

BenMAP is an open-source program and the development team welcomes contributions and scrutiny from the user community. If you are interested in receiving a current copy of the source code, see https://github.com/BenMAPCE/BenCloudServer and https://github.com/BenMAPCE/BenCloudApp.

**How does the web tool differ from the desktop BenMAP-CE tool?**

The user interface of the web tool is fundamentally different from the desktop BenMAP-CE program, though both versions are used for the same purpose. Additionally, the desktop BenMAP-CE program has advanced functionality that has not yet been incorporated into the web tool but is expected in future versions. Future functionality includes pooling, mapping, and additional support for custom inputs.

**Can I still use the desktop version of BenMAP?**

Yes, the desktop tool is still fully functional and available for download at
https://www.epa.gov/benmap. However, it is expected that the desktop tool will eventually
no longer be supported by EPA. Updates will be provided to the BenMAP user community as
this date approaches.

# Chapter 3

# Estimating Health Incidence

---

In this chapter, find…

- A description of how BenMAP estimates the incidence of health outcomes.
- The source of functions relating air pollutant exposures to health effects.
- The types of data needed to estimate air quality-related changes in health incidence.

# Chapter 3 Table of Contents

# 3.1   Introduction to Estimating Health Incidence Changes

Health incidence refers to the counts of new cases of an adverse **health effect** in a population over a specified period of time (e.g., a year). One of BenMAP's primary functions is to estimate changes in health incidence that result from changes in air **pollutant concentrations**. In order to generate these estimates, you will need to create and execute a BenMAP configuration that specifies all the details needed for an incidence analysis. These include:

- The pollutant you want to evaluate;
- The pollutant concentrations in two scenarios you wish to compare, typically referred to as the **pre-policy** and **post-policy scenarios**;
- The year for the analysis;
- The population dataset for the analysis;
- The health effects and health impact functions to be used; and
- The baseline rate of incidence for those health impacts in the specified population.

After entering this information, you may opt to save your configuration choices as a **template** to reuse in the future (see box).

BenMAP gives you flexibility in creating, editing, and saving configuration data. From the home screen, you can specify the details of a new run manually, open an existing **template** and run it as is, or open a template that you then modify to create a new run and/or template. If this is your first time running BenMAP or you have not saved a template from previous runs, you will need to click the **New Analysis** `NEW ANALYSIS` button.

> **Fundamental Concept: Templates** ★
>
> A **Template** includes all the user-specified data and choices for a BenMAP run. This includes the specified pollutant, scale of air quality grids, air quality surfaces, population data, **health impact functions**, and **baseline incidence** datasets. Templates may be saved to your user account prior to running a configuration to allow you to easily re-run or adjust a past analysis without needing to enter all your previous choices.  You can access your templates from the Home screen.

To load a configuration you saved previously, click the drop-down menu "Select a template", which is beside **New Analysis** on the home screen of BenMAP, select your desired template, and then click the **Analysis From Template** ANALYSIS FROM TEMPLATE 🏠 button.
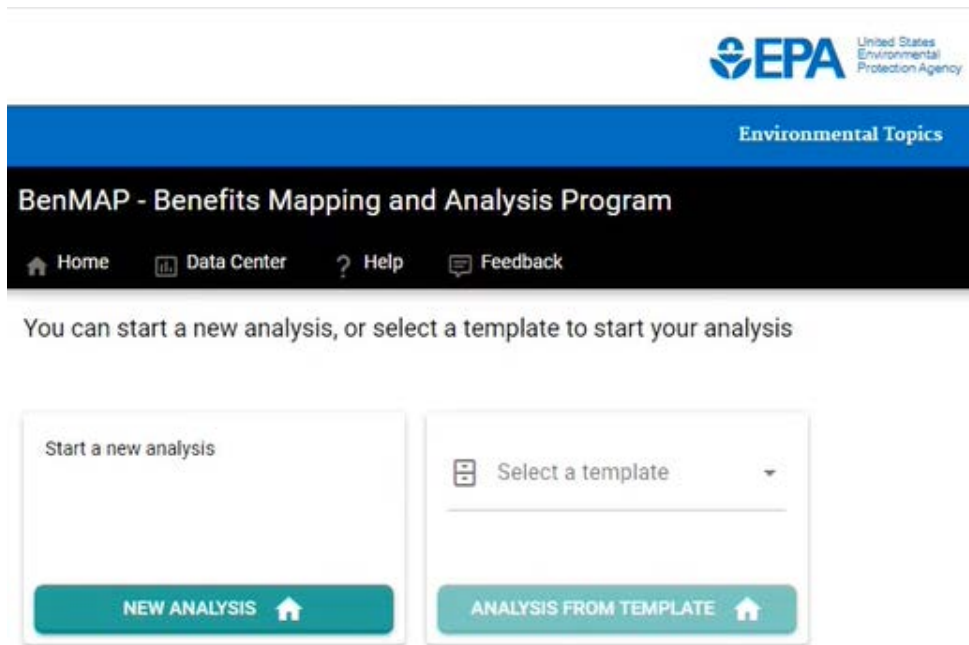


The steps to calculating health impacts are displayed in Figure 3-1 and described in detail throughout the rest of this chapter.

**Figure 3-1.    Steps to Calculating Health Impacts**

$$\Delta Y = Y_0 \left(1 - e^{-\beta \Delta PM}\right) * Pop$$
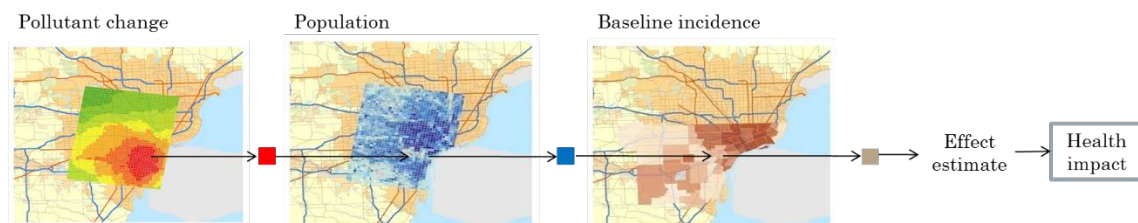
## 3.2 Pollutant Change

The **Pre-Policy** file contains the air quality concentrations for the conditions that exist either prior to or without any change in existing policy. The **Post-Policy** file specifies the air quality concentrations assuming that some type of policy or change has been implemented. The air quality files should contain concentration data for the same pollutant, using the same **metrics** (e.g., daily 24-hour average, 8-hour max) and the same units, such as micrograms per cubic meter ($\mu g/m^3$) or parts per billion (ppb).

The air quality files must also be mapped at the same spatial scale. If you choose a particular **Grid Type** (e.g., *County*) for the **Pre-Policy** file, then the same grid type must be used in the **Post-Policy** file.

| Fundamental Concept: Pre-Policy and Post-Policy Scenarios ★ |
| --- |
| BenMAP requires both a **Pre-Policy Scenario** air quality surface and **Post-Policy Scenario** air quality surface to estimate the effects of a change in air quality (**Delta)**. <br><br> • The **Pre-Policy Scenario** characterizes the air quality levels observed or expected in the absence of the policy change you are evaluating. It is sometimes referred to as "Business as Usual." The Pre-Policy scenario is usually considered to be the reference scenario against which to compare a potential scenario characterized by the implementation of regulations. <br><br> • The **Post-Policy Scenario** in BenMAP is the scenario in which emissions from one or more source sectors are changed (increased or decreased) from the Pre-Policy scenario. The Post-Policy scenario usually represents expected air quality levels after a new regulation or set of regulations has been implemented. <br><br> The air quality **Delta** is the change in air pollution between the **Pre-Policy** air quality grid and the **Post-Policy** air quality grid (Pre-Policy minus Post-Policy). A positive Delta indicates that air pollution has decreased (i.e., air quality has improved in the post-policy scenario compared to the pre-policy scenario). A negative Delta indicates that air pollution has increased, and is worse for human health . BenMAP uses the air quality Delta as the input to the health impact function. |

In future versions of the tool, the **Pollutant** specified in the air quality grids will determine the suite of **Health Impact Functions** available for the configuration. Only functions associated with the specified Pollutant will be available for the configuration. Furthermore, if only certain **Metrics** associated with the pollutant are present in the air quality grids, Health Impact Functions associated with those Metrics will show a notification that the air quality surface does not provide the metric specified in the health impact function.  Currently, you may select any suite of Health Impact Functions, regardless of the selected pollutant.

## 3.3   Population

The population data is used to estimate **population exposure** and in turn any adverse **health effects** associated with a change in air pollution. BenMAP allows you to specify race, ethnicity, gender, and age of the population, as well as the year of the population estimate.

The basis of U.S. population estimates in BenMAP are 2010 Census population counts. Future years are projected using a 2015 source (Woods and Poole) that estimates population through 2050. The rate of growth from 2045 to 2050 is then applied to the 2050 to 2055 period to estimate 2055 population.

Population data loaded into BenMAP must be associated with a **Population Configuration**, which defines the races, ethnicities, genders, and age ranges present in the data. Race, ethnicity, and gender are unique text values representing population subgroups (e.g., "Asian", or "Female"). Age ranges are defined by integer values for starting age and ending

> **Fundamental Concept: Population Configuration** ★
>
> BenMAP requires **population data** in order to estimate the adverse health effects associated with a change in air pollution. Population data may be stratified by age, sex, race, and/or ethnicity. The **population configuration** is a **template** that specifies the categories into which your population data are organized – specifically, the race, ethnicity, gender, and age group subdivisions present in the population data. Detailed population data allow you to more accurately estimate health impacts by better estimating who is exposed and better aligning your data with the study populations of population health studies. It also allows you to estimate and report benefits by age group, sex, race and/or ethnicity (e.g., asthma symptoms in African American males aged 5-17) that may be useful to support environmental justice analyses.

age (inclusive), and a unique text value representing the name of the age range. For example, '0TO1' might be used as a name for the age range defined by a start age of zero and an end age of one, thus consisting of infants through the first twelve months of life and all one-year-old infants. The population data should contain counts for all combinations of race, ethnicity, gender, and age range specified in the associated **population configuration**.

Population data must also be associated with a **grid definition** which specifies the geographic areas for which the data are available. If population data are available for multiple grid definitions (cities and neighborhoods, for example), you can choose to use different sets of population data for different analyses.

BenMAP can also estimate populations for **grid definitions** for which no population data are available by developing area-weighted crosswalks with grid definitions for which data are available.

## 3.4  Baseline Incidence

| Fundamental Concepts: Incidence and Prevalence ★ |
| --- |
| **Incidence** is a measure of the total number of new occurrences of an adverse health impact in a geographic area over time. The **incidence rate** is the average number of health effects (e.g., respiratory hospital admissions) per person per unit of time, typically a day or a year. The incidence rate must be expressed at the same time scale as the specified by the **health impact function**. For example, a health impact function quantifying day-to-day changes in premature death requires a daily mortality rate. The **baseline incidence rate**, also called the **background incidence rate,** is the incidence of a given adverse effect due to all causes including air pollution. BenMAP typically estimates and reports benefits as the change in incidence between the Baseline and Control scenarios, (e.g., the number of avoided asthma Emergency Department visits). <br><br> The **prevalence rate** is the percentage of individuals in a given population at a given point in time who are experiencing or have been diagnosed with a given health condition (e.g., the prevalence of asthmatics among children 0 – 17). It may be required for certain health impact functions, such as those that focus on asthmatics or other groups for which an existing health condition may make them particularly vulnerable to the health effect being studied. |

Most **C-R functions**, such as those developed from log-linear or logistic risk models, estimate the percent change in a **health effect** associated with a specified **pollutant concentration** change. In other words, the absolute effect of air pollution on a specific health effect depends in part on the rate of occurrence of that effect in the exposed population in the baseline. Therefore, most of the **HIFs** in BenMAP require the **baseline incidence rates** (and in some cases the **prevalence rate**) of the adverse health effect as inputs.

The **incidence rate** can be expressed as the number of **health effects** per person in the population per unit of time, and the **prevalence rate** is the percentage of people that have been diagnosed with a particular illness or condition at a given point in time. For example, in 2006-2008, the incidence rate for new asthma diagnoses among children was estimated by researchers to be 1.2 cases per 100 children per year. A recent estimate of the prevalence rate of asthma (measuring the percentage of the population that is already asthmatic) is 7.5 percent of the total population.[5,6]

NOTE: For both **incidence** and **prevalence rates**, BenMAP allows you to apply rates that vary by race, ethnicity, gender, and age group. BenMAP supports multiple sets of incidence and prevalence rates if the rates differ by year or by **grid definition**.

## 3.5  Health Impact Functions

**Health impact functions** relate a change in the concentration of a pollutant to a change in the incidence of a **health effect** (e.g., premature mortality or work-loss days). It is typically derived from the estimated relationship between the concentration of a pollutant and the

---

[5] Example incidence rate from Winer, RA, Qin, X, Harrington, T, Moorman, J, and Zahran, H. 2012. Asthma incidence among children and adults: findings from the Behavioral Risk Factor Surveillance System and asthma call-back survey – United States, 2006-2008. J Asthma 49(1): 16-22.
[6] Prevalence rate from 2018 National Health Interview Survey (NHIS) data, available at https://www.cdc.gov/asthma/nhis/2018/table4-1.htm

adverse health effects suffered by a given population in an **epidemiology** study. For example, the pollutant concentration being measured may be annual fine particulate matter (**PM₂.₅**), and the population response may be yearly deaths from all causes. For the purposes of estimating health benefits, the health impact function itself describes the relationship between the change in concentration of the pollutant, and the corresponding change in the population-health response. We may want to know, for example, if the concentration of $PM_{2.5}$ is reduced by 10 µg/m$^3$, how many premature deaths will be avoided?

To estimate changes in health **incidence**, the first step is to calculate the change in pollution concentrations for a particular policy scenario, such as an air quality improvement produced by a set of emissions controls. The concentration change in a pollutant is the increment between the **pre-policy scenario**, which could represent current conditions or a best estimate of future conditions based on "business-as-usual," and the

**post-policy scenario** that reflects the impacts of the expected pollution control actions.[7] A

> **Fundamental Concepts: Health Impact Function and Concentration-Response Function** ★
>
> A **health impact function** calculates the change in the number of adverse health effects ΔE associated with a change in air quality ΔQ. The inputs to a health impact function include the change in air quality concentration for a pollutant (using a specified **metric** such as annual 24HourMean); the size of the affected population (of specified age, race and ethnicity); the **baseline incidence rate** of the adverse health effect; and an effect coefficient derived from epidemiological studies.
>
> The coefficient for the health impact function is known as Beta (ß) and is derived from epidemiological studies. The value of ß typically represents the percent change in a given adverse health impact per unit change in **pollutant concentration**.
>
> Health impact functions are constructed using **hazard ratios (HR),** which estimate the relationship between the likelihood of adverse health effects as a function of concentration of an air pollutant. The terms C-R function and health impact function are often used interchangeably.

---

[7] You can also design the scenarios to look retrospectively at past pollution control efforts, where the post-policy scenario represents current conditions and the pre-policy represents a hypothetical counterfactual scenario that projects the likely air quality based on historical emissions rates and expected emissions growth in the absence of those historical strategies and controls.

spatial grid that maps each of these changes, a gridded population dataset, and local data on current **baseline health rates** are then fed into **health impact functions** to generate a map of the resulting changes in health effects (i.e., health incidence) that can be attributed to the changes in air pollution. These functions are based on epidemiological studies and are selected by you. Typically, the positive results from applications of these functions indicate a decrease in health incidence (e.g., the decrease in asthma, mortality) resulting from a decrease in air pollution.

### 3.5.1 Evaluating Sets of Health Effects

BenMAP currently includes a broad range of **health impact functions** to evaluate, organized into groups by type of **health effect** called **sets**. These sets are described in Table 3-1.

**Table 3-1.      Description of Health Effect Sets**

| | |
|---|---|
| Premature Death – Primary | Contains functions that estimate changes in premature death attributable to the air pollutant. These are the studies judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
| Premature Death – All | Contains an expanded set of functions for estimating premature deaths, including functions used in EPA's sensitivity analyses in RIAs, and functions specific to at-risk groups. |
| Chronic Effects – All | Contains functions that estimate longer-term health conditions, e.g., new cases of asthma. These are the studies judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
| Acute Effects – Primary | Contains functions that estimate short-term health effects, e.g., respiratory emergency department (ED) visits. These are the studies |

| | judged by EPA to be most appropriate to use for its primary benefits results when conducting major national regulatory impact analyses. |
|---|---|
| Acute Effects – All | Contains expanded set of functions that estimate short term health effects, including functions used in EPA's sensitivity analyses in RIAs, and functions specific to at-risk groups. |

## 3.5.2  Evaluating Individual Health Effects

Future versions of the tool will allow you to select individual **health effects** and **health impact functions** to evaluate, and this documentation will be updated.

## 3.5.3  Details on Health Impact Functions

The complete list of **health effects** currently available in BenMAP is presented in Table 3-2. Health effects that are also included in the "Primary" health effect sets are marked with a ✓.

### Table 3-2.    Health Effects in BenMAP

| Health Effect Set | Pollutant | Primary? | Health Effect |
|---|---|---|---|
| Premature Death – All | PM$_{2.5}$ | ✓ | Mortality, All Cause |
| | Ozone | | Mortality, All Cause |
| | Ozone | ✓ | Mortality, Respiratory |
| Chronic Effects - All | PM$_{2.5}$ | | Acute Myocardial Infarction, Nonfatal |
| | PM$_{2.5}$ | | Hospital Admissions, Alzheimer's Disease |
| | PM$_{2.5}$ | | Hospital Admissions, Parkinson's Disease |
| | PM$_{2.5}$ | | Incidence, Out of Hospital Cardiac Arrest |
| | PM$_{2.5}$ | | Incidence, Stroke |
| | PM$_{2.5}$ | | Incidence, Asthma |
| | PM$_{2.5}$ | | Incidence, Hay Fever/Rhinitis |
| | PM$_{2.5}$ | | Incidence, Lung Cancer |
| | Ozone | | Incidence, Asthma |
| | Ozone | | Incidence, Hay Fever/Rhinitis |

| | | | |
|---|---|---|---|
| | PM$_{2.5}$ | ✓ | Minor Restricted Activity Days |
| | PM$_{2.5}$ | ✓ | Asthma Symptoms, Albuterol Use |
| | PM$_{2.5}$ | | Emergency Hospital Admissions, All Respiratory |
| | PM$_{2.5}$ | ✓ | ER Visits, All Cardiac Outcomes |
| | PM$_{2.5}$ | ✓ | ER Visits, Respiratory |
| | PM$_{2.5}$ | | ER Visits, Asthma |
| | PM$_{2.5}$ | | Hospital Admissions, All Cardiac Outcomes |
| | PM$_{2.5}$ | ✓ | Hospital Admissions, Cardio-, Cerebro- and Peripheral Vascular Disease |
| | PM$_{2.5}$ | ✓ | Hospital Admissions, All Respiratory* |
| | PM$_{2.5}$ | | Hospital Admissions, Respiratory -1* |
| Acute Effects - All | PM$_{2.5}$ | ✓ | Hospital Admissions, Respiratory -2* |
| | PM$_{2.5}$ | ✓ | Work Loss Days |
| | Ozone | ✓ | Minor Restricted Activity Days |
| | Ozone | ✓ | Asthma Symptoms, Chest Tightness |
| | Ozone | ✓ | Asthma Symptoms, Cough |
| | Ozone | ✓ | Asthma Symptoms, Shortness of Breath |
| | Ozone | ✓ | Asthma Symptoms, Wheeze |
| | Ozone | ✓ | ER Visits, Respiratory |
| | Ozone | | ER Visits, Asthma |
| | Ozone | ✓ | Hospital Admissions, All Respiratory |
| | Ozone | | Hospital Admissions, Lower Respiratory Infection |
| | Ozone | ✓ | School Loss Days, All Cause |
| | Ozone | | Minor Restricted Activity Days |
| | Ozone | | Asthma Symptoms, Chest Tightness |
| | Ozone | | Asthma Symptoms, Cough |
| | Ozone | | Asthma Symptoms, Shortness of Breath |
| | Ozone | | Asthma Symptoms, Wheeze |
| Results for Regulatory Analysis | Ozone | | ER Visits, Respiratory |
| | Ozone | | Hospital Admissions, All Respiratory |
| | Ozone | | Incidence, Asthma |
| | Ozone | | Incidence, Hay Fever/Rhinitis |
| | Ozone | | Mortality, Respiratory |
| | Ozone | | School Loss Days, All Cause |

* Respiratory hospital admissions health impact functions evaluate different subsets of respiratory effects and are named accordingly to correspond with associated baseline incidence values. For more information, see section D.2 of Appendix D.

More information about individual **health impact functions** is displayed in the web tool under **Step 5: What health effects**, shown below.



Additionally, details are reported in the task log when you export results. Further information on the functions and underlying studies is available in Appendices E and F of this user manual.

# Chapter 4
# Valuation and Discounting

In this chapter, find…

- Introduction to health benefit valuation.
- How to value changes in mortality risks.
- Overview of discounting and income growth.

# Chapter 4 Table of Contents

# 4.1 Overview of Economic Valuation

In conducting valuation, the program estimates the economic value of the cases of premature death and illness described in the previous chapter." In the example below, we discuss how monetary values for **health effects** are estimated using U.S. based values. We also provide a brief introduction to **discounting**, which weights the value of health benefits depending on whether they would be realized today or in the future.

---

**Fundamental Concept: Valuation** ★

**Valuation Functions** are used by BenMAP to estimate the economic values of changes in the incidence of health effects. In the context of human health benefits assessment, these functions help express society's preferences for avoiding certain health effects as an economic value (e.g., in U.S. dollars).

For morbidity effects, BenMAP estimates monetized benefits using either **Willingness to Pay (WTP)** or **Cost of Illness (COI)**-based valuation functions. **WTP** is viewed by economists as the most complete and appropriate measure of the value of a risk reduction and reflects the willingness of individuals to exchange money for a reduction in his or her risk of illness or death. **COI** estimates the value of a health effect based on the observed direct and indirect costs associated with that condition. Direct costs would include medical costs such as hospital stays and pharmaceutical costs, while indirect costs include impacts such as lost earnings from days unable to work. A COI-based estimate is expected to understate the true economic value of reductions in risk of a health effect because it often captures only a subset of cost categories, and it does not include the value of impact categories such as avoided pain and suffering.

For mortality effects, BenMAP generally estimates monetized benefits using the **Value of Statistical Life (VSL)**, a WTP-based estimate derived from an extensive literature of observed or elicited estimates of the monetary value that an individual is willing to exchange for small reductions in his or her risk of death. It does NOT represent the value of the life of any one specified individual.

---

Improvements in ambient air quality generally lower the risk of developing an adverse **health effect** by a fairly small amount at the individual level. When aggregated across a large population exposed to air pollution, these small risk changes can result in substantial fewer numbers of premature deaths or of anticipated cases of illness, hospitalizations, emergency department visits and so forth. For mortality, it is the small risk changes that are being

valued, based on individuals' **WTP** (i.e., their willingness to exchange money for lower mortality risk). For other health effects, we estimate costs on a case-by-case basis, either by using individuals' measured WTP to avoid a particular effect or using the measurable costs incurred for a given case of illness as a proxy for the value of avoiding that effect (a **COI** approach).  These values are incorporated into **valuation functions** in BenMAP used to estimate the monetized health benefits of reducing air pollution.

These benefits (reductions in risk) may vary across the population (and could be zero for some individuals). Likewise, the **WTP** for a given benefit is likely to vary from one individual to another. In theory, the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations is generally taken to be the sum of everyone's WTP for the benefits they receive. For the **COI** approach, costs may vary from patient to patient, depending on severity of the case and from location to location. Wages may also vary from location to location. In BenMAP, we apply an estimate of the mean WTP or COI value per case and use county-level data on wages to estimate indirect impacts such as lost workdays.

## 4.2  Monetizing Health Benefits

Epidemiological studies allow us to estimate the number of cases of an adverse **health effect** that would be avoided by a given reduction in **pollutant concentrations**. If we have an estimate of the average individual's **WTP** for the risk reduction conferred upon him, we can derive an estimate of the value of a statistical case avoided. Suppose, for example, that a given reduction in pollutant concentrations results in a decrease in mortality risk of 1/10,000. Then for every 10,000 individuals, one individual would be expected to die in the absence of the reduction in pollutant concentrations (who would not be expected to die in the presence of the reduction in pollutant concentrations). If the average individual's WTP for this 1/10,000 decrease in mortality risk is $1,000, then the **VSL** is 10,000 x $1,000, or $10 million. The same type of calculation can produce values for statistical incidences of other health effects.

Sometimes those economic values come from stated preference studies in which study participants are asked about their **WTP** to avoid a specific adverse **health effect**. Many other WTP estimates are derived using revealed preference techniques, which rely on observed behavior in actual markets to infer individuals' preferences for relevant "non-market" goods such as changes in mortality risks. For example, wage-risk studies are a form of revealed preference techniques which rely on labor markets to understand how individuals trade off risks of death with a monetary value (wages). When estimates of WTP are not available, economic values can be approximated by other measures, most notably **COI** measures.

An individual's **WTP** to avoid an adverse **health effect** will include the amount of money he or she would have to pay for medical expenses associated with the illness. 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 **COI** approach attempts to estimate the total value of the medical resources used, the value of the individual's time lost resulting from the illness, and other costs such as caregiver time. Because this method does not include the value of avoiding the pain and suffering resulting from the illness (a potentially large component), it is generally believed to underestimate the total economic value of avoiding the illness, perhaps substantially.

The contingent valuation method attempts to elicit from people what they would be willing to pay to avoid the illness. Because of the distortion in the market for medical goods and services, whereby individuals generally do not pay the full value of medical care, this method too is likely to understate the total economic value of avoiding the illness.

Although the **COI** and **WTP** are the two most common methods, other methods have been used in certain circumstances. The method with which the benefit analyst chooses to value a particular **health effect** will depend in part on what data are available. The unit values

currently available for use in BenMAP are data or estimates that have been collected or generated by researchers and can be readily obtained in publicly available databases or in the open literature. When reviewing the economic literature to determine the appropriate valuation functions to use, it is important to have an economist assist.

## 4.3  Valuing Reductions in Premature Mortality

The economics literature discussing the value of changes in fatality risks is extensive and provides a basis for monetizing benefits when the number of deaths avoided as a result of an air quality improvement can be calculated, but the literature on certain issues regarding the appropriate method for valuing reductions in premature mortality risk is still developing. Issues such as the appropriate discount rate and whether there are factors, such as age or the quality of life, that should be taken into consideration when estimating the value of avoided premature mortality are still under discussion. BenMAP currently offers a variety of options reflecting the uncertainty surrounding the unit value for premature mortality. See Appendices H and I for more detail on the valuation functions available in BenMAP.

Monetary estimates of changes in premature mortality risk are often expressed in terms of the **VSL**. This term is easily misinterpreted and should be carefully described when used in benefit analysis. VSL is the aggregate dollar amount that a large group of people would be willing to pay for a reduction in their individual risks of dying in a year, such that we would expect one fewer death among the group during that year on average. The basic assumption underlying the VSL approach is that equal increments in fatality risks are valued equally. For similar reasons, the VSL approach is only appropriate for small changes in the risk of death and should not be used to value more extensive changes. Because changes in individual fatality risks resulting from environmental regulation are typically very small, the VSL approach is usually acceptable for these types of benefit analyses.

The U.S. EPA National Center for Environmental Economics provides answers to frequently asked questions regarding the economic value of mortality risk on its website: https://www.epa.gov/environmental-economics/mortality-risk-valuation. You may wish to consult this site as you have questions regarding how U.S. EPA derives VSL and applies it in an environmental benefits analysis.

## 4.4  Overview of Discounting

**What is discounting?**

In general, people prefer current consumption to future consumption. In other words, one dollar today is worth more than one dollar tomorrow, and that dollar continues to decrease in value as you go further out into the future. (This concept is also referred to as the social rate of time preference or the time value of money. This is a different concept than **inflation**, which is a general increase in the price level of goods and services.) **Discounting** is the process of converting a future dollar into a value that can be compared to the value of a dollar today. The discount rate expresses this process in quantitative terms. The higher the discount rate, the faster value decreases over time. For example, $1 twenty years from now is worth $0.55 today at a 3% annual discount rate, but worth only $0.26 at a 7% annual discount rate.

> **Fundamental Concept: Discounting / Discount Rate** ★
>
> In a cost-benefit analysis, **discounting** accounts for the fact that people value benefits that occur in the future less than benefits received today. The rate at which individuals discount the value of those benefits is the **discount rate.** Typically, if a benefit is expected to be realized as a stream of benefits over multiple years, as is often assumed for mortality risk reductions, the economic value of that benefit stream would be discounted back to the starting year of analysis and summed as a **Net Present Value (NPV)** of benefits.

A basic **discounting** function is as follows (Equation 4-1):

**Equation 4-1**

$$Present\ Value = \frac{Future\ Value}{(1 + r)^t}$$

where r is the **discount rate** and t is the time period (usually in years).

Example: $1 twenty years from now at a 3% annual discount rate is worth $0.55 today

$$Present\ Value = \frac{\$1.00}{(1 + 0.03)^{20}} = \frac{1}{(1.03)^{20}} = \frac{1}{1.806111} = 0.553676 = \$0.55$$

**Why do we discount benefits?**

The benefits of reductions in air pollution may need to be discounted for three key reasons:

1. Today's society values benefits that occur today more highly than benefits that will occur in the future. Therefore, we must discount to compare those future benefits with current benefits.

2. For a cost-benefit analysis, benefit estimates in a future year need to be comparable to the cost estimates for that same year (which are also discounted).

3. **Discounting** can be used to compare the future *streams* of benefits and costs. The BenMAP program estimates changes in adverse **health effects** based on changes in air quality for one specified analysis year, even though certain health benefits may occur after the analysis year. Discounting can be used to compare the value of future benefits with the value of benefits occurring during the analysis year.

**Under which scenarios would I need to discount benefits?**

**Discounting** of monetary values may be necessary in multiple scenarios, such as the following:

1. The costs and benefits of the rule or policy you are evaluating do not occur in the same year, and there may be a different temporal pattern for the anticipated streams of costs and benefits. For example, costs of implementing a rule may be incurred before emissions begin to be reduced. By discounting the stream of costs and the stream of benefits back to a common reference year and summing each of them into a single integrated value (**Net Present Value**), we can better compare the two values.

2. Certain health effects result in long-term, multi-year medical expenses, lost earnings, and/or other impacts. The future medical costs and impacts avoided will need to be discounted to a single integrated value (**Net Present Value**) to represent the overall value of that avoided health effect in the year it would have occurred. For example, an acute myocardial infarction in one year could result in medical costs and lost earnings for several years.

3. Following a pollution reduction, the resulting benefits may require a number of years to be fully realized as the overall population health improves; this effect is often referred to as a **cessation lag**. The monetized benefits of these future health risk changes need to be discounted to a single NPV in the year of the pollution change. For example, exposure to particulate matter in 2025 could result in a diagnosed case of lung cancer several years later.

**When would we not discount benefits?**

In other instances, it is not necessary to discount the benefits estimates generated by BenMAP. This may be true if:

1. The health benefit occurs within the same year as the exposure change; and
2. The health effect and its associated costs are expected to occur in the same year
3. The exposure change occurs in the same year as the analysis.

The first condition may be satisfied by individual incidence changes that occur in close proximity to the exposure change, such as changes in emergency department visits or inhaler use, where fluctuations in daily concentrations may result in fewer **health effects**. The second condition requires that the health effect results in no costs that extend beyond the year of impact. Impacts such as emergency department visits and school loss days would fall in this category, but not effects such as myocardial infarctions (heart attacks), where multiple years of ongoing treatment and therapy are likely required.  It is important to understand the assumptions within the health and **valuation functions** before you decide whether you need to discount. For example, if your analysis year for your benefits estimates does not match the analysis year for your costs estimates, you may need to discount in order to compare your benefits with your costs even if you meet the criteria listed above. The third condition requires that health impacts be realized in the same year as the analysis.  Health impacts costs and benefits due to an exposure in a future year should typically be discounted to a specified year of analysis, even if they occur concurrently with the exposure change.

**Which discount rate should I choose?**

Selecting a **discount rate** is challenging and is one of the most contentious methodological issues encountered in economic analyses of environmental policies. Because environmental regulations frequently have differing streams of costs and benefits over time, the selected discount rate may determine whether the benefits of a regulatory action exceed the costs. In addition, selecting a higher discount rate may result in a smaller benefits estimate because the future benefits are worth much less than they would be if a lower discount rate was selected. For benefits that occur well into the future, the issue of intergenerational equity further complicates the selection of the discount rate. (In the context of environmental policy, intergenerational equity refers to the fairness of the distribution of the costs and benefits of a long-lived policy when those costs and benefits are borne by different generations. Most criteria pollutants are not considered to have intergenerational equity issues, but the issue

frequently arises in analyses of climate-related air pollution impacts and analyses of mercury exposures.)

There are various economic arguments for and against various discount rates. To comply with OMB and EPA's recommendations, EPA currently uses discount rates of 3% and 7% for benefit analyses.

**Which health effects accrue medical expenses or lost earnings for multiple years, and how do I discount them?**

BenMAP includes **health** and **valuation functions** for several chronic **health effects**, including $PM_{2.5}$-related asthma, and non-fatal acute myocardial infarctions (AMIs, or heart attacks).

- Asthma is assumed to last from the initial onset of the illness throughout the rest of the individual's life. BenMAP currently includes two **COI** functions representing the two discount rates for asthma.
- Technically, AMIs are discrete, acute events, not chronic conditions. However, heart attacks cause chronic follow-up health effects that accrue medical expenses over time, similar to chronic conditions. You can discount the economic value of these chronic effects through the valuation function in BenMAP. AMIs are assumed to accrue costs over five years. Although WTP functions for AMIs are not available, BenMAP currently includes several COI functions that incorporate the direct medical costs and the opportunity cost (lost earnings) for specific age groups at two discount rates.
- Other health effects with multi-year valuations include Alzheimer's Disease, Parkinson's Disease, Out of Hospital Cardiac Arrest, and Non-fatal Lung Cancer.

See Appendix H for details on the categories and temporal patterns of costs and the discounting assumptions, if any, applied within the valuation functions.

**Should I discount the health incidence as well as the valuation?**

You should not discount the health incidence (i.e., the counts of cases avoided) for any of the scenarios mentioned above. The **cessation lag** does not change the total number of premature deaths attributable to a pollution change, but rather the timing of those deaths. If you discounted the health incidence along with valuation, you would be **discounting** twice.

**Which health benefits may not occur in the same year as exposure?**

In many cases, the health benefit from a decrease in exposure to air pollution occurs shortly after exposure, in a matter of hours or days for example, but there can be more significant lags between exposure change and the realization of the full health benefit. If exposure and the **health effect** do not occur within the same year, it is necessary to discount those benefits back to the analysis year. The only **PM$_{2.5}$** health functions currently in BenMAP that fall into this category are PM$_{2.5}$-related premature mortality and incidence of non-fatal lung cancer. **Discounting** the value of these impacts is subject to considerable uncertainty because in both cases, the structure of how the population risk changes with time is unknown. However, scientific literature on similar adverse health effects and new intervention studies suggest that premature mortality, including effects related to lung cancer disease progression, probably would not occur in the same year as the exposure. (See: Roosli M, Kunzli N, Braun-Fahrlander C, Egger M. 2005. "Years of life lost attributable to air pollution in Switzerland: dynamic exposure- response model." International Journal of Epidemiology 34[5]:1029-35.) VSL valuation functions in BenMAP incorporate **cessation lags** using both 3% and 7% discount rates.

EPA's Science Advisory Board recommends future research to support the development of defensible lag structures and provides a lag structure that could be assumed until additional research has been completed. Some example lag structures from the 2012 PM RIA are shown

in Figures 4-1 and 4-2 below.[8] Currently, BenMAP incorporates a 20-year **cessation lag** by default for **VSL** valuation of mortality and a separate cessation lag model for valuing non-fatal lung cancer that is based on historical rates of incidence by age (see Appendix H for details). At present, the web tool does not support estimating other lag structures, though this functionality is expected in future versions of the tool.

Note: **Discounting** is not necessary for short-term **ozone**-related premature mortality because it occurs within the analysis year. However, discounting is necessary for premature mortality associated with long-term ozone exposure.

**Figure 4-1.    Graphical representation of assumed lag structures analyzed in EPA's PM RIA as sensitivity analyses**



Assumed Lag Structures for PM$_{2.5}$ Premature Mortality

---
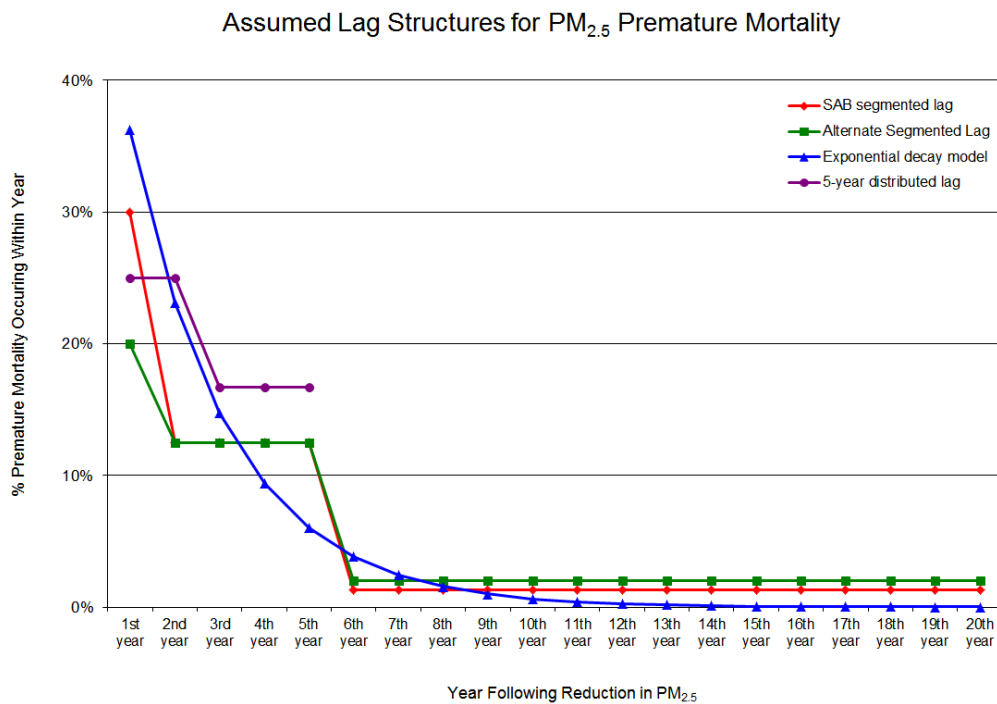
[8] Source: U.S. EPA. 2012. Regulatory Impact Analysis for the Final Revisions to that National Ambient Air Quality Standards for Particulate Matter. Office of Air Quality Planning and Standards. Research Triangle Park, NC. https://www3.epa.gov/ttnecas1/regdata/RIAs/finalria.pdf

**Figure 4-2.    Graphical representation of cumulative assumed lag structures analyzed in EPA's PM RIA as sensitivity analyses**



Assumed Lag Structures for $PM_{2.5}$ Premature Mortality (Cumulative)
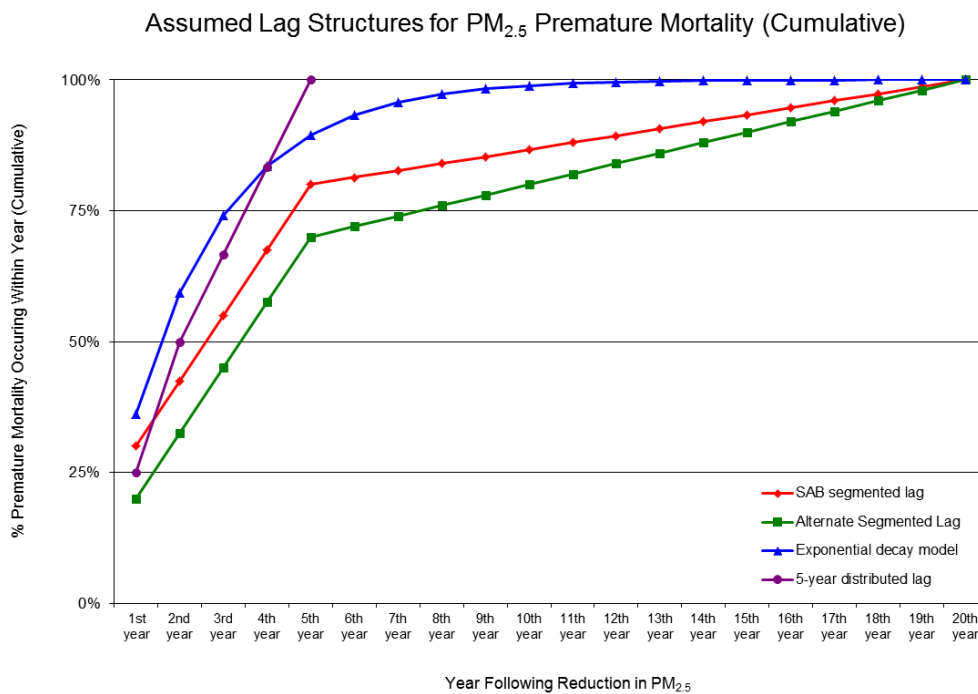
## 4.5  Valuing Incidence Results

When BenMAP runs, it will generate a valuation result for each **Valuation Method** you select by running the method's function on the selected incidence results. You do not need to select a **Valuation Method** for every incidence result—incidence results without any **Valuation Method** will simply be ignored when valuation results are generated.

By default, BenMAP will use the uncertainty estimates built into the **valuation functions**, along with the uncertainty estimated for health incidence changes, to generate lower- and upper-bound estimates of the value of health impacts (specifically, the 2.5$^{th}$ and 97.5$^{th}$ percentiles of the distribution, representing a 95% confidence interval around the central result).  The web tool does not currently support exporting other percentiles of the distribution, but this functionality will be included in a future release. To obtain the value of the health incidence, BenMAP draws samples from both the valuation and health incidence

distributions and multiplies each combination of values together, then sorts these results to estimate the combined uncertainty contributed by both incidence and valuation. More information on this procedure is found in Appendix K.

### 4.5.1 How to Specify Valuation Functions in BenMAP

After you have specified your **health effects**, you can choose valuation functions to generate a

> **Guidance/Best Practices** ✓
>
> When selecting Valuation Methods for your analysis, it is important to match the valuation function to the health incidence estimate as closely as possible. For example, if the health effect is *Asthma Symptoms, Albuterol use*, then the valuation function should correspond specifically to albuterol use rather than other asthma symptoms such as cough or wheeze.
>
> For long-term health impacts, the valuation function may also account for your preferred time span of analysis and discount rate.

monetized estimate of health benefits for each health effect. After selecting your **health effect sets**, you will be presented with a screen similar to the one below that lists all the individual health effects that you have chosen to analyze.



To value a **health effect**, click the pencil icon ✏ at the far right of its row. A pop-up window will appear that includes a drop-down menu you can use to select a valuation function. Scroll to the **valuation function** or functions that match the health effect and age range of the **health impact function** you are valuing. Click on a valuation function to select it. You may click multiple rows to select multiple valuation functions, each of which will be run to generate a separate valuation estimate.

Once you have selected all the valuation functions you wish to run for a given **health effect**, click **Save** to exit the pop-up window and return to the main screen with the full list of health effects. Repeat this process for each health effect you wish to value.

For more guidance on how to value health effects, see [Section 1.9](#) of this user manual.

## 4.5.2  Details on Valuation Functions

The complete list of health effects with **valuation functions** currently available in BenMAP is presented in Table 4-1.

**Table 4-1.     Details on Valuation Functions**

| Health Effect | Age Range |
|---|---|
| Acute Myocardial Infarction, Nonfatal | 0-24; 25-44; 45-54; 55-65; 66-99 |
| Minor Restricted Activity Days | 18-99 |
| Asthma Symptoms, Chest Tightness | 0-17; 18-99 |
| Asthma Symptoms, Cough | 0-17; 18-99 |
| Asthma Symptoms, Shortness of Breath | 0-17; 18-99 |
| Asthma Symptoms, Wheeze | 0-17; 18-99 |
| Asthma Symptoms, Albuterol Use | 0-99 |
| ER Visits, All Cardiac Outcomes | 0-99 |
| ER Visits, All Respiratory | 0-99 |
| ER Visits, Asthma | 0-99 |
| Emergency Hospital Admissions, All Respiratory | 65-99 |
| Hospital Admissions, All Cardiac Outcomes | 0-99 |
| Hospital Admissions, Cardio-, Cerebro- and Peripheral Vascular Disease | 65-99 |
| Hospital Admissions, All Respiratory | 0-18; 65-99 |
| Hospital Admissions, Respiratory-1* | 0-99 |
| Hospital Admissions, Respiratory-2* | 65-99 |
| Hospital Admissions, Alzheimer's Disease | 65-99 |
| Hospital Admissions, Parkinson's Disease | 18-99; 65-99 |
| Incidence, Out of Hospital Cardiac Arrest | 35-99 |
| Incidence, Stroke | 18-99 |
| Incidence, Asthma | 0-17; 4-21; 35-99 |
| Incidence, Hay Fever/Rhinitis | 0-17 |
| Incidence, Lung Cancer | 30-34; 35-44; 45-54; 55-64; 65-74; 75-84; 85-99 |
| Mortality, All Cause | 0-99 |
| School Loss days | 0-17 |
| Work Loss Days | 18-65 |

* Respiratory hospital admissions valuation functions evaluate different subsets of respiratory effects and are named accordingly to correspond with associated health impact functions and baseline incidence values. For more information, see section H.2.1 of Appendix H.

## 4.6 Currency Year and Income Growth

The web tool maintains the **Variable Datasets** from the desktop tool, which includes economic variables such as income and poverty data, inflation indices, and **income growth** factor tables. The relevant year for **inflation** and **income growth** matches the year chosen for population when you prepare your BenMAP run configuration. You can confirm the years used for these adjustments in the Task Log.

**Inflation Adjustment**

The **Inflation Adjustment** needs to be carefully considered in relation to the valuation dataset that you are using. The web tool uses the same inflation factor table as the desktop version of BenMAP. These indices were derived using data from the Bureau of Labor Statistics (all goods index, medical cost index, wage index). The default valuation database in the *United States* setup has a currency year of *2015*, so the inflation dataset has a value of *1* for the year *2015*. The web tool sets the inflation year is equal to the Population Year you selected in Step 4, or to the latest year for which inflation data are available if less than the population year.

**Income Growth Adjustment**

Economic studies generally provide evidence that WTP estimates are influenced by the income of individuals. As income rises over time, people are expected to allocate more money towards safety and risk reduction, and thus WTP estimates are likely to increase as well. The **Income Growth Adjustment** is designed to take this phenomenon into account, allowing you to account for income growth between the time when WTP estimates were calculated and the year of your analysis.

As with the **Inflation Adjustment**, the **Income Growth Adjustment** has a close connection to the valuation estimates. For example, the valuation estimates in the *United States* setup are

assumed to be based on income levels from 1990, so the income growth adjustment database has a value of 1 for the year 1990.

The web tool automatically incorporates an **Income Growth Adjustment**, where the income year is set equal to the Population Year you selected in Step 4, or to the latest year for which income growth adjustment data are available if less than the population year. Historical income growth (1990-2016) is from the U.S. Bureau of Economic Analysis (BEA). Future changes in annual income are based on data presented in the 2020 Annual Energy Outlook.

# Chapter 5
# Customizing a Benefits Analysis

In this chapter…

- Learn more about the file structure for data inputs.
- Learn how to import custom datasets.

# Chapter 5 Table of Contents

# 5.1  Introduction to Customizing a Benefits Analysis

BenMAP contains numerous datasets that can be used to conduct a complete health benefits assessment. However, you can also import custom datasets to the tool. Custom datasets allow you to tailor a given analysis to use parameters specific to his or her scenario and/or geographic area.

Currently, the web tool supports import of custom air quality surfaces. Future versions of the tool will support customization via additional custom inputs, such as population datasets, **incidence** and **prevalence** data, **health impact functions**, and **valuation functions**.

# 5.2  Custom Air Quality Surfaces

BenMAP does not include an **air quality modeling** component; thus, it relies on user-provided estimates of air quality based on air quality models. Future versions of the tool will support surfaces derived from measured air quality data as well, such as concentrations based on networks of air quality models. Any custom air quality file must be assigned to a **grid structure**, where each cell contains data for one or more **air quality metrics** (e.g., 24-hour average, 8-hour maximum) in that location.

> ### Fundamental Concept: Air Quality Surface ★
>
> An **air quality surface** contains modeled or monitored air pollution data arranged spatially in a series of cells; these cells may be a regular shape (like a 12km by 12km grid) or an irregular shape (like a county or census tract). BenMAP uses one air quality surface to represent the **pre-policy scenario** and a second surface to represent the **post-policy scenario**. These pre- and post-policy surfaces must use the same air quality grid. The program calculates the difference between pre- and post-policy scenarios as an input to the health impact functions.

The **grids** are either regularly shaped areas like those used by **air quality models**, or irregular shapes, like provinces, local government areas, cities, or nations. Each **grid cell** is identified by a Row and Column identifier, as described below. At present, you must assign your custom air quality files to the 12km grid in BenMAP.

Future versions will accept custom air quality files at the U.S. National, State, and County level as well. Future versions will also enable you to first upload a GIS shapefile in order to define a custom grid to which you may then assign air quality concentrations.

## 5.2.1 Model Data File Structure

To create air quality surfaces, BenMAP uses a number of inputs, including modeling data. You may enter your own modeling data, provided that the data are in a format recognized by BenMAP. BenMAP will accept air quality data saved in the comma separated values (CSV) format. Each CSV value must represent a single time period (e.g., one-year or a multi-year average); data for additional time periods must be uploaded in a separate file.

Table 5-1 presents the required elements for these files; variable names must be matched exactly. Table 5-2 presents a sample data file. Each CSV file may contain data for multiple **air quality metrics**; for example, an **ozone** file may contain measurements at each **grid cell** for both 8-hour maximum and 1-hour maximum ozone metrics, subject to the constraints in Table 5-1.

**Table 5-1.     Air Modeling Data File Variables**

| Variable | Type | Required | Notes |
|---|---|---|---|
| Column<br><br>Row | Integer<br><br>Integer | Yes<br><br>Yes | The column and the row uniquely identify each set of modeling values and link the modeling data with cells in a grid definition. |
| Metric | Text | No | This variable is either blank (signifying that the Values are Observations, rather than Metric values), or must reference an already defined Metric (e.g., 24-hour daily mean) for the appropriate Pollutant. |
| Seasonal Metric | Text | No | This variable is either blank (signifying that the Values are not Seasonal Metric values) or must reference an already defined Seasonal Metric for the Metric (e.g., mean of the 1-hour maximum values for the months of June through August). Files may contain data for more than one metric, but the values for each metric/grid cell combination must appear in its own row. |
| Annual Metric | Text | Yes | For use with annual values, this variable must be one of: *None*, *Mean*, *Median*, *Max*, *Min*, *Sum* (e.g., mean of the 1-hour maximum values for the year) |
| Values[9] | Number | Yes | The tool currently accepts annual values, i.e., a single value for the year for each grid cell. See Table 5-2 for a sample air quality file. |

---

[9] Future functionality of the tool will support daily values, which must be supplied as a comma-delimited string of values for the year [e.g., 365 or 366 (leap year) values for daily data]. To input daily values, Annual Metric must be blank. Missing values must be signified with a period ('.').

**Table 5-2.      Sample Air Modeling Data File**

| Column | Row | Metric | Seasonal Metric | Statistic | Values |
|---|---|---|---|---|---|
| 24 | 101 | D24HourMean | QuarterlyMean | Mean | 17.53 |
| 24 | 102 | D24HourMean | QuarterlyMean | Mean | 17.15 |
| 24 | 103 | D24HourMean | QuarterlyMean | Mean | 20.00 |
| 24 | 104 | D24HourMean | QuarterlyMean | Mean | 17.07 |
| 24 | 105 | D24HourMean | QuarterlyMean | Mean | 13.63 |
| 24 | 106 | D24HourMean | QuarterlyMean | Mean | 17.97 |
| 24 | 107 | D24HourMean | QuarterlyMean | Mean | 15.05 |
| 24 | 108 | D24HourMean | QuarterlyMean | Mean | 13.47 |
| 24 | 109 | D24HourMean | QuarterlyMean | Mean | 19.56 |
| 24 | 110 | D24HourMean | QuarterlyMean | Mean | 13.94 |
| 25 | 101 | D24HourMean | QuarterlyMean | Mean | 17.07 |
| 25 | 102 | D24HourMean | QuarterlyMean | Mean | 15.32 |
| 25 | 103 | D24HourMean | QuarterlyMean | Mean | 16.20 |
| 25 | 104 | D24HourMean | QuarterlyMean | Mean | 18.13 |
| 25 | 105 | D24HourMean | QuarterlyMean | Mean | 17.13 |
| 25 | 106 | D24HourMean | QuarterlyMean | Mean | 15.64 |
| 25 | 107 | D24HourMean | QuarterlyMean | Mean | 19.99 |
| 25 | 108 | D24HourMean | QuarterlyMean | Mean | 15.16 |
| 25 | 109 | D24HourMean | QuarterlyMean | Mean | 12.06 |
| 25 | 110 | D24HourMean | QuarterlyMean | Mean | 14.08 |

## 5.2.2 Loading Custom Air Quality Surfaces

There are multiple ways to add a custom air quality layer to BenMAP: through the **Data Center** or during **Step 3: What Air Quality**.

To review air quality layers saved in the tool's database and add or delete any custom datasets, go to the **Data Center**. Click **Review Air Quality Layers**.
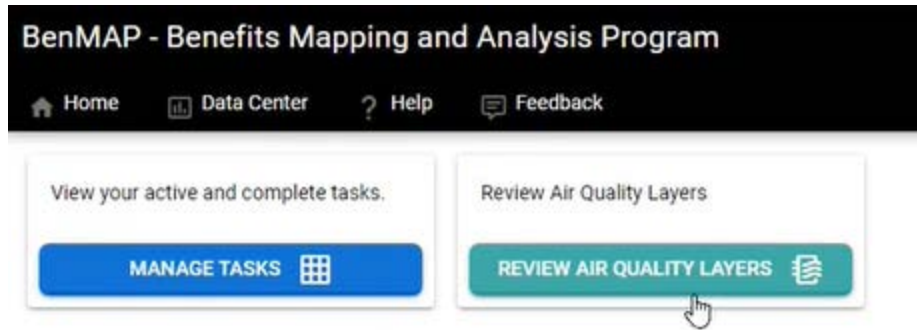
Select a pollutant from the drop-down menu.



All air quality surfaces that fit the pollutant you selected will be displayed, as well as the option to **Add an Air Quality Layer.**
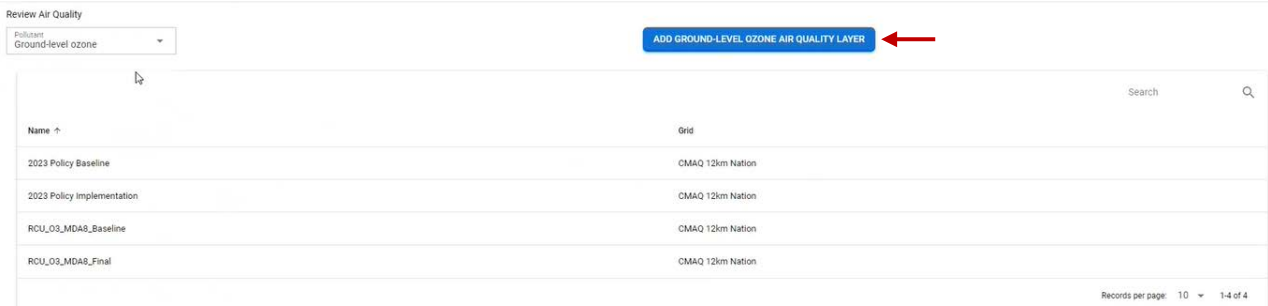


You can also add a custom air quality layer in **Step 3: What Air Quality.** When you reach this step, you will see a button at the top of the screen to **Add an Air Quality Layer.**
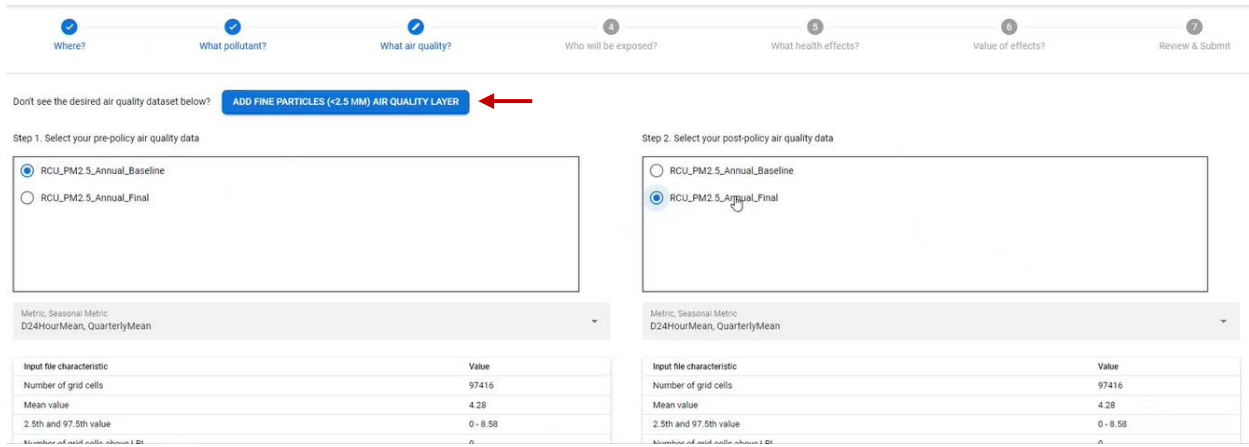
A window will pop up, where you will be asked to provide a name for your custom air quality layer, select the grid definition it uses, and upload the file from your computer.
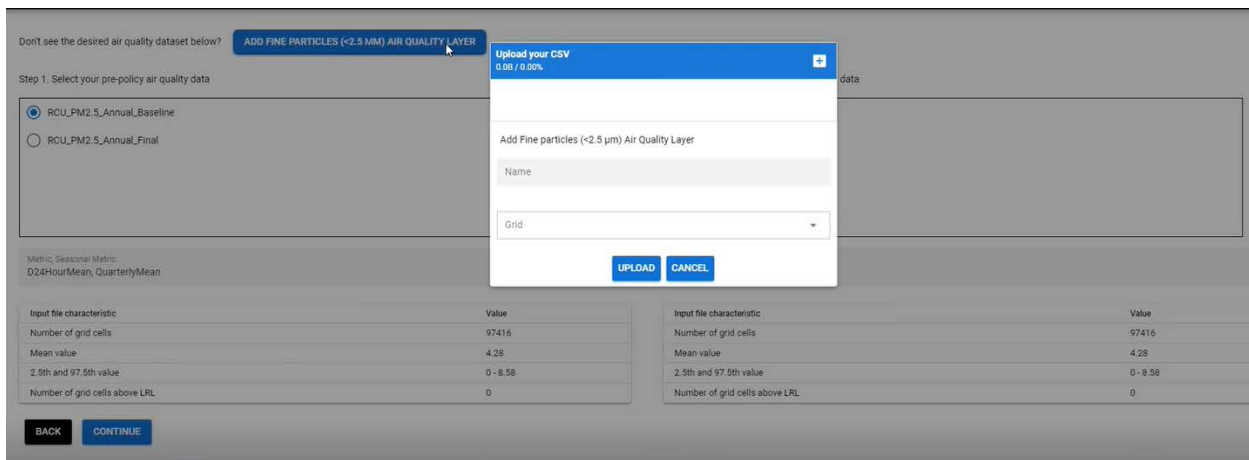


For more information on uploading custom air quality surfaces, refer to Section 1.5 of this user manual.

## 5.2.3 Validating Custom Air Quality Surfaces

After uploading your custom air quality layer, BenMAP will validate the surface to ensure it is formatted correctly. If your air quality layer passes validation and is successfully uploaded, the following pop-up message will appear. Click **OK** to dismiss.
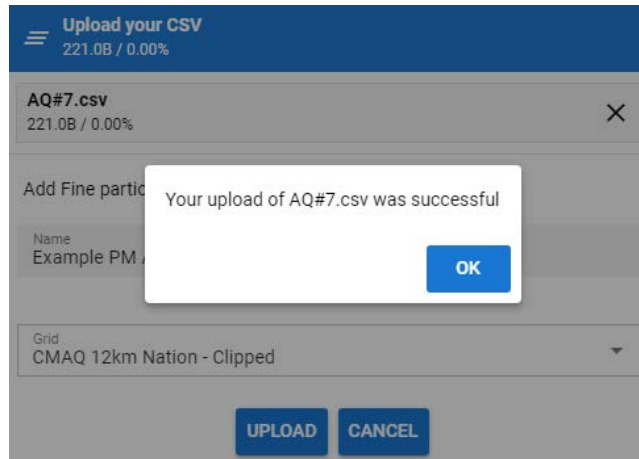
Your custom air quality surface will appear in the **Review Air Quality Layers** window of the data center. You can select the layer and review the statistics displayed below to check the data that you just imported.



If there is an error with your custom air quality surface, it will not pass the validation step and an error message will appear. A pop-up with possible errors is shown below.

Other potential errors not shown here include using an unavailable pollutant or using an air quality surface name that's already taken. Click **OK** and address the listed error(s) in the input file you created before reuploading.

# Chapter 6
# Terminology

## In this chapter, find…

- Definitions for common terms used in the BenMAP tool and in this manual.

**Aggregation.** The summing of grid cell level resulting in a larger spatial scale, such as county, state, or national levels.

**Air Quality Metric.** A value that expresses both the time period over which air quality values are modeled or observed and whether that modeled or observed air quality value is an average, maximum, or minimum. For example, the metric DailyMean represents the average concentration for the sampling day. This could be taken directly from a single 24-hour observation or from an average of hourly (or more frequent) observations. In addition to the time period, some metrics also specify the method used for averaging or aggregation. For example, a typical ozone metric D8HourMax represents the highest of the 8-hour moving averages during the day.

**Air Quality Model.** Air quality management tools that mathematically describe pollution transport, dispersion, and related physical and chemical processes in the atmosphere. Air quality models (like CMAQ[10] and CAMx[11]) are used to estimate the air pollutant concentration at specific locations, which are referred to as receptors, or over a spatial area that has been divided into uniform grid squares. The number of receptors or grid cells in a model far exceeds the number of monitors one could typically afford to deploy in a monitoring study. Therefore, models provide a cost-effective way to analyze pollutant impacts over a wide spatial area where factors such as meteorology, topography, and emissions from both local and remote sources could be important. BenMAP does not contain an air quality model but can use the output from these models to estimate health impacts.

**Air Quality Surface.** A file containing modeled or monitored air pollution data in a series of cells; these cells may be a regular shape (such as a 12km by 12km grid) or an irregular shape

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[10] Community Multi-scale Air Quality (CMAQ) Model is available at: https://www.epa.gov/cmaq or https://www.cmascenter.org/cmaq/.
[11] Comprehensive Air Quality Model with Extensions (CAMx) is available at: http://www.camx.com/.

(such as a county or census tract). These surfaces are also referred to as air quality grids. BenMAP uses one air quality grid to represent the pre-policy scenario and a second grid to represent the post-policy scenario. These pre-policy and post-policy grids must share the same geographic structure. The program calculates the difference between the pre- and post-policy grids as an input to the health impact function.

**Attainment.** The state of meeting the National Ambient Air Quality Standard (NAAQS) standard for a pollutant and not contributing to a nearby area exceeding the standard. A geographical area that meets the NAAQS is called an "attainment area." BenMAP modeling results are not designed to designate attainment for regulatory purposes.

**Background Concentration.** The concentration of a pollutant, generally in the absence of human sources.

**Baseline Incidence.** The incidence of a given adverse effect due to all causes including air pollution. Also called background incidence rates.

**Beta.** The coefficient for the health impact function that measures the strength of the impact of air pollution exposure on a health effect. The value of beta (ß) typically represents the percent change in a given adverse health impact per unit of pollutant exposure.

**Cessation Lag.** This term represents the time that is expected to elapse between a reduction in pollutant exposure and the achievement of new steady state health risk level in the exposed population. The cessation lag model also specifies the proportion of the overall risk reduction occurring in the population during each time step (e.g., year) of the lag period. The result of applying the cessation lag model is an expected time stream of health benefits from the initial exposure reduction to the end of the lag period, which is then used in the benefits valuation step.

**Concentration-Response (C-R) Function.** An equation that estimates the relationship between adverse health effects and ambient air pollution and is used to derive health impact functions (defined below). You will often see that the term C-R function and [health impact function](#) are used interchangeably.

**Cost of Illness (COI).** An estimate of the value of a health effect that includes the direct medical costs incurred and indirect impacts such as lost earnings associated with illness. These estimates generally understate the true economic value of reductions in risk of a health effect because they only include the direct expenditures related to treatment and lost earnings but not the value of other impacts such as avoided pain and suffering.

**Deltas.** The difference between two data points. As used in BenMAP, mapping the air quality deltas shows the change in air pollution between the pre-policy air quality grid and the post-policy air quality grid.

**Discount Rate.** In a cost-benefit analysis, the discount rate is a measure of the degree to which individuals value benefits or costs today more highly than future benefits and costs Typically, if a benefit occurs over multiple years, the economic benefit would be discounted.

**Epidemiology.** The study of factors affecting the health and illness of populations.

**FIPS Code.** Federal Information Processing Standard codes. FIPS codes uniquely identify geographic areas; the number of digits vary depending on the level of geography. Each state in the United States is assigned a 2-digit code (e.g., "06" refers to California). Each county is assigned a 5-digit code of which the first two digits identify the state to which the county belongs (e.g., "06037" refers to Los Angeles County, California). Counties can be further subdivided into census tracts, denoted by FIPS codes of 11 digits.

**Grid Cell.** One of the many geographic, or spatial, components within a [grid definition](#). These can be regularly or irregularly shaped.

**Grid Definition.** A BenMAP grid definition provides a means of breaking a geographic region into areas of interest (Grid Cells) that allow for spatial variation in data relevant to a benefits analysis. All grid definitions must have a unique (i.e., non-repeating) column and row index. BenMAP currently includes one grid definition with uniformly shaped and sized cells (12 km x 12 km).

**Health Effect.** A subset of a health effect set which represents a specific type of adverse health effect and may span multiple medical diagnostic codes. For example, the health effect set *Chronic Effects - All* includes health effects *Asthma Incidence*, *Lung Cancer Incidence*, and *Stroke Incidence*.

**Health Effect Set.** A group of health impact functions that quantify impacts within certain categories, including mortality, acute (short-term) morbidity effects, and chronic (long-term) morbidity effects.

**Health Impact Function.** An equation that calculates the change in adverse health effects associated with a change in exposure to air pollution. Based on a C-R function, a typical health impact function accepts inputs specifying the air quality metric and pollutant; the change in air pollutant concentration, the size, age, race and ethnicity of the population affected; and the baseline rate of occurrence of the adverse health effect being evaluated.

**Incidence.** The total number of adverse health effects in a geographic region in a given unit of time. In BenMAP, this is the total number of adverse health effects avoided due to a change in air pollution levels, often reported for a single year.

**Incidence Rate.** The background rate of new health effects per person over a particular period of time in a given geographic region. The unit of time is typically a day or a year, but could be other periods as well (e.g., warm season). The incidence rate must be expressed at the same time scale as the remainder of the health impact function. For example, a health

impact function quantifying day-to-day changes in premature death must specify a daily death rate.

**Income Growth Adjustment.** Adjusting certain valuation functions to reflect increases in real income over time. Generally, an increase in real income implies an increase in the WTP values used to monetize health effects.

**Inflation Adjustment.** Adjusting monetized benefits occurring over different years to a constant dollar year (e.g., 2020 dollars) to account for historical or projected future increases in prices over time.

**Metadata.**  Data that serves to provide context or additional information about other data. BenMAP stores a minimum set of standardized metadata fields for imported data files (e.g., file name, file date, reference, import date, and description).  For certain data types, additional metadata are recorded.

**Micrograms per Cubic Meter (μg/m³).** The unit of measure for particulate matter in the NAAQS. This unit represents the mass of PM and other pollutants found in a cubic meter of air.

**Model Data.** Pollutant concentration data that are generated by running an air quality model such as CMAQ. This is different from "monitor data," which are based upon observed measurements of pollutant concentrations.

**Monetize.** In the context of human health benefits assessment, this is the practice of expressing society's preferences for avoiding certain health effects as an economic value (e.g., in U.S. dollars). In BenMAP we estimate monetized benefits by using either Willingness to Pay or Cost of Illness valuation functions.

**Monitor Data.** Pollutant concentration data that are collected from an air quality monitor. "Raw" monitor data usually refers to data that are taken directly from measurement networks, with no additional processing of the data. Monitor data are different from "model data," which are based upon numerical predictions from an air quality model. Use of monitor data is not available in the current version of the tool but is expected in future versions.

**Monitoring.** The systematic, long-term assessment of pollutant levels by measuring the quantity and types of certain pollutants in the surrounding outdoor air. The U.S. EPA reports monitoring data, as well as other information related to monitoring, available through its Air Quality System (AQS): https://www.epa.gov/aqs.

**Monte Carlo Simulation.** A technique used in BenMAP to quantify the confidence intervals around mean incidence and economic value estimates by randomly sampling uncertainty distributions related to effect coefficients and/or willingness to pay estimates.

**Morbidity.** A measure of being diseased or afflicted by an illness (generally non-fatal).

**Mortality.** A measure of the number of deaths in a given population.

**National Ambient Air Quality Standards (NAAQS).** The U.S. EPA establishes levels for pollutants that are considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including protection against decreased visibility and against damage to animals, crops, vegetation, and buildings. The U.S. EPA has set NAAQS for six principal pollutants, which are called "criteria" pollutants: carbon monoxide, lead, nitrogen dioxide, ozone, particulate matter ($PM_{2.5}$, $PM_{10}$), and sulfur dioxide.

**Net Present Value (NPV).** The economic value of a stream of monetized benefits discounted back to the starting year of the analysis and summed.

**Odds Ratio**. A quantitative measure reported in epidemiology studies of the relationship between exposure to air pollution and a health outcome. Odds Ratios must be converted to beta coefficients to be used in BenMAP.

**Ozone ($O_3$).** BenMAP focuses on ground-level or "bad" ozone, which is not emitted directly into the air, but is created by chemical reactions between oxides of nitrogen (NOx) and volatile organic compounds (VOCs) in the presence of sunlight. Emissions from industrial facilities and electric utilities, motor vehicle exhaust, gasoline vapors, and chemical solvents are some of the major sources of NOx and VOC. Breathing ozone can trigger a variety of health problems, particularly for children, the elderly, and people of all ages who have lung diseases such as asthma. Ground level ozone can also have harmful effects on sensitive vegetation and ecosystems.

**Particulate Matter.** Particulate matter, also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of multiple components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. Once inhaled, these particles can affect the heart and lungs and cause serious health effects. Includes $PM_{2.5}$ (particles less than 2.5 microns in aerodynamic diameter), $PM_{10}$ (particles less than 10 microns in aerodynamic diameter), and $PM_{10-2.5}$ (particles between 2.5 and 10 microns in aerodynamic diameter).

**Parts per Billion (ppb).** This unit represents the concentration of the pollutant in a billion parts of air. Ozone concentrations in BenMAP are reported in units of ppb.

**Parts per Million (ppm).** This unit represents the concentration of the pollutant in a million parts of air. Carbon monoxide is often measured in units of ppm.

**Population Exposure versus Personal Exposure.** Population (or ambient) exposure refers to the average air pollution level measured in a [grid cell](). In contrast, personal exposure keeps track over the course of a day the exposure individuals encounter in different micro-environments, such as the freeway, outdoors and indoors. BenMAP only represents population exposure.

**Population-weighted Air Quality.** Modeled or monitored ambient concentrations that have been weighted according to the number of people exposed.

**Post-Policy Scenario.** In a modeling study, this is a scenario in which emissions from one or more source sectors are changed (increased or decreased) from a given "pre-policy scenario". The post-policy scenario generally represents air quality levels after a new policy has been implemented.

**Pre-Policy Scenario.** The air quality levels prior to the policy change you are evaluating. The pre-policy scenario is sometimes referred to as "Business as Usual." This scenario is usually considered to be the reference scenario against which to compare a potential "post-policy scenario," in which air quality levels are changed from the baseline levels.

**Prevalence Rate.** The percentage of individuals in a given population who already have a given adverse health condition. Used to calculate changes in health conditions among those who already have a health condition, such as asthmatics.

**Regulatory Impact Analysis (RIA).** A policy tool used to assess the likely effects of a proposed regulation or regulatory change. It usually includes detailed analyses to quantify the costs and benefits of the regulation.

**Relative Risk.** A measure of the change in risk of an adverse health effect associated with an increase in air pollution levels in an epidemiology study. More specifically, relative risk is the ratio of the risk of illness with a higher pollution level to the risk of illness with a lower

pollution level, where the "risk" is defined as the probability that an individual will become ill. Also sometimes referred to as a risk ratio or hazard ratio.

**Shapefile.** A shapefile is a particular type of GIS file that has a *.shp* extension. These files are accompanied by companion files with *.shx* and *.dbf* extensions and can be used to create Shapefile Grid Definitions. See http://www.esri.com/library/whitepapers/pdfs/shapefile.pdf for more information.

**Task Log.** This is a report that contains a record of all the choices involved in creating a particular file. The task log is exported when exporting results files from the BenMAP tool.

**Template.** A saved configuration from a previous BenMAP run, specifying all the necessary details to run a benefits analysis. After loading a template, you may run it as is or make changes to one or more elements prior to running.

**Unit Value.** The estimated mean economic value of avoiding a single case of a particular health effect.

**Valuation Function.** Valuation functions are used by BenMAP to estimate the economic values of changes in the incidence of health effects. They usually include unit value estimates and may also include potential adjustments for inflation, income growth, and discounting.

**Variable Datasets.** Health Impact functions and valuation functions may sometimes refer to variables other than those for which BenMAP automatically calculates values. For example, some valuation functions reference the median income within each area of analysis. To facilitate this, BenMAP stores variables for use in functions; these variables may be used globally (e.g., inflation adjustments) or may vary geographically (meaning they are associated with a particular Grid Definition).

**WTP (Willingness to Pay).** The willingness of individuals to pay for a good or service, such as a reduction in the risk of illness. In general, economists tend to view an individual's WTP for an improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness to accept (WTA) compensation for not receiving an improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits.

# BenMAP

## Environmental Benefits Mapping and Analysis Program

## User's Manual
## Appendices

### December 2022

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# Appendix A.    Monitor Rollback Algorithms

This Appendix explains the algorithms BenMAP applies when you perform a "monitor rollback" to create an air quality surface. The monitor rollback procedure adjusts the air quality monitoring data using simplified rules to reflect hypothetical broad-scale changes in air pollution across a given study area. **This functionality is not implemented in the current version of the web-based tool, though it is planned for a future version.**

# Appendix B.   Algorithms for Estimating Air Pollution Exposure

BenMAP groups counts of individuals into what we refer to as "population grid cells," where the grid cells typically correspond to a regular grid such as those used in an air quality models, or to a grid defined by political boundaries such as the counties of the United States. The default data in the BenMAP web tool allocates the United States population to the same 12km by 12km grid used in the Community Multiscale Air Quality modeling system (CMAQ). BenMAP estimates the air pollution exposure for the population in each grid-cell by assuming that people living within a particular grid-cell experience the same air pollution levels.

The goal of estimating exposure is to provide the necessary input for concentration-response functions, so that BenMAP can estimate the impact of air pollution on adverse health effects. BenMAP can recognize multiple air pollution metrics. Table B-1 lists the types of metrics commonly used in concentration-response functions.

**Table B-1.    Example Metrics Used in Concentration-Response Functions for Criteria Air Pollutants**

| Measurement Frequency | Metric Name | Metric Description |
|---|---|---|
| Daily (e.g., PM$_{2.5}$) | Daily Average | Daily 24-hour average |
| | Annual Average | Average of four quarterly averages. The four quarters are defined as:  Jan-Mar, April-June, Jul-Sep, Oct-Dec. |
| Hourly (e.g., Ozone) | 1-hour Daily Max | Highest hourly value from 12:00 A.M. through 11:59 P.M. |
| | 8-hour Daily Average | Average of hourly values from 9:00 A.M. through 4:59 P.M. |
| | 24-hour Daily Average | Average of hours from 12:00 A.M. through 11:59 P.M. |

Multiple approaches exist to estimate the exposure to air pollution for the people living within a given population grid-cell, including the use of air quality model-based concentrations, and data from ground-level measurements at monitors.

## B.1   Direct Modeling

When using direct modeling data to estimate exposure, BenMAP assumes that all people living within a particular air pollution grid-cell experience the air pollution concentration estimated by the air quality model for the given air quality metric.

## B.2   Monitor Data

An alternative approach is to use air pollution monitoring data, where you may choose the closest monitor data to the center of a grid-cell or spatially interpolate values based on of nearby monitors. **Placeholder: this functionality is planned for a future version of the tool.**

# Appendix C.   Deriving Health Impact Functions

This Appendix provides of an overview regarding the health impact functions that BenMAP uses to estimate the impact of a change in air pollution on adverse health effects. It provides a description of the particular types of health impact functions that BenMAP uses.

The functional form of the relationship between the change in pollutant concentration, $\Delta x$, and the change in population health response (usually an incidence rate), $\Delta y$ depends on the functional form of the C-R function from which it is derived, and this depends on the underlying relationship assumed in the epidemiological study chosen to estimate a given effect. For expository simplicity, the following subsections refer simply to a generic adverse health effect, "y" and uses particulate matter (PM) as the pollutant - that is, $\Delta x = \Delta PM$ - to illustrate how the relationship between $\Delta x$ and $\Delta y$ is derived from each of several different C-R functions.

Estimating the relationship between $\Delta PM$ and $\Delta y$ can be thought of as consisting of three steps:

1.  choosing a functional form of the relationship between PM and y (the C-R function),
2.  estimating the values of the parameters in the C-R function assumed, and
3.  deriving the relationship between $\Delta PM$ and $\Delta y$ (the health impact function) from the relationship between PM and y (the C-R function).

Epidemiological studies have used a variety of functional forms for C-R functions. Some studies have assumed that the relationship between adverse health and pollution is best described by a linear form, where the relationship between y and PM is estimated by a linear

regression in which y is the dependent variable and PM is one of several independent variables. Log-linear regression and logistic regression are other common forms.

Note that the log-linear form used in the epidemiological literature is often referred to as "Poisson regression" because the underlying dependent variable is a count (e.g., number of deaths), believed to be Poisson distributed. The model parameters may be estimated by regression techniques but are often estimated by maximum likelihood techniques. The form of the model, however, is still log-linear.

## C.1  Overview

The relationship between the concentration of a pollutant, x, and the population response, y, is called the concentration-response (C-R) function. For example, the concentration of fine particulate matter ($PM_{2.5}$) may be in $\mu g/m^3$ per day, and the population response may be the number of premature deaths per 100,000 population per day. C-R functions are estimated in epidemiological studies. A functional form is chosen by the researcher, and the parameters of the function are estimated using data on the pollutant (e.g., daily levels of $PM_{2.5}$) and the health response (e.g., daily mortality counts). There are several different functional forms, discussed below, that have been used to estimate C-R functions. The one most commonly used is the log-linear form, in which the natural logarithm of the health response is a linear function of the pollutant concentration.

For the purposes of estimating benefits, we are not interested in the C-R function itself, however, but the relationship between the change in concentration of the pollutant, Δx, and the corresponding change in the population health response, Δy. We want to know, for example, if the concentration of $PM_{2.5}$ is reduced by 10 $\mu g/m^3$, how many premature deaths will be avoided? The relationship between Δx and Δy can be derived from the C-R function, as described below, and we refer to this relationship as a health impact function.

Many epidemiological studies, however, do not report the C-R function, but instead report some measure of the change in the population health response associated with a specific change in the pollutant concentration. The most common measure reported is the relative risk associated with a given change in the pollutant concentration. A general relationship between Δx and Δy can, however, be derived from the relative risk. The relative risk and similar measures reported in epidemiological studies are discussed in the sections below. The derivation of the relationship of interest for BenMAP - the relationship between Δx and Δy - is discussed in the subsequent sections.

## C.2 Review Relative Risk and Odds Ratio

The terms relative risk and odds ratio are related but distinct. Table C-1 provides the basis for demonstrating their relationship.

**Table C-2.     Relative Risk and Odds Ratio Notation**

| Exposure | Fraction of Population | | Adverse Effect Measure | |
|---|---|---|---|---|
| | Affected | Not Affected | Relative Risk | Odds |
| Baseline Pollutant Exposure | $y_0$ | $1-y_0$ | $y_0/y_c$ | $y_0/(1-y_0)$ |
| Control Pollutant Exposure | $y_c$ | $1-y_c$ | | $y_c/(1-y_c)$ |

The "risk" that people with baseline pollutant exposure will be adversely affected (e.g., develop chronic bronchitis) is equal to $y_0$, while people with control pollutant exposure face a risk, $y_0$, of being adversely affected. The relative risk (RR) is simply:

**Equation C-1**

$$RR = \frac{y_0}{y_c}$$

The odds that an individual facing high exposure will be adversely affected is:

**Equation C-2**

$$Odds = \frac{y_0}{1 - y_0}$$

The odds ratio is then:

**Equation C-3**

$$Odds\ Ratio = \frac{\left(\dfrac{y_0}{1 - y_0}\right)}{\left(\dfrac{y_c}{1 - y_c}\right)}$$

This can be rearranged as follows:

**Equation C-4**

$$Odds\ Ratio = \frac{y_0}{y_c} \times \left(\frac{1 - y_c}{1 - y_0}\right) = RR \times \left(\frac{1 - y_c}{1 - y_0}\right)$$

As the risk associated with the specified change in pollutant exposure gets small (i.e., both $y_0$ and $y_c$ approach zero), the ratio of $(1-y_c)$ to $(1-y_0)$ approaches one, and the odds ratio approaches the relative risk. This relationship can be used to calculate the pollutant coefficient in the C-R function from which the reported odds ratio or relative risk is derived, as described below.

# C.3 Linear Model

A linear relationship between the rate of adverse health effects (incidence rate) and various explanatory variables is of the form:

**Equation C-5**

$$y = \alpha + \beta \times PM$$

where α incorporates all the other independent variables in the regression (evaluated at their mean values, for example) times their respective coefficients. The relationship between the change in the rate of the adverse health effect from the baseline rate ($y_0$) to the rate after control ($y_c$) associated with a change from $PM_0$ to $PM_c$ is then:

**Equation C-6**

$$\Delta y = y_0 - y_c = \beta * (PM_0 - PM_c) = \beta * \Delta PM$$

For example, Ostro et al. (1991, Table 5) reported a $PM_{2.5}$ coefficient of 0.0006 (with a standard error of 0.0003) for a linear relationship between asthma and $PM_{2.5}$ exposure.

The lower and upper bound estimates for the $PM_{2.5}$ coefficient are calculated as follows:

**Equation C-7**

$$\beta_{lowerbound} = \beta - \left(1.96 \times \sigma_\beta\right) = 0.0006 - (1.96 \times 0.0003) = 1.2 \times 10^{-5}$$

$$\beta_{upperbound} = \beta + \left(1.96 \times \sigma_\beta\right) = 0.0006 + (1.96 \times 0.0003) = 0.00119$$

It is then straightforward to calculate lower and upper bound estimates of the change in asthma.

# C.4 Log-linear Model

The log-linear relationship defines the incidence rate (y) as:

### Equation C-8

$$y = B \times e^{\beta * PM}$$

Or, equivalently,

### Equation C-9

$$ln(y) = \alpha + \beta * PM,$$

where the parameter B is the incidence rate of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and α = ln(B). 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,

### Equation C-10

$$B = B_0 \times e^{\beta_1 x_1 + \cdots + \beta_n x_n}$$

where Bo is the incidence of y when all covariates in the model are zero, and x1, ... , xn 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 relationship between ΔPM and Δy is:

### Equation C-11

$$\Delta y = y_0 - y_c = B\left(e^{\beta PM_0} - e^{\beta PM_c}\right)$$

This may be rewritten as:

**Equation C-12**

$$\Delta y = B \times e^{\beta \bullet PM_0}\left(1 - e^{-\beta(PM_0 - PM_c)}\right) = y_0\left(1 - \frac{1}{\exp(\beta \times \Delta PM)}\right)$$

where $y_0$ is the baseline incidence rate of the health effect (i.e., the incidence rate before the change in PM).

The change in the incidence of adverse health effects can then be calculated by multiplying the change in the incidence rate, $\Delta y$, by the relevant population (e.g., if the rate is number per 100,000 population, then the relevant population is the number of 100,000s in the population).

When the PM coefficient ($\beta$) and its standard error ($\sigma\beta$) are published (e.g., Ostro et al., 1989), then the coefficient estimates associated with the lower and upper bound may be calculated easily as follows:

**Equation C-13**

$$\beta_{lowerbound} = \beta - \left(1.96 \times \sigma_\beta\right)$$

$$\beta_{upperbound} = \beta + \left(1.96 \times \sigma_\beta\right),$$

where the adjustment on the mean of ±1.96 times the standard error produces the 2.5th and 97.5th percentiles of the normal distribution, which are used to approximate a 95% confidence interval. These values can be changed to capture different lower and upper bounds.

Epidemiological studies often report a relative risk for a given ΔPM, rather than the coefficient, $\beta$ (e.g., Schwartz et al., 1995, Table 4). Recall that the relative risk (RR) is simply the ratio of two risks:

**Equation C-14**

$$RR = \frac{y_0}{y_c} = e^{\beta \bullet \Delta PM}$$

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

**Equation C-15**

$$\beta = \frac{In(RR)}{\Delta PM}$$

The coefficients associated with the lower and upper bounds (e.g., the 2.5[th] and 97.5[th] percentiles) can be calculated by using a published confidence interval for relative risk, and then calculating the associated coefficients.

Because of rounding of the published RR and its confidence interval, the standard error for the coefficient implied by the lower bound of the RR will not exactly equal that implied by the upper bound, so an average of the two estimates is used. The underlying standard error for the coefficient ($\sigma_\beta$) can be approximated by:

**Equation C-16**

$$\sigma_{\beta, 2.5\, percentile} = \frac{\beta - \beta_{2.5\, percentile}}{1.96}$$

$$\sigma_{\beta, 97.5\, percentile} = \frac{\beta_{97.5\, percentile} - \beta}{1.96}$$

$$\sigma_\beta \cong \frac{\sigma_{\beta, 2.5\, percentile} + \sigma_{\beta, 97.5\, percentile}}{2}$$

# C.5 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 vector of explanatory variables, X, the logistic model assumes the probability of an occurrence is:

**Equation C-17**

$$y = prob(occurrence \mid X \times \beta) = \left( \frac{e^{X \bullet \beta}}{1 + e^{X \bullet \beta}} \right),$$

where β is a vector of coefficients. Greene (1997, p. 874) presents models with discrete dependent variables, such as the logit model. See also Judge et al. (1985, p. 763). This may be rewritten as:

**Equation C-18**

$$y = \frac{e^{X \bullet \beta}}{1 + e^{X \bullet \beta}} \times \frac{e^{-X \bullet \beta}}{e^{-X \bullet \beta}} = \frac{1}{1 + e^{-X \bullet \beta}}$$

The odds of an occurrence is:

**Equation C-19**

$$odds = \frac{y}{1-y} = \frac{\left(\dfrac{1}{1+e^{-X\bullet\beta}}\right)}{1-\dfrac{1}{1+e^{-X\bullet\beta}}}$$

$$\Rightarrow \ odds = \frac{\left(\dfrac{1}{1+e^{-X\bullet\beta}}\right)}{1-\dfrac{1}{1+e^{-X\bullet\beta}}} = \frac{\left(\dfrac{1}{1+e^{-X\bullet\beta}}\right)}{\left(\dfrac{e^{-X\bullet\beta}}{1+e^{-X\bullet\beta}}\right)} = \frac{1}{e^{-X\bullet\beta}} = e^{X\bullet\beta}$$

$$\Rightarrow ln(odds) = X \times \beta$$

The odds ratio for the control scenario (odds$_c$) versus the baseline (odds$_0$) is then:

**Equation C-20**

$$odds\ ratio = \frac{odds_c}{odds_0} = \frac{\left(\dfrac{y_c}{1-y_c}\right)}{\left(\dfrac{y_0}{1-y_0}\right)} = \frac{\left(\dfrac{1}{e^{-X_c\bullet\beta}}\right)}{\left(\dfrac{1}{e^{-X_0\bullet\beta}}\right)} = \frac{e^{X_c\bullet\beta}}{e^{X_0\bullet\beta}}$$

The *change* in the probability of an occurrence from the baseline to the control (Δy), assuming that all of the other covariates remain constant, may be derived from this odds ratio:

**Equation C-21**

$$odds\ ratio = \frac{\dfrac{y_0}{1-y_0}}{\dfrac{y_c}{1-yc}} = \frac{e^{X_0 \bullet \beta}}{e^{X_i \bullet \beta}} = e^{\beta \bullet \Delta X} = e^{\beta \bullet \Delta PM}$$

$$\frac{y_c}{1-y_c} = \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}$$

$$y_c = (1-y_c) \times \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}$$

$$y_c + y_c \times \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM} = \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}$$

$$y_c \left(1 + \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}\right) = \frac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}$$

$$y_c = \frac{\dfrac{y_0}{1-y_0} \times e^{-\beta \bullet PM}}{1 + \dfrac{y_0}{1-y_0} \times e^{-\beta \bullet \Delta PM}} = \frac{y_0 \times e^{-\beta \bullet \Delta PM}}{1-y_0+y_0 \times e^{-\beta \bullet \Delta PM}} = \frac{y_0}{(1-y_0) \times e^{\beta \bullet \Delta PM} + y_0}$$

$$\Delta y = y_0 - y_c = y_0 - \frac{y_0}{(1-y_0) \times e^{\beta \bullet \Delta PM} + y_0}$$

$$\Delta Incidence = \Delta y \times pop = y_0 \times \left(1 - \frac{1}{(1-y_0) \times e^{\beta \bullet \Delta PM} + y_0}\right) \times pop$$

When the coefficient (β) and its standard error ($\sigma_\beta$) are published (e.g., Pope et al., 1991, Table 5), then the coefficient estimates associated with the lower and upper bound may be calculated easily as follows:

**Equation C-22**

$$\beta_{lowerbound} = \beta - \left(1.96 \times \sigma_\beta\right)$$

$$\beta_{upperbound} = \beta + \left(1.96 \times \sigma_\beta\right),$$

where the adjustments to the mean of plus or minus 1.96 times the standard error represent the 2.5th and 97.5th percentiles of the normal distribution and are used to approximate a 95% confidence interval. These values can be changed to capture different lower and upper bounds.

Often the logistic regression coefficients are not published, and only the odds ratio corresponding to a specified change in PM is presented (e.g., Schwartz et al., 1994). It is easy to calculate the underlying coefficient as follows:

**Equation C-23**

$$ln(odds\ ratio) = \beta \times \Delta PM$$

$$\Rightarrow \beta = \frac{ln(odds\ ratio)}{\Delta PM}$$

The coefficients associated with the lower and upper bound estimates of the odds ratios are calculated analogously. The underlying standard error for the coefficient ($\sigma_\beta$) can be approximated by:

### Equation C-24

$$\sigma_{\beta, 2.5\ percentile} = \frac{\beta - \beta_{2.5\ percentile}}{1.96}$$

$$\sigma_{\beta, 97.5\ percentile} = \frac{\beta_{97.5\ percentile} - \beta}{1.96}$$

$$\sigma_\beta \cong \frac{\sigma_{\beta, 2.5\ percentile} + \sigma_{\beta, 97.5\ percentile}}{2}$$

Sometimes, however, the relative risk is presented. The relative risk does not equal the odds ratio, and a different procedure should be used to estimate the underlying coefficient. Note that ESEERCO (1994, p. V-21) calculated (incorrectly) the underlying regression coefficient for Abbey et al. (1993, Table 5) by taking the logarithm of the relative risk and dividing by the change in TSP.

The relative risk (RR) is simply:

### Equation C-25

$$RR = \frac{y_0}{y_c},$$

where $y_0$ is the risk (i.e., probability of an occurrence) at the baseline PM exposure and $y_c$ is the risk at the control PM exposure. When the baseline incidence rate ($y_0$) is given, then it is easy to solve for the control incidence rate ($y_c$):

**Equation C-26**

$$y_c = \frac{y_0}{RR},$$

The odds ratio, may then be calculated:

**Equation C-27**

$$odds\,ratio = \frac{\dfrac{y_0}{1-y_0}}{\dfrac{y_c}{1-y_c}}$$

Given the odds ratio, the underlying coefficient (β) may be calculated as before:

**Equation C-28**

$$\beta = \frac{In(odds\,ratio)}{\Delta PM},$$

The odds ratio and the coefficient calculated from it are dependent on the baseline and control incidence rates. Unfortunately, it is not always clear what the baseline and control incidence rates should be. Abbey et al. (1995b, Table 2) reported that there are 117 new cases of chronic bronchitis out of a sample of 1,631, or a 7.17 percent rate. In addition, they reported the relative risk (RR = 1.81) for a new case of chronic bronchitis associated with an annual mean concentration "increment" of 45 µg/m³ of $PM_{2.5}$ exposure.

Assuming that the baseline rate for chronic bronchitis (y0) should be 7.17 percent, the question becomes whether the "increment" of 45 µg/m³ should be added to or subtracted from the existing $PM_{2.5}$ concentration. If added, the control incidence rate ($y_c$) would be

greater than the baseline rate ($y_0$), while subtraction would give a control rate less than the incidence rate. In effect, one might reasonably derive two estimates of the odds ratio:

### Equation C-29

$$odds\ ratio_1 = \frac{\left(\dfrac{y_0}{1-y_0}\right)}{\left(\dfrac{y_c}{1-y_c}\right)} = \frac{\left(\dfrac{1.81\times 0.0717}{1-(1.81\times 0.0717)}\right)}{\left(\dfrac{0.0717}{1-0.0717}\right)} = 1.931$$

$$odds\ ratio_2 = \frac{\left(\dfrac{y_0}{1-y_0}\right)}{\left(\dfrac{y_c}{1-y_c}\right)} = \frac{\left(\dfrac{0.0717}{1-0.0717}\right)}{\left(\dfrac{\dfrac{0.0717}{181}}{1-\dfrac{0.0717}{1.81}}\right)} = 1.873$$

$$\Rightarrow \beta_1 = \frac{In(1.931)}{45} = 0.01462$$

$$\Rightarrow \beta_2 = \frac{In(1.873)}{45} = 0.01394$$

An alternative is to simply assume that the relative risk (1.81) is reasonably close to the odds ratio and calculate the underlying coefficient. It is easy to show that the relative risk equals:

### Equation C-30

$$RR = \frac{y_0}{y_c} = (1-y_0)\times e^{-\Delta PM \bullet \beta} + y_0$$

Assuming that:

**Equation C-31**

$$e^{-\Delta PM \bullet \beta} \cong (1 - y_0) \times e^{-\Delta PM \bullet \beta} + y_0$$

$$\Rightarrow RR \cong e^{-\Delta PM \bullet \beta}$$

It is then possible to calculate the underlying coefficient:

**Equation C-32**

$$\frac{In(RR)}{-\Delta PM} \cong \beta$$

$$\Rightarrow \beta_3 = \frac{In(1.81)}{45} = 0.01319$$

Since this coefficient estimate is based on the assumption that

**Equation C-33**

$$e^{-\Delta PM \bullet \beta} \cong (1 - y_0) \times e^{-\Delta PM \bullet \beta} + y_0 ,$$

it should be used in a C-R function that maintains this assumption. In effect, it should be applied to a log-linear C-R function:

**Equation C-34**

$$\Delta y = \left[ y_0 \times \left( e^{\beta \bullet \Delta PM} - 1 \right) \right]$$

Using the formula for the change in the incidence rate and assuming a 10 µg/m³ decline in PM$_{2.5}$, it is shown that this results in changes within the bounds suggested by the two estimates based on using the estimated odds ratios:

<p align="center">**Equation C-35**</p>

$$\Delta y_1 = \frac{.0717}{(1-0.0717)\times e^{10\times 0.01462} + 0.0717} - 0.0717 = -0.00914$$

$$\Delta y_2 = \frac{.0717}{(1-0.0717)\times e^{10\times 0.01394} + 0.0717} - 0.0717 = -0.00874$$

$$\Delta y_3 = 0.0717 \times \left(e^{-10\times 0.01319} - 1\right) = -0.00886$$

In this instance, it seems that simply using the relative risk to estimate the underlying coefficient results in a good approximation of the change in incidence. Since it is unclear which of the two other coefficients ($\beta_1$ or $\beta_2$) should be used - as the published work was not explicit - the coefficient based on the relative risk and the log-linear functional form is a reasonable approach.

# C.6  Cox Proportional Hazards Model

Use of a Cox proportional hazards model in an epidemiological study results in a C-R function that is log-linear in form. It is often used to model survival times, and as a result, this discussion focuses on mortality impacts.

The Cox proportional hazards model is based on a hazard function, defined as the probability that an individual dies at time t, conditional on having survived up to time t (Collet, 1994, p.

10). More formally, the hazard function equals the probability density function for the risk of dying divided by one minus the cumulative probability density function:

**Equation C-36**

$$h(X,t) = \frac{f(X,t)}{1 - F(X,t)}$$

The proportional hazards model takes the form:

**Equation C-37**

$$h(X,t) = h_0(t)e^{X \bullet \beta},$$

where X is a vector of explanatory variables, β is a vector of coefficients, and $h_0(t)$ is the so-called "baseline hazard" rate. This terminology differs from that used in most of this discussion: this "baseline hazard" is the risk when all of the covariates (X) are set to zero; this is *not* the risk in the baseline scenario.

The Cox proportional hazards model is sometimes termed a "semi-parametric" model, because the baseline hazard rate is calculated using a non-parametric method, while the impact of explanatory variables is parameterized. Collet (1994) details the estimation of Cox proportional hazards models; in particular, see Collet's discussion (pp. 95-97) of nonparametric estimation of the baseline hazard.

Taking the ratio of the hazard functions for the baseline and control scenarios gives the relative risk:

**Equation C-38**

$$RR = \frac{h(X_0, t)}{h(X_c, t)} = \frac{h_0(t)e^{X_0 \bullet \beta}}{h_0(t)e^{X_c \bullet \beta}} = e^{\Delta PM \bullet \beta}$$

,

where it is assumed that the only difference between the baseline and control is the level of PM pollution.

The relative risk is often presented rather than the coefficient β, so it is necessary to estimate β in order to develop the functional relationship between ΔPM and Δy, as described previously for log-linear C-R functions.

# Appendix D.　U.S. Health Incidence & Prevalence Data in BenMAP

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. And 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 and prevalence rates for the health effects considered in this analysis.

## D.1　Mortality

This section describes how we developed county mortality rates for the years 2015 through 2050 to use in BenMAP. First, we describe the source of 2012-2014 baseline mortality data and how we calculated county-level mortality rates. We then describe how we used national-level Census mortality rate projections to develop county-level mortality rate projections for years 2015-2060.

### D.1.1 Mortality Data for 2012-2014

We obtained county-level mortality and population data from 2012-2014 for 11 causes for the contiguous United States by downloading the data from the Centers for Disease Control (CDC) WONDER database (http://wonder.cdc.gov).

Since the detailed mortality data obtained from CDC do not include population, we combined them with U.S. Census Bureau population estimates exported from BenMAP. We then

generated age-, cause-, and county-specific mortality rates using the following formula
(Equation D-1):

### Equation D-1

$$R_{i,j,k} = \frac{D_{i,j,k}(2012) + D_{i,j,k}(2013) + D_{i,j,k}(2014)}{P_{i,k}(2012) + P_{i,k}(2013) + P_{i,k}(2014)}$$

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 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;

### Equation D-2

$$R_{s,i,j} = \frac{D_{T,i,j} - D_{u,i,j}}{P_{s,i,j}}$$

Where $R_{s,i,j}$ is the state average suppressed mortality rate for age group $i$ and cause $j$; $D_{T,i,j}$ is the
total state death count for age group $i$ and cause $j$; $D_{u,i,j}$ is the aggregated state-level
unsuppressed death count for age group $i$ and cause $j$; and $P_{s,i,j}$ is the aggregated population
for age group $i$ and cause $j$ in 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. 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 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 "unreliable" counties.

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, according to the regional definitions shown below in Table D-1. 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."

**Table D-3.    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." 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."

**Table D-4.    National Mortality Rates (per 100 people per year) by Health Effect and Age Group, 2012-2014**

| Mortality Category | ICD-10 codes | Infant* | 1-17 | 18-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75-84 | 85+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mortality, All Cause | All | 0.19242 | 0.01951 | 0.07804 | 0.10665 | 0.17264 | 0.40542 | 0.86162 | 1.79670 | 4.62837 | 13.58034 |
| Mortality, Non-Accidental | A00-R99 | 0.15747 | 0.00949 | 0.01874 | 0.04112 | 0.10876 | 0.33084 | 0.79395 | 1.73208 | 4.49595 | 13.20867 |
| Mortality, Respiratory | J00-J98 | 0.01250 | 0.00102 | 0.00127 | 0.00253 | 0.00570 | 0.02013 | 0.06560 | 0.20585 | 0.57827 | 1.42362 |
| Mortality, Chronic Lung | J40-J47, J67 | 0.00052 | 0.00032 | 0.00040 | 0.00074 | 0.00186 | 0.01033 | 0.04045 | 0.13873 | 0.36008 | 0.68593 |
| Mortality, Lung Cancer | C34 | 0.00000 | 0.00001 | 0.00007 | 0.00033 | 0.00282 | 0.02378 | 0.07992 | 0.19701 | 0.32952 | 0.31820 |
| Mortality, Ischemic Heart Disease | I20-I25 | 0.00018 | 0.00004 | 0.00039 | 0.00234 | 0.01242 | 0.04854 | 0.12174 | 0.25698 | 0.68000 | 2.27271 |
| Mortality, Cardio-Pulmonary | I00-I78, J10-J18, J40-J47, J67 | 0.01365 | 0.00069 | 0.00099 | 0.00214 | 0.00502 | 0.01794 | 0.05877 | 0.18453 | 0.51055 | 1.26213 |
| Mortality, NCD + LRI | ** | 0.08961 | 0.00618 | 0.01168 | 0.02751 | 0.08129 | 0.26214 | 0.63767 | 1.37694 | 3.44731 | 9.47467 |
| Mortality, Lower Respiratory Infection | A48.1, A70, B97.4-B97.6, J09-J15.8, J16, J20-J21, P23.0-P23.4, U04 | 0.00249 | 0.00618 | 0.01168 | 0.00030 | 0.00062 | 0.00112 | 0.00196 | 0.00300 | 0.00758 | 0.02693 |
| Mortality, Cerebro-vascular | G45-G46.8, I60-I63.9, I65-I66.9, I67.0-I67.3, I67.5-I67.6, I68.1-I68.2, I69.0-I69.3 | 0.00097 | 0.00012 | 0.00034 | 0.00096 | 0.00314 | 0.00809 | 0.01455 | 0.02892 | 0.08553 | 0.20863 |
| Mortality, COPD | J40-J44, J47 | 0.00048 | 0.00005 | 0.00004 | 0.00015 | 0.00102 | 0.00904 | 0.03888 | 0.13689 | 0.35661 | 0.67457 |

*We estimate post-neonatal mortality (deaths after the first month) for infants because the health impact function (see Appendix E) estimates post-neonatal mortality. Neonatal deaths were removed from the infant mortality total, and total infant population was used as the denominator in post-neonatal mortality incidence.

**For a full list of codes for non-communicable diseases (NCD) and lower respiratory infections (LRI), see the IHME GBD Code mapping: http://ghdx.healthdata.org/record/ihme-data/gbd-2017-cause-icd-code-mappings.

## D.1.2 Mortality Rate Projections 2015-2060

To estimate age- and county-specific mortality rates in years 2015 through 2060, we calculated annual adjustment factors, based on a series of Census Bureau projected national mortality rates (for all- 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 2013 rate in Table D-3) based on Census Bureau projected life tables.

We then calculated, separately for each age group, the ratio of Census Bureau national mortality rate in year Y (Y = 2014, 2015, ..., 2060) to the 2013 rate. These ratios are shown for selected years in Table D-4.

Finally, to estimate mortality rates in year Y (Y = 2015, 2020, ..., 2060) 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 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 for ages 18-24 to Census Bureau national mortality rate in 2013 for ages 18-24.

**Table D-5.** **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 | 0.192* | 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. BenMAP uses post-neonatal mortality (deaths after the first month, i.e., 0.23 per 100 people) because the health impact function (see Appendix E) estimates post- neonatal mortality. For comparison purpose, we also calculated the rate for all deaths in the first year, which is 0.684 per 100 people).

**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 2012 National Projections," which is available on http://www.census.gov/population/projections/files/methodology/methodstatement12.pdf

**Table D-6.** **Ratio of Future Year 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+ |
|---|---|---|---|---|---|---|---|---|---|---|
| 2015 | 0.93 | 0.93 | 0.96 | 1.02 | 0.96 | 0.96 | 1.01 | 1.02 | 1.03 | 1.00 |
| 2020 | 0.94 | 0.94 | 0.98 | 1.04 | 0.97 | 0.98 | 1.02 | 1.03 | 1.03 | 1.00 |
| 2025 | 0.85 | 0.81 | 0.74 | 0.80 | 0.75 | 0.77 | 0.85 | 0.91 | 0.93 | 0.97 |
| 2030 | 0.81 | 0.75 | 0.66 | 0.70 | 0.67 | 0.69 | 0.78 | 0.86 | 0.89 | 0.92 |
| 2035 | 0.76 | 0.70 | 0.58 | 0.62 | 0.60 | 0.62 | 0.71 | 0.81 | 0.87 | 0.87 |
| 2040 | 0.73 | 0.65 | 0.51 | 0.53 | 0.53 | 0.56 | 0.64 | 0.76 | 0.84 | 0.86 |
| 2045 | 0.70 | 0.60 | 0.45 | 0.46 | 0.46 | 0.50 | 0.58 | 0.71 | 0.80 | 0.86 |
| 2050 | 0.67 | 0.56 | 0.39 | 0.40 | 0.40 | 0.44 | 0.53 | 0.66 | 0.77 | 0.87 |
| 2055 | 0.64 | 0.52 | 0.34 | 0.35 | 0.35 | 0.39 | 0.48 | 0.62 | 0.73 | 0.88 |
| 2060 | 0.61 | 0.48 | 0.30 | 0.30 | 0.31 | 0.34 | 0.43 | 0.58 | 0.70 | 0.87 |

## D.1.3 Race-Stratified Mortality Incidence

To estimate race-stratified and age-stratified incidence rates at the county level, we downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC

WONDER mortality database (https://wonder.cdc.gov/). Race-stratified incidence rates were calculated for the following age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, 75-84 years, and 85+ years.[12] To address the frequent county-level data suppression for race-specific death counts, we stratified the county-level data into two broad race categories, White and Non-White. In a later step, we stratified the non-White incidence rates by race (Black, Asian, Native American) using the relative magnitudes of incidence values by race at the regional level, described in more detail below.

We followed the methods outlined in section D.1.1 with one notable difference in methodology; we included an intermediate spatial scale between county and state for imputation purposes. We designated urban and rural counties within each state using CDC WONDER and, where possible, imputed missing data using the state-urban and state-rural classifications before relying on broader statewide data. We followed methods for dealing with suppressed and unreliable data at each spatial scale as described in section D.1.1.

A pooled non-White incidence rate inherently underestimates the mortality risk for some race groups and overestimates mortality risk for others. To estimate county-level mortality rates by individual race (Black, Asian, Native American), we applied regional race-specific incidence relationships to the county-level pooled non-White incidence rates. We calculated a weighted average of race-specific incidence rates using regional incidence rates for each region/age/race group normalized to one reference population (the Asian race group) and county population proportions based on race-specific county populations from CDC WONDER where available. In cases of population suppression across two or more races per county, we replaced all three race-specific population proportions derived from CDC WONDER with population proportions derived from 2010 Census data in BenMAP.

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[12] Infant mortality dates for race- and ethnicity-stratified datasets do not currently exclude neonatal deaths.

### D.1.4 Ethnicity-Stratified Mortality Incidence

To estimate ethnicity-stratified and age-stratified incidence rates at the county level, we downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC WONDER mortality database (https://wonder.cdc.gov/). Ethnicity-stratified incidence rates were calculated for the following age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, 75-84 years, and 85+ years. We stratified county-level data by two groups, Hispanic and non-Hispanic, and did not stratify further by race due to suppression constraints. We followed the methods outlined in section D.1.1 to deal with suppressed and unreliable data. We also included an intermediate spatial scale between county and state designating urban and rural counties for imputation purposes, described in detail in section D.1.3.

# D.2  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). 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:

**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 categorizes hospital admissions in various ways. Hospitalization admissions are reported as emergency (admitted from the emergency department), urgent (admitted from

another hospital), elective (admitted from another health facility, including long-term care), newborn (admitted for delivery), trauma (not used by all states), or other/missing/invalid. While a substantial subset of the ISA-identified literature evaluating respiratory hospitalizations restricted analyses to emergency hospital admissions (EHAs), all hospital admission baseline incidence data within BenMAP reflects total hospital admissions due to time constraints limiting the ability to stratify incidence by admission type. In general, the vast majority of respiratory and cardiovascular hospitaizations appear to be emergency or urgent admissions. As such, the total hospital admissions rates in BenMAP should largely align with analogous EHA rates (albeit biased upward due to the small share of hospitalizations that are elective).

**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. South Carolina provided county-level 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.

Figure D-1 shows the level of hospitalization data (e.g, discharge-level or state-level) for each state. Note that for some states neither discharge-level, county-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.

## Figure D-1.    Hospitalization Data from HCUP

More information about HCUP can be found at http://www.hcup-us.abrq.gov/

The procedures for calculating hospitalization rates are summarized as follows:

For states with discharge-level data:

- We calculated age-, health effect-, and county-specific hospitalization counts. South Carolina was the only state that, while not providing discharge-level data, did provide county-level data for each age group-effect combination.
- 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-, effect-, and county-specific counts using an adjustment factor obtained as follows:

- o We first counted the number of discharges for a specific health effect in the state including those discharges with missing age or county FIPS.

- o We then counted the number of discharges for the health effect in the state excluding those records with missing age or county FIPS.

- o 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.

- For health outcomes deemed acute (acute myocardial infarction; cerebrovascular events; stroke; pneumonia; lower respiratory infection; acute cases of asthma), we distributed patients within the hospital state in cases where the patient resided out of state. We assume that everyone admitted to the hospital in a given state developed that acute condition while in that state.

- 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 (see Section D.1.1).

For states with summarized state statistics (from HCUPnet) we calculated the state-, age-, effect- 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 effect-specific hospitalization counts in each region from HCUPnet/NIS (we refer to this count for the ith effect in the jth region as "TOTALij")

- For those states in the jth region that do have discharge-level or state-level data, we summed the hospital admissions by effect (we refer to this count for the ith effect in the jth region as "SUB ij").

- We then estimated the hospitalization count for states without discharge or state data for the ith effect in the jth region as TOTALij - SUB ij. Note that while this count is effect- and region- specific, it is not age-specific. We obtained the distribution of hospital admission counts across age groups based on the Central Distributor data and assumed the same distribution for the HCUPnet hospitalizations. We then applied this distribution to the estimated hospital counts (i.e., TOTALij - SUB ij) to obtain effect-, region-, and age-specific counts.

- Using the corresponding age- and region-specific populations in BenMAP from Woods and Poole (2015), we calculated age-specific hospitalization rates for the ith effect in the jth region and applied them to those counties in the region that didn't have discharge-level or state-level data.

The health effects in hospitalization studies are defined using different combinations of ICD codes. Rather than generating a unique baseline incidence rate for each ICD code combination, for the purposes of this analysis, we identified a core group of hospitalization rates from the studies and applied the appropriate combinations of these rates in the health impact functions:

- congestive heart failure (ICD-9 428)

- dysrhythmia (ICD-9 427)

- heart rhythm disturbances (ICD-9 426-427)

- acute myocardial infarction (ICD-9 410)

- ischemic heart disease - 1 (ICD-9 410-414)

- ischemic heart disease - 2 (ICD-9 410-414, 429)

- ischemic heart disease (less myocardial infarction) (ICD-9 411-414)

- all cardiovascular (ICD-9 390-429)

- all cardiovascular (less myocardial infarctions) (ICD-9 390-409, 411-429)

- cardiovascular, cerebrovascular and peripheral vascular diseases (ICD-9 410-414, 429, 426-427, 428, 430-438, 440-449)

- all cardiac outcomes (ICD-9 390-459)

- cerebrovascular events (ICD-9 430-438)

- stroke (ICD-9 431-437)

- peripheral vascular disease - 1 (ICD-9 440-448)

- peripheral vascular disease - 2 (ICD-9 440-449)

- all respiratory (ICD-9 460-519)

- respiratory illness - 1 (ICD-9 466, 480-486, 490-493)

- respiratory illness -2 (ICD-9 464-466, 480-487, 490-492)

- chronic lung disease (ICD-9 490-496)

- chronic lung disease (less asthma) (ICD-9 490-492, 494-496)

- chronic lung disease (less asthma) -2 (ICD-9 490-492, 494, 496)

- chronic lung disease (less asthma) -3 (ICD-9 490-492)

- chronic lung disease (less asthma) -4 (ICD-9 491,492, 494, 496)

- pneumonia (ICD-9 480-486)

- asthma (ICD-9 493)

- lower respiratory infection (ICD-9 466.1, 466.0, 480-487, 490, 510-511)

- respiratory – 1 (ICD-9 491, 492, 493, 496)

- respiratory – 2 (ICD-9 464-466, 480-487, 490-492, 493)

- alzheimer's disease (ICD-9 331.0)

- parkinson's disease (ICD-9 332)

In addition to the hospitalization effects above, we developed a set of county level baseline incidence for one EHA effect, All Respiratory (see Section E.7.8 for epidemiological description). We generated the EHA rates by applying the HCUPnet national ratio of All Respiratory hospitalizations originating from the emergency department (77%) to the county level incidence rates developed from the discharge and state-level data.

For each C-R function, we selected the baseline rate or combination of rates that most closely matches to the study health effect definition. For studies that define chronic lung disease as ICD 490- 492, 494-496, we subtracted the incidence rate for asthma (ICD 493) from the chronic

lung disease rate (ICD 490-496). In some cases, the baseline rate will not match exactly to the health effect definition in the study. For example, Burnett et al. (2001) studied the following respiratory conditions in infants <2 years of age: ICD 464.4, 466, 480-486, 493. For this C-R function we apply an aggregate of the following rates: ICD 464, 466, 480-487, 493. Although they do not match exactly, we assume that relationship observed between the pollutant and study-defined health effect is applicable for the additional codes. Table D-5 presents a summary of the national hospitalization rates for 2014 from HCUP.

**Table D-7.    Hospitalization Rates (per 100 people per year), by Health Effect and Age**

| Hospitalization Category | ICD-9 Code | Age 0-1 | 2-17 | 18-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75-84 | 85+ |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Respiratory* | | | | | | | | | | | |
| All Respiratory | 460-519 | 2.387 | 0.363 | 0.166 | 0.212 | 0.340 | 0.737 | 1.297 | 2.292 | 4.151 | 6.343 |
| Pneumonia | 480-486 | 0.477 | 0.101 | 0.039 | 0.063 | 0.103 | 0.196 | 0.336 | 0.640 | 1.426 | 2.660 |
| Chronic Lung Disease | 490-496 | 0.226 | 0.151 | 0.041 | 0.056 | 0.105 | 0.281 | 0.496 | 0.837 | 1.276 | 1.306 |
| Asthma | 493 | 0.217 | 0.147 | 0.036 | 0.048 | 0.076 | 0.123 | 0.136 | 0.157 | 0.218 | 0.243 |
| *Cardiovascular* | | | | | | | | | | | |
| All Cardiovascular | 390-429 | 0.044 | 0.017 | 0.061 | 0.138 | 0.377 | 0.914 | 1.747 | 3.131 | 5.886 | 8.832 |
| Acute Myocardial Infarction, Nonfatal | 410 | 0.000 | 0.000 | 0.002 | 0.010 | 0.068 | 0.202 | 0.380 | 0.575 | 0.921 | 1.332 |
| Ischemic Heart Disease | 410-414 | 0.000 | 0.000 | 0.002 | 0.014 | 0.105 | 0.350 | 0.689 | 1.090 | 1.570 | 1.734 |
| Dysrhythmia | 427 | 0.016 | 0.005 | 0.014 | 0.025 | 0.057 | 0.145 | 0.319 | 0.684 | 1.357 | 1.917 |
| Congestive Heart Failure | 428 | 0.010 | 0.001 | 0.005 | 0.021 | 0.061 | 0.165 | 0.344 | 0.700 | 1.727 | 3.513 |
| Stroke | 431-437 | 0.009 | 0.003 | 0.007 | 0.021 | 0.070 | 0.199 | 0.417 | 0.816 | 1.639 | 2.488 |
| *Neurological* | | | | | | | | | | | |
| Alzheimer's Disease | 331.0 | 0.000 | 0.000 | 0.00 | 0.00 | 0.00 | 0.0004 | 0.0035 | 0.027 | 0.129 | 0.248 |
| Parkinson's Disease | 332 | 0.000 | 0.000 | 0.00011 | 0.0037 | 0.020 | 0.025 | | | | |

# D.3 Nonfatal Heart Attacks

The relationship between short-term particulate matter exposure and heart attacks was quantified in a case-crossover analysis by Peters et al. (2001). 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 Healthcare Cost and Utilization Project (HCUP), assuming that all heart attacks that are not instantly fatal will result in a hospitalization. Details about HCUP data are described in Section D.2. According to the 2014 HCUP data there were approximately 608,795 hospitalizations due to heart attacks (acute myocardial infarction: ICD-9 410, primary diagnosis). We used rates based on HCUP data over estimates extrapolated from cohort studies because the former is a national database with a larger sample size, which is intended to provide reliable national estimates. The incidence rate calculation is also described in Section D.2 and the incidence rates for AMI hospitalization are presented in Table D-5.

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 estimated number of PM-related acute myocardial infarctions to exclude the number of cases that result in death within the first month. 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.

## D.4  Emergency Department Visits

The data source for emergency department/room (ED or ER) visits is also HCUP, i.e., SID, SEDD, and NEDS. And the types of data providers are also the same as those described in Section D.2. Figure D-2 shows the emergency department data in each state.

**Figure D-2.    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, health effect, 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; we assumed that ER visit rates are reasonably constant across these three years and consider them as 2014 rates.

Instead of using HCUPnet/NIS in the last step as described in Section D.2., we used HCUPnet/NEDS to calculate ER visit rates for states without discharge level or state level data. Table D-6 presents the estimated asthma emergency room rates by health effect and age group.

**Table D-8.    Emergency Department Visit Rates (per 100 people per year) by Health Effect and Age Group**

| Emergency Department Category | ICD-9 Codes | 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 |
| Respiratory | 491-493, 460-466, 477.0-477.9, 480-486, 496, 786.07, 786.09 | 6.069 | 3.214 | 2.837 | 2.332 | 2.447 | 2.418 | 2.908 | 4.382 | 5.651 |
| Cardiovascular | 410-414, 427-428, 433-437, 440.0-440.9, 443-445, 451-453 | 0.030 | 0.107 | 0.212 | 0.496 | 1.151 | 2.023 | 3.451 | 6.726 | 11.028 |
| All Cardiac Outcomes | 390-459 | 0.067 | 0.314 | 0.568 | 1.105 | 2.021 | 3.086 | 4.921 | 9.345 | 14.596 |

# D.5  School Loss Days

Epidemiological studies have examined the relationship between air pollution and a variety of measures of school absence. These measures include: school loss days for all causes, illness- related, and respiratory illness-related. We have two sources of information. The first is the National Center for Education Statistics, which provided an estimate of all-cause school loss days, and the other is the National Health Interview Survey (Adams et al., 1999, Table 47), which has data on different categories of acute school loss days. Table D-7 presents the estimated school loss day rates. Further detail is provided below on these rates.

**Table D-9.    School Loss Day Rates (per student per year)**

| Type | Northeast | Midwest | South | West |
|---|---|---|---|---|
| Respiratory illness-related absences | 1.3 | 1.7 | 1.1 | 2.2 |
| Illness-related absences | 2.4 | 2.6 | 2.6 | 3.7 |
| All-cause | 9.9 | 9.9 | 9.9 | 9.9 |

\* We based illness-related school loss day rates on data from the 1996 NHIS and an estimate of 180 school days per year. This excludes school loss days due to injuries. We based the all-cause school loss day rate on data from the National Center for Education Statistics.

**All-Cause School Loss Day Rate**

Based on data from the U.S. Department of Education (1996, Table 42-1), the National Center for Education Statistics estimates that for the 1993-1994 school year, 5.5 percent of students are absent from school on a given day. This estimate is comparable to study-specific estimates from Chen et al. (2000) and Ransom and Pope (1992), which ranged from 4.5 to 5.1 percent.

**Illness-Related School Loss Day Rate**

The National Health Interview Survey (NHIS) has regional estimates of school loss days due to a variety of acute conditions (Adams et al., 1999). NHIS is a nationwide sample-based survey of the health of the noninstitutionalized, civilian population, conducted by NCHS. The survey collects data on acute conditions, prevalence of chronic conditions, episodes of injury, activity limitations, and self-reported health status. However, it does not provide an estimate of all-cause school loss days.

In estimating illness-related school loss days, we started with school loss days due to acute problems (Adams et al., 1999, Table 47) and subtracted lost days due to injuries, in order to match the definition of the study used in the C-R function to estimate illness-related school absences (Gilliland et al., 2001). We then divided by 180 school days per to estimate illness-related school absence rates per school day. Similarly, when estimating respiratory illness-

related school loss days, we use data from Adams et al. (1999, Table 47). Note that we estimated 180 school days in a year to calculate respiratory illness-related school absence rates per year.

# D.6  Asthma–Related Health Effects

Several studies have examined the impact of air pollution on asthma development or exacerbation. Many of the baseline incidence rates used in the health impact functions are based on study-specific estimates. The baseline rates for the various health effects are described below and summarized in Table D-9. The prevalence of asthma is summarized in Table D-10.

**Table D-10.    Asthma-Related Health Effects Incidence Rates**

| Health Effect | Age | Parameter | Rate | Source |
|---|---|---|---|---|
| New Onset Asthma | 0-4 | Incidence | 0.0234 | Winer et al. (2012, Table 1 & Table 2) |
| New Onset Asthma | 5-11 | Incidence | 0.0111 | |
| New Onset Asthma | 12-17 | Incidence | 0.0044 | |
| New Onset Asthma | 18-34 | Incidence | 0.0040 | |
| New Onset Asthma, Women | 35-44 | Incidence | 0.0051 | |
| New Onset Asthma, Women | 45-54 | Incidence | 0.0046 | |
| New Onset Asthma, Women | 55-64 | Incidence | 0.0059 | |
| New Onset Asthma, Women | 65+ | Incidence | 0.0039 | |
| Asthma Exacerbation, Shortness of Breath, African American | 8-13 | Prevalence | 7.40% | Ostro et al. (2001, p. 202) |
| Asthma Exacerbation, Wheeze, African American | 8-13 | Prevalence | 17.30% | |
| Asthma Exacerbation, Cough, African American | 8-13 | Prevalence | 14.50% | |
| Asthma Symptoms, Shortness of Breath | 5-12 | Prevalence | 18.50% | Lewis et al. (2013, p. 51) |
| Asthma Symptoms, Wheeze | 5-12 | Prevalence | 19.40% | |
| Asthma Symptoms, Cough | 5-12 | Prevalence | 30.10% | |
| Asthma Symptoms, Chest Tightness | 5-12 | Prevalence | 12.70% | |
| Asthma Symptoms, Albuterol Use | 6-13 | Incidence | 2.2 | Rabinovitch et al. (2006, Table 1) |
| Upper Respiratory Symptoms (URS) | 9-11 | Incidence | 124.79 | Pope et al. (1991, Table 2) |

## D.6.1 New Onset Asthma

The annual rate of new asthma onset is estimated from Winer et al. (2012, Table 1 and Table 2). Winer et al., 2012 identify newly diagnosed asthma from the 2006-2008 Asthma Call-Back Survey (ACBS) and Behavioral Risk Factor Surveillance System (BRFSS) as individuals

diagnosed by a doctor, or other health professional, within the 12 months prior to the surveys.

### D.6.2 Shortness of Breath

To estimate the annual rate of new shortness of breath episodes among African-American asthmatics, ages 8-13, we used the rate reported by Ostro et al. (2001, p.202). To estimate the annual rate of new shortness of breath episodes among asthmatic children ages 5-12, we used the rate reported by Lewis et al. (2013, p.51).

### D.6.3 Wheeze

The daily rate of new wheeze episodes among African-American asthmatics, ages 8-13, is reported by Ostro et al. (2001, p.202) as 0.076. We multiplied this value by 100 and by 365 to get the annual incidence rate per 100 people. To estimate the annual rate of new wheeze episodes among asthmatic children ages 5-12, we used the rate reported by Lewis et al. (2013, p.51).

### D.6.4 Cough

The daily rate of new cough episodes among African-American asthmatics, ages 8-13, is reported by Ostro et al. (2001, p.202) as 0.067. We multiplied this value by 100 and by 365 to get the annual incidence rate per 100 people. To estimate the annual rate of new cough episodes among asthmatic children ages 5-12, we used the rate reported by Lewis et al. (2013, p.51).

### D.6.5 Albuterol Use

The average number of albuterol inhaler actuations ('puffs') per day for an asthmatic child, age 6-13, is reported by Rabinovith et al. (2006, Table 1) as 2.2 'puffs' per child per day.

### D.6.6 Upper Respiratory Symptoms

Upper Respiratory Symptoms are defined as one or more of the following: runny or stuffy nose; wet cough; burning, aching, or red eyes. Using the incidence rates for upper respiratory symptoms among asthmatics, published in Pope et al. (1991, Table 2), we calculated a sample size-weighted average incidence rate.

### D.6.7 Asthma Population Estimates

In studies examining the association between air pollution and the development or exacerbation of asthma, oftentimes an estimate of the percent of the population with asthma is required. Asthma percentages were obtained from an American Lung Association (2010b) report summarizing data from NHIS. Table D-10 presents asthma prevalence rates used to define asthmatic populations in the health impact functions.

**Table D-11.  Asthma Prevalence Rates Used to Estimate Asthmatic Populations**

| Population Group | Asthma Prevalence | Source |
|---|---|---|
| All Ages | 7.80% | American Lung Association (2010b, Table 7) |
| <5 | 6.14% | |
| <18 | 9.41% | |
| 5-17 | 10.70% | |
| 18-44 | 7.19% | |
| 45-64 | 7.45% | |
| 65+ | 7.16% | |
| African-American, <5 | 9.98% | American Lung Association (2010b, Table 9) |
| African-American, 5 to 17 | 17.76% | |
| African-American, <18 | 15.53% | American Lung Association* |

*Calculated by ALA for U.S. EPA, based on NHIS data (CDC, 2008).

# D.7  Other Acute and Chronic 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 are likely to increase the uncertainty around the estimate because they are often estimated from a single location using a relatively small sample. These health effects include: acute bronchitis, chronic bronchitis, upper respiratory symptoms, lower respiratory symptoms. Table D-8 presents a summary of these baseline rates.

**Table D-12.   Selected Acute and Chronic Incidence (Cases / Person-Year) & Prevalence (Percentage Population)**

| Health Effect | Age | Parameter | Rate | Source |
|---|---|---|---|---|
| Acute Bronchitis | 8-12 | Incidence | 0.043 | American Lung Association (2002b, Table 11) |
| Chronic Bronchitis | 27+ | Incidence | 0.00378 | Abbey et al. (1993, Table 3) |
| Chronic Bronchitis | 18+ | Prevalence | 4.37% | American Lung Association (2010a, Table 4). The rate numbers may be slightly different from those in Table 4 because we received more current estimates form ALA. |
| | 18-44 | | 3.15% | |
| | 45-64 | | 5.49% | |
| | 65+ | | 5.63% | |
| Lower Respiratory Symptoms (LRS) | 7-14 | Incidence | 0.483 | Schwartz et al. (1994, Table 2) |
| Minor Restricted Activity Days (MRAD) | 18-64 | Incidence | 7.8 | Ostro and Rothschild (1989, p. 243) |
| Work Loss Day (WLD) | 18-64 | Incidence | 2.172 | Adams et al. (1999, Table) U.S. Bureau of the Census (1997, No.22) |
| | 18-24 | | 1.971 | |
| | 25-44 | | 2.475 | |
| | 45-64 | | 1.796 | |

NOTE: 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.

## D.7.1 Acute Bronchitis

The annual rate of acute bronchitis for children ages 5 to 17 was obtained from the American Lung Association (2002b, Table 11). The authors reported an annual incidence rate per person of 0.043, derived from the 1996 National Health Interview Survey.

## D.7.2 Chronic Bronchitis Incidence Rate

The annual incidence rate for chronic bronchitis[13] is estimated from data reported by Abbey et al. (1993, Table 3). The rate is calculated by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995a, Table 1).

Age-specific incidence rates are not available. Abbey et al. (1995a, Table 1) did report the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis. However, they did not report an overall incidence rate for bronchitis by age-group. Since, the cough and sputum types of bronchitis overlap to an unknown extent, we did not attempt to generate age-specific incidence rates for the over-all rate of bronchitis.

## D.7.3 Chronic Bronchitis Prevalence Rate

We obtained the annual prevalence rate for chronic bronchitis from the American Lung Association (2010a, Table 4). Based on an analysis of 2008 National Health Interview Survey data, they estimated a rate of 0.0437 for persons 18 and older; they also reported the following prevalence rates for people in the age groups 18-44, 45-64, and 65+: 0.0315, 0.0549, and 0.0563, respectively.

## D.7.4 Lower Respiratory Symptoms

Lower respiratory symptoms (LRS) are defined as two or more of the following: cough, chest pain, phlegm, wheeze. The proposed yearly incidence rate for 100 people, 43.8, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). The authors did not report

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[13] Please note that this health effect is not regularly considered in U.S. EPA analyses (July 2018).

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. (For example, the 62.5th percentile would have an estimated incidence rate per person per day of 0.145 percent.) We used the latter approach in this analysis.

### D.7.5 Minor Restricted Activity Days (MRAD)

Ostro and Rothschild (1989, p. 243) provide an estimate of the annual incidence rate of MRADs per person of 7.8.

### D.7.6 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, No. 22). The average annual rate of work loss days per individual is 2.17. Using a similar approach, we calculated work-loss-day rates for ages 18-24, 25-44, and 45-64, respectively.

## D.8  Other Health Effect Incidence

Baseline incidence estimates for health effect occurrences other than a hospitalization or emergency department visit are described below, listed in alphabetical order.

## D.8.1 Allergic Rhinitis

Prevalence rates of hay fever/rhinitis are presented by Parker et al. (2009). Parker et al. investigate the associations between long-term ozone exposure and respiratory allergies in children ages 3 to 17 years old. The authors use prevalence data from the NHIS household interview survey and define allergic rhinitis as children with reported hay fever, respiratory allergy, or both within the 12 months prior to the survey. Of the eligible population (72,279), 19.2% of respondents experience allergic rhinitis symptoms within the year prior to the survey, therefore, the national prevalence rate of hay allergic rhinitis is 0.192.

## D.8.2 Lung Cancer

The baseline incidence rates for non-fatal lung cancer were calculated using the existent baseline incidence rate for lung cancer *mortality* in combination with the five-year lung cancer survival rate from NCI (2015). We first used the five-year lung cancer survival rate to calculate the total incidence of lung cancer (both fatal and non-fatal) from the baseline mortality rate using the following formula: baseline mortality rate / (1 – five-year survival rate). We then calculated the incidence of non-fatal lung cancer as the difference between total lung cancer incidence and fatal lung cancer incidence (NCI, 2015). Table D-11 presents the baseline incidence of lung cancer mortality, the SEER five-year survival rate, the estimated total lung cancer incidence, and the estimated non-fatal lung cancer incidence rate by age group.

**Table D-13.    Lung Cancer Incidence Rates**

| Age Group | Annual Lung Cancer Mortality Incidence [A] | Five-Year Survival Rate [B] | Total Lung Cancer Incidence [C] = [A] / (1 - [B]) | Non-fatal Lung Cancer Incidence [D] = [C] – [A] |
|-----------|--------------------------------------------|------------------------------|---------------------------------------------------|--------------------------------------------------|
| 25-34 | 0.0000033 | 34.6% | 0.0000050 | 0.00000175 |
| 35-44 | 0.0000282 | 34.6% | 0.0000431 | 0.00001492 |
| 45-54 | 0.0002378 | 22.1% | 0.0003053 | 0.00006746 |
| 55-64 | 0.0007922 | 20.8% | 0.0010003 | 0.00020805 |
| 65-74 | 0.00019701 | 21.0% | 0.0002494 | 0.00005237 |
| 75-84 | 0.0032952 | 14.9% | 0.0038722 | 0.00057695 |
| 85+ | 0.0031820 | 14.9% | 0.0037391 | 0.00055713 |

## D.8.3 Out of Hospital Cardiac Arrest

The baseline incidence of cardiac arrests occurring outside of the hospital (OHCA) is estimated using the incidence and survival rates reported by Daya et al. (2015). Daya et al. (2015) utilize Resuscitation Outcomes Consortium data to calculate the incidence per 100,000 of OHCA and the survival rate broken down into four age categories, 0 to 17, 18 to 39, 40 to 64, and 65+. We combined the age-specific incidence and survival rates to calculate the baseline incidence for non-fatal OHCA (Table D-12).

**Table D-14.    Out of Hospital Cardiac Arrest Incidence and Survival Rates**

| Age | Annual incidence per 100,000 people | Survival Rate | Annual non-fatal incidence per 100 people |
|-----|-------------------------------------|---------------|--------------------------------------------|
| 0-17 | 10.1 | 8.4% | 0.008 |
| 18-39 | 33.5 | 9.8% | 0.033 |
| 40-64 | 137.3 | 14.9% | 0.205 |
| 65+ | 553.5 | 8.8% | 0.487 |

## D.8.4 Stroke

We developed non-fatal stroke baseline incidence rates using similar data as was utilized to develop the non-fatal OHCA incidence rates. Yao et al. (2019) provide the annual incidence of stroke in 2013 for individuals over 65 years old, as well as the survival rate, broken down by race, gender, and stroke type (hemorrhagic or ischemic). We combined the incidence and survival rates to calculate the rate of non-fatal stroke by gender and race. We then calculated the overall annual baseline incidence rate of stroke in all individuals over the age of 65 by calculating a weighted averaged from the stratified. This resulted in a rate of 0.004 strokes per person per year. Table D-13 presents the stratified incidence and survival rates.

**Table D-15.   Stroke Incidence and Survival Rates**

| Characteristic | Annual incidence per 100,000 people | | Survival Rate | | Non-fatal Incidence per 100 people per year | | | Weight (Study Population) |
|---|---|---|---|---|---|---|---|---|
| | Ischemic | Hemorrhagic | Ischemic | Hemorrhagic | Ischemic | Hemorrhagic | Total | |
| Black Men | 551 | 93 | 92% | 73% | 0.00507 | 0.00068 | 0.00575 | 6,155 |
| White Men | 407 | 75 | 88% | 60% | 0.00358 | 0.00045 | 0.00403 | 54,079 |
| Black Women | 641 | 94 | 91% | 69% | 0.00583 | 0.00065 | 0.00648 | 9,819 |
| White Women | 466 | 77 | 85% | 56% | 0.00396 | 0.00043 | 0.00439 | 78,839 |
| **Weighted Average** | | | | | | | **0.00446** | |

# Appendix E.  Core Particulate Matter Health Impact Functions in BenMAP

In this Appendix, we present the core PM-related health impact functions in BenMAP, i.e., the functions that, as of the current release, U.S. EPA routinely uses in its regulatory analyses. Each sub-section has a table with a brief description of the health impact function and the underlying parameters. Following each table, we present a brief summary of each of the studies and any items that are unique to the study.

Note that Appendix C mathematically derives the standard types of health impact functions 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 each health impact function.

## E.1  Long–term Mortality

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in annual mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure. Table E-1 lists the long-term mortality health impact functions.

**Table E-16.    Core Health Impact Functions for Particulate Matter and Long-Term Mortality**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form |
|---|---|---|---|---|---|---|---|---|---|
| Mortality, All Cause | Wu et al. | 2020 | Nationwide | 65-99 | | Annual | 0.006391 | 0.000383 | Log-linear |
| Mortality, All Cause | Pope et al. | 2019 | Nationwide | 18-99 | | Annual | 0.011333 | 0.001602 | Log-linear |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | $O_3$ | Annual | 0.007046 | 0.000095 | Log-linear |
| Mortality, All Cause | Turner et al. | 2016 | Nationwide | 30-99 | $O_3$ | Annual | 0.005827 | 0.000963 | Log-linear |
| Mortality, All Cause | Woodruff et al. | 2008 | Nationwide | 0-0 | | Annual | 0.005603 | 0.004539 | Logistic |

### E.1.1 Wu et al. (2020)

Wu et al. (2020) evaluated the relationship between long-term PM2.5 exposure and all-cause mortality in more than 68.5 million Medicare enrollees (over the age of 64), using Medicare claims data from 2000-2016 representing over 573 million person-years of follow-up and over 27 million deaths. This cohort included over 20% of the U.S. population and was, at the time of publishing, the largest air pollution study cohort to date. The authors modeled PM2.5 exposure at a 1-km2 grid resolution using a hybrid ensemble-based prediction model that combined three machine learning models and relied on satellite data, land-use information, weather variables, chemical transport model simulation outputs, and monitor data. Wu et al. (2020) fit five different statistical models: a Cox proportional hazards model, a Poisson regression model, and three causal inference approaches (GPS estimation, GPS matching, and GPS weighting). All five statistical approaches provided consistent results; we report the results of the Cox proportional hazards model here. The authors adjusted for numerous individual-level and community-level confounders, and sensitivity analyses suggest that the results are robust to unmeasured confounding bias.

***All-Cause Mortality***

In a single-pollutant model, the coefficient and standard error for PM2.5 are estimated from the hazard ratio (1.066) and 95% confidence interval (1.058-1.074) associated with a change in annual mean PM2.5 exposure of 10.0 ug/m3 (Wu et al. 2020, Table S3, Main analysis, 2000-2016 Cohort, Cox PH).

## E.1.2  Pope et al. (2019)

Pope et al. (2019) examined the relationship between long-term PM2.5 exposure and all-cause mortality in a cohort of 1,599,329 U.S. adults (aged 18-84 years) who were interviewed in the National Health Interview Surveys (NHIS) between 1986 and 2014 and linked to the National Death Index (NDI) through 2015. The authors also constructed a subcohort of 635,539 adults from the full cohort for whom body mass index (BMI) and smoking status data were available. They employed a hybrid modeling technique to estimate annual-average PM2.5 concentrations derived from regulatory monitoring data and constructed in a universal kriging framework using geographic variables including land use, population, and satellite estimates. Pope et al. (2019) assigned annual-average PM2.5 exposure from 1999-2015 to each individual by census tract and utilized complex (accounting for NHIS's sample design) and simple Cox proportional hazards models for the full cohort and the subcohort. We report the results of the complex model for the subcohort, which controls for individual-level covariates including age, sex, race-ethnicity, inflation-adjusted income, education level, marital status, rural versus urban, region, survey year, BMI, and smoking status.

***All-Cause Mortality***

In a single-pollutant model, the coefficient and standard error for PM2.5 are estimated from the hazard ratio (1.12) and 95% confidence interval (1.08-1.15) associated with a change in annual mean PM2.5 exposure of 10.0 ug/m3 (Pope et al. 2019, Table 2, Subcohort).

## E.1.3 Di et al. (2017)

Di et al. (2017) evaluated the relationship between long-term PM$_{2.5}$ exposure and all-cause mortality in nearly 61 million U.S. Medicare enrollees (over the age of 64) through 460 million person-years of follow-up and roughly 22 million observed deaths. This cohort comprised approximately 15% of the total U.S. population, included people living in rural areas, and is one of the largest cohort studies published to date. The authors modeled PM$_{2.5}$ exposure across the contiguous U.S. using a hybrid methodology that included land use regression, satellite data, and monitor data, and resolved estimations to 1 x 1-kilometer areas. Di et al. (2017) used Cox proportional-hazards models with a generalized estimating equation. Adjustment for potential confounding by the co-pollutant O$_3$ was performed, which slightly attenuated the relationship between PM$_{2.5}$ and mortality. The authors also performed statistical testing of the potential for non-linear effects and concluded that the data supported a nearly-linear concentration-response relationship with no signal of a threshold down to at least 5 μg/m$^3$.

***All-Cause Mortality***

In a two-pollutant model, the coefficient and standard error for PM$_{2.5}$ are estimated from the hazard ratio (1.073) and 95% confidence interval of (1.071-1.075) associated with a change in annual mean PM$_{2.5}$ exposure of 10.0 μg/m$^3$ (Di et al., 2017, Table 2 Main Analysis, Cox PH with GEE).

## E.1.4 Turner et al. (2016)

Turner et al. (2016) examined the relationship between long-term PM$_{2.5}$ exposure (1982-2004) and mortality (all-cause, cause-specific) in American Cancer Society Cancer Prevention Study-II participants (aged 30-99 years). Estimated PM$_{2.5}$ concentrations were obtained using monthly PM$_{2.5}$ monitor data (1999-2008) and a national-level hybrid land use regression (LUR) and Bayesian maximum entropy (BME) interpolation model. Turner et al. (2016) utilized random-effects Cox proportional hazard models adjusted *a priori* for individual, socio-demographic, and ecological variables. In addition to adjusting for individual-level and ecological covariates, Turner et al. (2016) also controlled for occupational PM$_{2.5}$ exposure and adjusted for the potential co-pollutants O$_3$ and nitrogen dioxide.

### *All-Cause Mortality*

In a multi-pollutant model, the coefficient and standard error for PM$_{2.5}$ are estimated from the hazard ratio (1.06) and 95% confidence interval of (1.04–1.08) associated with a change of 10.0 μg/m$^3$ in the mean PM$_{2.5}$ exposure level from 1999-2004 (Turner et al., 2016, Table E10 HBM PM$_{2.5}$, MP model, controlling for HBM O$_3$ 1982-2004).

## E.1.5 Woodruff et al. (2008)

Woodruff et al. (2008) examined the relationship between long-term exposure to fine $PM_{2.5}$ air pollution and post-neonatal infant mortality in 3,583,495 births from 96 counties containing >249,999 residents across the U.S. between 1999-2002 using data from the National Center for Health Statistics (NCHS). They linked average $PM_{2.5}$ monitoring data over the first two months of life with 6,639 post neonatal deaths, using logistic regression that incorporated generalized estimating equations (GEE) to estimate the odds ratios for all-cause and cause-specific post-neonatal mortality by exposure to air pollution. The study population experienced a median $PM_{2.5}$ concentration of 14.8 $\mu g/m^3$, with 25% of the population experiencing concentrations below 12 $\mu g/m^3$ and above 18.8 $\mu g/m^3$. The study included an evaluation of the appropriateness of a linear form from analysis based on quartiles of exposure and identified the linear form as a reasonable assumption.

*All-Cause Mortality*

In a single-pollutant model, the coefficient and standard error for $PM_{2.5}$ are estimated from the odds ratio (1.04) and 95% confidence interval of (0.98–1.11) associated with a change of 7 $\mu g/m^3$ in the mean $PM_{2.5}$ exposure level during the first two months of life (Woodruff et al., 2008, Table 4 $PM_{2.5}$ single-pollutant model, all causes).

## E.2  Chronic/Severe Illness

Table E-2 below summarizes the health impact functions used to estimate the relationship between PM$_{2.5}$ and chronic health effects. We present a brief summary of each of the studies below.

### Table E-17.    Core Health Impact Functions for Particulate Matter and Chronic Illness

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Cardiac Arrest | Ensor et al. | 2013 | Houston, TX | 18-99 | | D24HourMean | 0.006376 | 0.002823 | Logistic | |
| Cardiac Arrest | Rosenthal et al. | 2008 | Indianapolis, Indiana | 0-99 | | D24HourMean | 0.00198 | 0.005018 | Logistic | |
| Cardiac Arrest | Silverman et al. | 2010 | New York City | 0-99 | | D24HourMean | 0.003922 | 0.00222 | Logistic | |
| Lung Cancer | Gharibvand et al. | 2017 | Nationwide U.S. and 5 Canadian provinces | 30-99 | | Mean | 0.037844 | 0.013121 | Log-linear | |
| Alzheimer's Disease | Kioumourtzoglou et al | 2016 | 50 Northeastern U.S. cities | 65-99 | | Mean | 0.139762 | 0.017753 | Log-linear | |
| Parkinson's Disease | Kioumourtzoglou et al | 2016 | 50 Northeastern U.S. cities | 65-99 | | Mean | 0.076961 | 0.018905 | Log-linear | |
| Stroke | Kloog et al. | 2012 | New England | 65-99 | | Mean | 0.00343 | 0.001265 | Log-linear | |
| Acute Myocardial Infarction, Nonfatal | Peters et al. | 2001 | Boston, MA | 18-99 | | D24HourMean | 0.024121 | 0.009285 | Logistic | |
| Acute Myocardial Infarction, Nonfatal | Pope et al. | 2006 | Greater Salt Lake City, UT | 0-99 | | D24HourMean | 0.0048 | 0.0019 | Logistic | Index MI and unstable angina |
| Acute Myocardial Infarction, Nonfatal | Sullivan et al. | 2005 | King County, WA | 0-99 | | D24HourMean | 0.0019 | 0.0022 | Logistic | |
| Acute Myocardial Infarction, Nonfatal | Zanobetti and Schwartz | 2006 | Greater Boston, MA | 0-99 | | D24HourMean | 0.0053 | 0.0022 | Logistic | Age range adjusted Admissions through ER visits only. |
| Acute Myocardial Infarction, Nonfatal | Zanobetti et al. | 2009 | 26 U.S. Comm | 0-99 | | D24HourMean | 0.0022 | 0.0006 | Log-linear | Age range adjusted. All Seasons. |
| Acute Myocardial Infarction, Nonfatal | Wei et al. | 2019 | Nationwide | 65-99 | | D24HourMean | 0.0011 | 0.0002 | Logistic | |

## E.2.1 Ensor et al. (2013)

Ensor et al. (2013) studied the association between short-term ambient air pollution ($PM_{2.5}$ and $O_3$) exposure and out-of-hospital cardiac arrest (OHCA). Ensor et al. (2013) gathered medical and demographic data for all ages from an Emergency Medical Services database in Houston, Texas between 2004 and 2011. Authors assessed the medical data and defined out-of-hospital cardiac arrest as emergency medical services performing chest compressions. Authors collected ambient air pollution and weather data from Texas Commission of Environmental Quality monitors and calculated hourly and daily averages for $PM_{2.5}$ and $O_3$. The authors used a time-stratified case crossover analysis and conditional logistic regression.

### *Out-of-Hospital Cardiac Arrest*

In a single-pollutant model, the coefficient and standard error are estimated from a reported excess risk of OHCA of 3.9 percent (95% CI: 0.5 -7.4) for a 6 $\mu g/m^3$ increase in the averaged daily mean $PM_{2.5}$ concentration 0- and 1-days prior to onset (Ensor et al. 2013, Table 4).

## E.2.2  Gharibvand et al. (2017)

Gharibvand et al. (2017) evaluated whether positive associations exist between $PM_{2.5}$ exposure and incidence of lung cancer in non-smokers among the Adventist Health and Smog Study-2 (AHSMOG-2), a group of health-conscious individuals of which 81% are never smokers. Authors collected ambient air pollution data ($PM_{2.5}$ and $O_3$) from the US EPA Air Quality system over two years (January 2000-December 2001). Three a priori factors were added to the models as covariates: time spent outdoors, residence length, and moving distance during follow-up. Authors modeled the association between $PM_{2.5}$ exposure and incidence of lung cancer using a Cox proportional hazards regression, with attained age as the time variable. The authors conducted both a single and a two-pollutant ($PM_{2.5}$ and $O_3$) analyses. The study concluded that each 10 µg/m$^3$ increase in ambient $PM_{2.5}$ concentrations was positively associated with increased lung cancer risks within the single-pollutant and two-pollutant multivariable models with $O_3$.

### *Incidence, Lung Cancer*

In a two-pollutant multivariable model with $O_3$ (including *a priori* covariates), the coefficient and standard error were estimated from a hazard ratio of 1.46 (95% CI: 1.13-1.89) for each 10 µg/m$^3$ increase in mean monthly ambient $PM_{2.5}$ concentrations (Gharibvand et al. 2016, Table 3).

## E.2.3 Kioumourtzoglou et al. (2016)

Kioumourtzoglou et al. (2016) evaluated the potential impact of long-term $PM_{2.5}$ exposure on first hospital admission for dementia, Alzheimer's, or Parkinson's diseases among Medicare beneficiaries (>= 65 years old) in 50 cities in the northeastern U.S. (Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, and Washington, D.C.). Authors retrieved medical data from the Center for Medicaid and Medicare from the years 1999-2010. The study followed enrollees as a cohort, which included annual follow-up records identifying the first hospital admissions for dementia (ICD-9 290), Alzheimer's (ICD-9 331.0), Parkinson's (ICD-9 332), and other cardiovascular comorbidities. With respect to Alzheimer's disease, the study evaluated 9,817,806 Medicare enrollees and included 266,725 cause-specific hospital admissions indicating disease onset. With respect to Parkinson's disease, the study evaluated 9,817,806 Medicare enrollees and included 119,425 cause-specific hospital admissions indicating disease onset. Annual average $PM_{2.5}$ concentrations were estimated for each city using data from the U.S. EPA Air Quality System database. Kioumourtzoglou et al. (2016) fit a time-varying Cox proportional hazards model for each city, using the city-wide annual $PM_{2.5}$ concentrations as the time-varying exposure of interest and a linear term for the calendar year. This eliminated the impact of $PM_{2.5}$ variation by city and any $PM_{2.5}$ trends within cities. The model adjusted for cardiovascular comorbidities, and incorporated a counting process extension which created an observation for each year of follow-up per person. The results were then pooled across individuals and cities.

***Incidence, Alzheimer's Disease (ICD-9 331.0)***

In a single-pollutant model, the coefficient and standard error were estimated from a hazard ratio of 1.15 (95% CI: 1.11-1.19) for a 1 μg/m³ increase in the average annual $PM_{2.5}$ concentrations (Kioumourtzoglou et al. 2016, Table 1).

***Incidence, Parkinson's Disease (ICD-9 332)***

In a single-pollutant model, the coefficient and standard error were estimated from a hazard ratio of 1.08 (95% CI: 1.04-1.12) for a 1 μg/m$^3$ increase in the average annual PM$_{2.5}$ concentrations (Kioumourtzoglou et al. 2016, Table 1).

## E.2.4 Kloog et al. (2012)

Kloog et al. (2012) analyzed the effects of long- and short-term PM$_{2.5}$ exposure on hospital admissions due to strokes with a new PM$_{2.5}$ exposure model in New England (Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont) from 2000 to 2006. We use this health effect as a surrogate for PM$_{2.5}$-attributable stroke incidence. Authors collected medical data from 67,678 adults aged 65 to 99 in the U.S. Medicare program database from 2000 to 2006. They defined all respiratory, cardiovascular disease, stroke, and diabetes based on emergency department visits and primary discharge diagnosis records. Authors used a hybrid exposure technique comprised of daily PM$_{2.5}$ concentration data from aerosol optical depth (AOD) measurements and ambient air monitors from the U.S. EPA and Interagency Monitoring of Protected Visual Improvements (IMPROVE). Authors also obtained land use regressions, meteorological data (National Climatic Data Center), and socioeconomic data (U.S. Census Bureau) matched to zip codes in order to perform land use Poisson regressions.

***Incidence, Stroke (ICD Codes 430-436)***

In a single-pollutant model for patients over the age of 65, the coefficient and standard error were estimated from the percent change (3.49%) and 95% confidence interval (0.09-5.18%) for a 10 μg/m$^3$ increase in the 7-year mean PM$_{2.5}$ concentrations (Kloog et al., 2012, Table 3).

## E.2.5 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 $PM_{10}$, $PM_{10}$-2.5, $PM_{2.5}$, "black carbon", $O_3$, CO, $NO_2$, and $SO_2$ in a case-crossover analysis. For each subject, the case period was matched to three control periods, each 24 hours apart. In univariate analyses, the authors observed a positive association between heart attack occurrence and $PM_{2.5}$ levels hours before and days before onset. The authors estimated multivariate conditional logistic models including two-hour and twenty-four hour pollutant concentrations for each pollutant. They found significant and independent associations between heart attack occurrence and both two-hour and twenty-four hour $PM_{2.5}$ concentrations before onset. Significant associations were observed for $PM_{10}$ as well. None of the other particle measures or gaseous pollutants 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).

***Acute Myocardial Infarction, Nonfatal***

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

**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).

### E.2.6 Pope et al. (2006)

Pope et al. (2006) evaluated the association between short-term exposure to $PM_{2.5}$ 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$^3$ increase in $PM_{2.5}$. The researchers determined that a 10 µg/m$^3$ increase in $PM_{2.5}$ resulted in a 4.5% increase (95% CI: 1.1-8.0) in unstable angina and myocardial infarction.

***Acute Myocardial Infarction, Nonfatal***

In 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$^3$ increase in daily 24-hour mean $PM_{2.5}$ (Pope et al., 2006, Table 3).

**Incidence Rate:** AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.5.

**Population:** All ages

**Adjustment:** See the adjustment description in Section E.2.5.

## E.2.7 Rosenthal et al. (2008)

Rosenthal et al. (2008) examined the effects of short-term $PM_{2.5}$ exposure on out-of-hospital cardiac arrest incidence and whether these effects were connected to demographic data or presence of heart rhythm. Additionally, Rosenthal et al. (2008) compared exposure time and measurement method on the effects of short-term $PM_{2.5}$ exposure and out-of-hospital cardiac arrest incidence. Authors obtained medical data from the Wishard Ambulance Service, a local emergency medical service in Indianapolis, Indiana, from July 2, 2002 to July 7, 2006. The study defined out-of-hospital cardiac arrest using the same criteria as Ensor et al. (2013) and Silverman et al. (2010). Authors collected daily and hourly $PM_{2.5}$ concentrations from two City of Indianapolis monitoring sites and using two separate methods: the Federal Reference Method (FRM) for 24-hour filter samples, and a Federal Equivalence Method (FEM). The authors used a case crossover analysis with conditional logistic regressions in order to study the effects of short-term $PM_{2.5}$ exposure on out-of-hospital cardiac arrest incidence. Rosenthal et al. (2008) found a positive but statistically insignificant association between non-dead on arrival (DOA) out-of-hospital cardiac arrest cases and ambient $PM_{2.5}$ concentrations. Although they also noted a statistically significant positive association when restricted to witnessed, non-DOA out-of-hospital cardiac arrest cases, that subgroup is less applicable to the available baseline incidence rate of non-DOA out-of-hospital cardiac arrest cases.

***Out-of-Hospital Cardiac Arrest***

In a single-pollutant model of all non-DOA OHCA cases, the coefficient and standard error were estimated from a hazard ratio of 1.02 (95% CI: 0.92-1.12) for each 10 $\mu g/m^3$ increase in daily mean $PM_{2.5}$ concentrations, lagged by 0-1 days (Rosenthal et al. 2008, Table 5).

## E.2.8  Silverman et al. (2010)

Silverman et al. (2010) investigated the link between short-term ambient air pollution exposure (PM[2.5], $NO_2$, $SO_2$, $O_3$, and CO) and out-of-hospital cardiac arrest in New York City between 2002 and 2006. Authors obtained medical data from the Emergency Medical Services of the New York City Fire Department for 8,216 subjects aged 0 to 99, average age 65.6 with slightly more men than women. Authors collected air pollution and weather data from the US EPA's Air Quality System monitors within a 20-mile radius of New York City and averaged over 24-hour periods. Authors conducted time series and case crossover analyses with 0- and 1- day lagged air pollution levels and by season.

### *Out-of-Hospital Cardiac Arrest*

In a single-pollutant case-crossover model, the coefficient and standard error were estimated from a relative risk of 1.04 (95% CI: 0.99-1.08) for a 10 µg/m³ increase in the averaged daily mean $PM_{2.5}$ concentration 0- and 1-day prior to onset (Silverman et al. 2010, Table 4).

## E.2.9 Sullivan et al. (2005)

Sullivan et al. (2005) studied the relationship between onset time of acute myocardial infarction and the preceding hourly $PM_{2.5}$ concentrations in 5,793 confirmed cased 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$^3$ increase in PM 2.5 the hour before the 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 $PM_{2.5}$ and onset of myocardial infarction.

### *Acute Myocardial Infarction, Nonfatal*

In 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$^3$ increase in daily 24-hour mean $PM_{2.5}$ lagged 1 day (Sullivan et al., 2005, Table 3).

**Incidence Rate:** AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.5.

**Population:** All ages

**Adjustment:** See the adjustment description in Section E.2.5.

## E.2.10 Zanobetti and Schwartz (2006)

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 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 $NO_2$ with a 12.7% increase 95% CI: 5.8-18.0), $PM_{2.5}$ 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 $PM_{2.5}$ 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.

### *Acute Myocardial Infarction, Nonfatal*

The study looked at hospital admissions of AMI through the ER. Under the assumption that all heart attacks will end in hospitalization, we consider the health effect as heart attack events to be consistent with other studies. In 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 $\mu g/m^3$ increase in daily 24-hour mean $PM_{2.5}$ for an average of the 0- and 1-day lag (Zanobetti A. and Schwartz, 2006, Table 4).

**Incidence Rate:** AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.5.

**Population:** All ages. Note that although Zanobetti and Schwartz (2006) reports results for the 65-99 year old age range, for comparability to other studies, we apply the results to all ages. Since the vast majority of AMIs occur among population 65-99, over-counting may not be an issue when applying the risk coefficient to all ages.

**Adjustment:** See the adjustment description in Section E.2.5.

## E.2.11 Zanobetti et al. (2009)

Zanobetti et al. (2009) examined the relationship between daily PM$_{2.5}$ 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$_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. Overall, the authors found that PM$_{2.5}$ mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. For a 10 μg/m$^3$ increase in 2-day averaged PM$_{2.5}$, 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$_{2.5}$ and cardiovascular admissions was significantly modified when the mass of PM$_{2.5}$ 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.

### *Acute Myocardial Infarction, Nonfatal*

The study looked at hospital admissions of AMI through ER. Under the assumption that all heart attacks will end in hospitalization, we consider the health effect as heart attack events to be consistent with other studies. In 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 μg/m$^3$ increase in 2-day averaged PM$_{2.5}$ (Zanobetti et al., 2009, Table 3).

**Incidence Rate:** AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.5.

**Population:** All ages. Note that although Zanobetti et al. (2009) reports results for the 65-99 year old age range, for comparability to other studies, we apply the results to all ages. Since the vast majority of AMIs occurs among population 65-99, over-counting may not be an issue when applying the risk coefficient to all ages.

**Adjustment:** See the adjustment description in Section E.2.5.

## E.2.12 Wei et al. (2019)

Wei et al. (2019) evaluated the relationship between short-term PM2.5 exposure and hospital admissions for 214 mutually exclusive disease groups, including acute myocardial infarction, in a time-stratified, case-crossover analysis of over 95 million Medicare inpatient hospital claims from 2000-2012. The authors estimated daily PM2.5 levels at a 1-km2 grid cell level using a satellite based, neural network model that was calibrated using monitor data and assigned 0-1 day lagged PM2.5 exposure to each participant by zip code of residence. For each disease group, Wei et al. (2019) created a case crossover dataset that controlled for individual level and zip code level variables, day of the week, seasonality, and long-term time trends. They used conditional logistic regression models to estimate associations between PM2.5 exposure and risk of hospital admission and found positive associations for numerous rarely studied and numerous well-studied disease groups.

### *Acute Myocardial Infarction, Nonfatal*

In a single-pollutant model, the coefficient and standard error are estimated from a reported relative increase in risk (0.11%) and 95% confidence interval (0.07%-0.16%) associated with a 1 ug/m3 increase in 0-1 day lagged PM2.5 exposure (Wei et al. 2019, Figure 3, CCS 100 Acute Myocardial Infarction).

# E.3   Hospitalizations

Table E-3 summarizes the health impacts functions used to estimate the relationship between PM$_{2.5}$ and hospital admissions. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-18.    Core Health Impact Functions for Particulate Matter and Hospital Admissions**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Cardiovascular | Bell et al. | 2015 | 213 U.S. Counties | 65-99 | | D24HourMean | 0.000648 | 0.000089 | Log-linear | |
| Respiratory | Bell et al. | 2015 | 213 U.S. Counties | 65-99 | | D24HourMean | 0.00025 | 0.000120 | Log-linear | |
| Respiratory | Ostro et al. | 2009 | 6 California counties | 0-18 | | D24HourMean | 0.002752 | 0.000772 | Log-linear | |

### E.3.1 Bell et al. (2015)

Bell et al. (2015) investigated the effects of short-term fine particulate matter ($PM_{2.5}$) exposure on respiratory health (ICD-9 464-466, 480-487, 490-492, 493) and cardiovascular health (ICD-9 410, omitting 410.x2; 410-414; 426-427; 428; 429; 430-438; and 440-448) in older adults (>64 years). Authors acquired data for 213 U.S. counties (1999-2010) from the Medicare Claims Inpatient Files for U.S. residents >65 years of age. Authors chose variables including sex, age, county of residence, and cause of hospital admission, as determined by ICD-9 codes. Authors collected $PM_{2.5}$ exposure data from county population-based ambient monitors from the US EPA Air Quality System and averaged for county and day. Data were present for 56.5% of study days due to the sampling schedule of the monitors. Bell et al. (2015) utilized Bayesian hierarchal modeling to examine the links between $PM_{2.5}$ and hospital admissions, running separate models to generate risk models for time lags (0-2 days) and season for any estimated variation in health effects.

***Hospital Admissions, Cardio-, Cerebro- and Peripheral Vascular Disease (ICD Codes 410, omitting 410.x2; 410-414; 426-427; 428; 429; 430-438; and 440-448)***

In a single-pollutant model, the coefficient and standard error are estimated from a percent increase in risk of 0.65% (95% CI: 0.48-0.83%) for an increase of 10 $\mu g/m^3$ in same-day daily mean $PM_{2.5}$ concentrations (Bell et al. 2015, Table 1).

***Hospital Admissions, Respiratory-2 (ICD Codes 490-492, 464-466, 480-487, 493)***

In a single-pollutant model, the coefficient and standard error are estimated from a percent increase in risk of 0.25% (95% CI: 0.01-0.48%) for an increase of 10 $\mu g/m^3$ in same-day daily mean $PM_{2.5}$ concentrations (Bell et al. 2015, Table 1).

## E.3.2  Ostro et al. (2009)

Ostro et al. (2009) estimated the association between ambient $PM_{2.5}$, EC, organic carbon (OC), $NO_3$, and $SO_4$ on hospital admissions for respiratory diseases in children ages 5 to 19. The study used the California Office of Statewide Health Planning and Development, Healthcare Quality and Analysis Division hospitalization data from six California counties for the 2000 to 2003 study period. Ostro et al. (2009) classified hospital admissions into: all respiratory disease (ICD-9 codes 460-519), asthma (ICD-9 code 493), acute bronchitis (ICD-9 code 466), and pneumonia (ICD-9 codes 480-486). They aggregated the hospital admission data to the county level to create a daily time series of admissions for each county. Authors took air quality measurements from the California Air Resources Board, which captured speciated 24-hour average pollutant measurements using a filter-based Met One Speciation Air Sampling System. Meteorological measurements for average daily temperature and relative humidity came from the California Air Resources Board or the California Irrigation Management Information System. Authors analyzed data using a Poisson regression with time, day of the week, temperature, relative humidity, and pollutant as explanatory variables. Ostro et al. (2009) controlled for seasonality and time dependent effects by including a natural spline smoother for the daily time trend and meteorology.

### *Hospital Admissions, All Respiratory (ICD Codes 460-519)*

In a single-pollutant model, the coefficient and standard error are estimated from an excess risk of 4.1% (95% CI: 1.8-6.4%) for a 14.6 µg/m³ increase in the daily mean $PM_{2.5}$ concentrations, lagged by 3 days (Ostro et al. 2009, Table 2, pg. 477).

# E.4  Emergency Room Visits

Table E-4 summarizes the health impacts functions used to estimate the relationship between PM$_{2.5}$ and emergency room visits. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-19.    Core Health Impact Functions for Particulate Matter and Emergency Room Visits**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Cardiovascular | Ostro et al. | 2016 | 8 California cities | 0-99 | | D24Hour Mean | 0.000612 | 0.000422 | Logistic | |
| Respiratory | Krall et al. | 2016 | Atlanta, GA | 0-99 | | D24Hour Mean | 0.000545 | 0.000267 | Log-linear | |
| Respiratory | Krall et al. | 2016 | Birmingham, AL | 0-99 | | D24Hour Mean | 0.000968 | 0.000352 | Log-linear | |
| Respiratory | Krall et al. | 2016 | St. Louis, MO | 0-99 | | D24Hour Mean | 0.000832 | 0.000329 | Log-linear | |
| Respiratory | Krall et al. | 2016 | Dallas, TX | 0-99 | | D24Hour Mean | 0.001353 | 0.000588 | Log-linear | |

## E.4.1 Ostro et al. (2016)

Ostro et al. (2016) investigated the association between short-term, source-specific (vehicular emissions, biomass burning, soil, and secondary $NO_3^-$ and $SO_4$ sources) $PM_{2.5}$ concentrations and emergency department visits for respiratory and cardiovascular diseases in eight cities in California from 2005 to 2008. Authors obtained medical and demographic data from the Office of Statewide Health Planning and Development in California, and diagnosis was defined with ICD-9 codes: all cardiovascular (390-459), ischemic heart disease (410–414), AMI (410), cardiac dysrhythmia (427), and heart failure (428). Ostro et al. (2016) conducted a case cross-over analysis, stratified by year and month, controlling for weather and day of the week covariates. Authors used a county-level logistic regression and random-effects meta-analysis to examine the association between source-specific $PM_{2.5}$ and emergency department visits for respiratory and cardiovascular diseases. Results indicate a positive association between vehicle $PM_{2.5}$ emissions and emergency department visits for all cardiovascular diseases.

***ER Visits, All Cardiac Outcomes (ICD Codes 390-459)***

In a single-pollutant model, the coefficient and standard error were estimated from the excess risk of 0.7% (95% CI: -0.2-1.7%) for a 11.4 μg/m³ (interquartile range) increase in daily mean $PM_{2.5}$ concentration, lagged by 2 days (Ostro et al. 2016, Table 4).

## E.4.2  Krall et al. (2016)

Krall et al. (2016) investigated the associations between short-term, source-specific (traffic and coal combustion) ambient $PM_{2.5}$ exposure and emergency department visits for respiratory diseases in U.S. cities (Atlanta, GA, Birmingham, AL, St. Louis, MO, and Dallas, TX). Authors obtained medical data from hospital electronic billings for emergency department visits due to respiratory disease, identified using ICD-9 codes (460-465, 466, 477, 480-486, 491, 492, 493, 496, 786.07). Authors collected $PM_{2.5}$ concentrations from one ambient air monitor in each of the four cities and gathered meteorological data from the National Climactic Data Center. Krall et al. (2016) estimated source-specific $PM_{2.5}$ using apportionment models, which separate $PM_{2.5}$ sources based on chemical composition. This model also included data on gaseous pollutant concentrations from the Community Multiscale Air Quality (CMAQ) with Tracers model. Krall et al. (2016) used Poisson time series regression models to analyze associations between short-term $PM_{2.5}$ exposure and emergency department visits for respiratory diseases. They then compared source-specific $PM_{2.5}$ exposures across cities to estimate associations with the emergency department visit data. To limit confounders, the authors adjusted models for indicator variables, meteorological variables, and long-term trends in emergency department visits.

***ER Visits, Respiratory (ICD Codes 480-486, 491, 492, 496, 460-465, 466, 477, 493, 786.07)***

In a single-pollutant model, the coefficient and standard error were estimated from a relative risk of 1.005 (95% CI: 1.000-1.010) for Atlanta, GA; 1.009 (95% CI: 1.003-1.015) for Birmingham, AL; 1.008 (95% CI: 1.002-1.014) for St. Louis, MO; and 1.012 (95% CI: 1.002-1.023) for Dallas, TX. All relative risks were calculated for a 9.16 µg/m$^3$ increase in daily mean $PM_{2.5}$ concentrations, lagged by 0 days (Krall et al. 2016, Figure 1).

# E.5  Minor Effects

Table E-5 summarizes the health impacts functions used to estimate the relationship between PM$_{2.5}$ and minor effects. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-20.  Core Health Impact Functions for Particulate Matter and Minor Effects**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form |
|---|---|---|---|---|---|---|---|---|---|
| Work Loss Days | Ostro | 1987 | Nationwide | 18-64 | | D24Hour Mean | 0.004600 | 0.000360 | Log-linear |
| Minor Restricted Activity Days | Ostro and Rothschild | 1989 | Nationwide | 18-64 | Ozone | D24Hour Mean | 0.007410 | 0.000700 | Log-linear |
| Hay Fever/ Rhinitis | Parker et al. | 2009 | Nationwide | 3-17 | Summer O$_3$, PM$_{2.5-10}$, NO$_2$, SO$_2$ | Annual | 0.025464 | 0.009618 | Logistic |

## E.5.1 Ostro (1987)

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

***Work Loss Days***

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

**Equation E-1**

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

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

**Equation E-2**

$$\sigma_{\beta}^{2} = \mathrm{var}\left(\frac{\displaystyle\sum_{i-1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^{2}}}{\displaystyle\sum_{i=1976}^{1981} \frac{1}{\sigma_{\beta_i}^{2}}}\right) = \mathrm{var}\left(\frac{\displaystyle\sum_{i-1976}^{1981} \frac{\beta_i}{\sigma_{\beta_i}^{2}}}{\gamma}\right) = \sum_{i-1976}^{1981} \mathrm{var}\left(\frac{\beta_i}{\sigma_{\beta_i}^{2} \times \gamma}\right)$$

This eventually reduces down to:

**Equation E-3**

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

**Incidence Rate:** daily work-loss-day incidence rate per person ages 18 to 64 = 0.00595 (U.S. Bureau of the Census, 1997, No. 22; Adams et al., 1999, Table 41)

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

## E.5.2 Ostro and Rothschild (1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations. The annual national survey results used in this analysis were conducted in the period 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone has highly variable association with RRADs and MRADs.  Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health effects in most years.

***Minor Restricted Activity Days***

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

**Equation E-4**

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

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

**Equation E-5**

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

This reduces down to:

**Equation E-6**

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

**Incidence Rate:** daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild, 1989, p. 243)

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

## E.5.3 Parker et al. (2009)

Parker et al. (2009) investigated the associations between long-term $PM_{2.5}$ exposure and respiratory allergies in an unrestricted population of children (aged 3-17 years) sampled from the United States National Health Interview Survey. Authors obtained symptom data from participant parents, who reported respiratory allergies on annual surveys. Parker et al. (2009) placed all study participants reporting symptoms of respiratory allergies or hay fever into a combined rhinitis group. Parker et al. (2009) then linked annual averages of $SO_2$, $NO_2$, $PM_{2.5}$, and $PM_{2.5-10}$ and warm season (May to September) $O_3$ averages to participant's addresses through ambient air pollution and meteorological data ($O_3$, $SO_2$, $NO_2$, $PM_{2.5}$, and $PM_{10-2.5}$) collected from US EPA Air Quality System monitors. The authors adjusted their logistic regression models for survey year, poverty-level, race/ethnicity, age, family structure, insurance coverage, usual source of care, education of adult, urban-rural status, region, and median county-level income.

***Incidence, Hay Fever/Rhinitis***

In a multi-pollutant model, the coefficient and standard error were estimated from an odds ratio of 1.29 (95% CI: 1.07-1.56) for a 10 µg/m$^3$ increase in $PM_{2.5}$ concentrations (Parker et al. 2009, Table 4).

# E.6  Asthma–Related Effects

Table E-6 summarizes the health impacts functions used to estimate the relationship between $PM_{2.5}$ and asthma exacerbation. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-21.  Core Health Impact Functions for Particulate Matter and Asthma-Related Effects**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Asthma Symptoms, Albuterol use | Rabinovitch et al. | 2006 | Denver, CO | 6-17 | | D24HourMean | 0.001996 | 0.001477 | Log-linear | Albuterol use |
| Asthma Onset | Tétreault et al. | 2016 | Québec, Canada | 0-17 | | Annual | 0.043672 | 0.000885 | Log-linear | Separate HIFs for ages 0-4; 5-17 |

## E.6.1 Rabinovitch et al. (2006)

Rabinovitch et al. (2006) analyzed the relationship between short-term $PM_{2.5}$ exposure and asthma exacerbation in children. The study followed children, ages 6 to 13 attending the Kunsberg School at the National Jewish Medical Research Center with diagnosed asthma for two consecutive winters from 2001-2003. Authors gave an electronic bronchodilator (albuterol) to the children to capture the frequency of use within a 24-hour period. The children also responded to three questions to determine if they may have an upper respiratory infection (URI), and urine samples were taken to measure urinary leukotriene $E_4$ levels on select days. The authors collected hourly ambient $PM_{2.5}$ levels from the Colorado Department of Health Air Pollution Control Division's Tapered Element Oscillating Microbalance (TEOM) monitor, located 2.7 miles west of the school. Additionally, a Federal Reference Monitor (FRM) located next to the TEOM measured 24-hour $PM_{2.5}$ levels. The authors obtained meteorological data from the Colorado Department of Health Air Pollution Control Division and the National Climatic Data Center. A Poisson regression modeled albuterol use as a function of the morning (12:00am to 11:00 am) maximum hourly $PM_{2.5}$ level or the morning mean hourly $PM_{2.5}$ level. The model used both the TEOM and FRM data, individually, incorporated four lag periods (0 to 2 days and 0- to 2-day average), and included several covariates: temperature, pressure, humidity, time trend, Friday indicator, and URI indicator. Rabinovitch et al. (2006) found that, although the $PM_{2.5}$ pollution levels were well below the National Ambient Air Quality Standards, there is a consistent association between peak ambient $PM_{2.5}$ levels and increased albuterol use in asthmatic children.

***Asthma Symptoms, Albuterol use***

In a single-pollutant model, the coefficient and standard error were estimated from a percentage of use increase of 1.2% (95% CI: -0.6-2.9%) for a 6 $\mu g/m^3$ increase in averaged daily mean $PM_{2.5}$ concentration lagged by 0-, 1-, and 2-days (Rabinovitch et al. 2006, Table 4, pg. 1099).

## E.6.2  Tétreault et al. (2016)

Tétreault et al. (2016) investigated the relationship between childhood asthma onset and long-term pollution exposure ($PM_{2.5}$, $NO_2$, $O_3$) in Quebec, Canada. The authors obtained data from four medical-administrative databases collectively known as Quebec Integrated Chronic Disease Surveillance System (QICDSS) between April 1, 1996 and March 31, 2011. The study defined the onset of asthma as a hospital discharged diagnosis of asthma or two reports of asthma from two separate physicians within a two-year period. The authors used Cox proportional hazard models to estimate the association between asthma onset and pollution exposure, controlling for demographics and socioeconomic status. Time-varying exposure models assessed time-varying exposures to the three pollutants in question. Tétreault et al. (2016) showed that childhood asthma onset may be associated with exposure to $PM_{2.5}$, $NO_2$, and $O_3$.

As the physiology and etiology of lung development in children is similar in children 6-17, we apply the 4-12 year age-striated effect estimate from Tétreault et al. (2016) to children ages 4-17 (Baena-Cagnani et al., 2007, Guerra et al., 2004, Ochs et al., 2004, Sparrow et al., 1991, Trivedi and Denton, 2019).

### *Incidence, Asthma*

In a single-pollutant time-varying model, the coefficient and standard error were estimated from a hazard ratio of 1.33 (95% CI: 1.31-1.34) for a 6.53 µg/m³ (interquartile range) increase in annual $PM_{2.5}$ concentration at the residential address (Tétreault et al. 2016, Table 5).

# E.7 Sensitivity Analysis – General

Table E-7 summarizes the PM$_{2.5}$ health impacts functions considered by EPA to be sensitivity analyses. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-22.    Core Health Impact Functions for Particulate Matter Sensitivity Analyses**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | | Annual | 0.008066 | 0.000118 | Log-linear | Single-pollutant model |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | $O_3$ | Annual | 0.005921 | 0.000096 | Log-linear | Nearest monitor analysis |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | $O_3$ | Annual | 0.007789 | 0.000118 | Log-linear | Cox model with mixed effects |
| Hospital Admissions, Respiratory | Jones et al. | 2015 | New York State | 0-99 | | D24Hour Mean | 0.000800 | 0.000170 | Logistic | HA, Respiratory-1 |
| Incidence, Asthma | McConnell et al. | 2010 | 13 Southern California communities | 4-17 | | Annual | 0.029127 | 0.017732 | Log-linear | |
| Incidence, Asthma | Nishimura et al. | 2013 | 5 Urban regions | 7-21 | | Annual | 0.029559 | 0.069101 | Logistic | Black, Hispanic |
| Mortality, All Cause | Pope et al. | 2015 | Nationwide | 30-99 | | Annual | 0.006766 | 0.000712 | Log-linear | LURBME model |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | Massachusetts | 0-99 | $O_3$ | D24Hour Mean | 0.000499 | 0.000355 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | New Jersey | 0-99 | $O_3$ | D24Hour Mean | 0.001094 | 0.000227 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | New Mexico | 0-99 | $O_3$ | D24Hour Mean | 0.001094 | 0.001943 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | New York | 0-99 | $O_3$ | D24Hour Mean | 0.001094 | 0.000151 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | Florida | 0-99 | $O_3$ | D24Hour Mean | -0.000401 | 0.000307 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | New Hampshire | 0-99 | $O_3$ | D24Hour Mean | -0.001207 | 0.001238 | Logistic | |
| Hospital Admissions, All Cardiac Outcomes | Talbott et al. | 2014 | Washington | 0-99 | $O_3$ | D24Hour Mean | -0.000904 | 0.000540 | Logistic | |
| Mortality, All Cause | Turner et al. | 2016 | Nationwide | 30-99 | | Annual | 0.005827 | 0.000963 | Log-linear | Single-pollutant model |
| Emergency Hospital Admissions, All Respiratory | Zanobetti et al. | 2009 | 26 U.S. communities | 65-99 | | D24Hour Mean | 0.002049 | 0.000437 | Log-linear | |

## E.7.1 Di et al. (2017)

See full study description under Di et al. (2017) in Appendix E, Section E.1.1.

***Mortality, All-Cause (Single-Pollutant Model)***

In a single-pollutant model, the coefficient and standard error for $PM_{2.5}$ are estimated from the hazard ratio (1.084) and 95% confidence interval of (1.081-1.086) associated with a change in annual mean $PM_{2.5}$ exposure of 10.0 μg/m$^3$ (Di et al., 2017, Table 2 Single-pollutant analysis).

***Mortality, All-Cause (Nearest Monitor Analysis)***

In a two-pollutant model, the coefficient and standard error for $PM_{2.5}$ are estimated from the hazard ratio (1.061) and 95% confidence interval of (1.059-1.063) associated with a change in annual mean $PM_{2.5}$ exposure of 10.0 μg/m$^3$ (Di et al., 2017, Table 2 Nearest Monitor Analysis, Cox PH with GEE).

***Mortality, All-Cause (Cox Proportional Hazards Model with Mixed Effects (COXME))***

In a two-pollutant model, the coefficient and standard error for $PM_{2.5}$ are estimated from the hazard ratio (1.081) and 95% confidence interval of (1.078-1.083) associated with a change in annual mean $PM_{2.5}$ exposure of 10.0 μg/m$^3$ (Di et al., 2017, Table S3 Main Analysis, Cox PH with mixed effects (COXME)).

## E.7.2  Jones et al. (2015)

Jones et al. (2015) assessed the impacts of $PM_{2.5}$ and its chemical constituents (sulfate ($SO_4$), ammonium ($NH_4$), nitrate ($NO_3$), elemental carbon (EC), and carbon-only portion of organic carbon aerosol) on respiratory health. The study encompassed all ages, races, and ethnicities with a case-crossover analysis in New York state. Analysis used 24-hour average $PM_{2.5}$ chemical constituent concentrations from the Community Multiscale Air Quality (CMAQ) model, and meteorological data from the National Climactic Data Center. The authors assessed hospital discharge data from the New York State Department of Health State Planning and Research Cooperative System (SPARCS) through principle diagnosis categorized by ICD-9 code (chronic bronchitis (ICD-9 491), emphysema (ICD-9 492), asthma (ICD-9 493), and chronic airway obstruction (ICD-9 496)). Authors used a single pollutant conditional logistic regression model to analyze the respiratory hospital admission and $PM_{2.5}$ chemical constituent data over time and by season. The authors calculated hazard ratios (HRs) using the PHREG procedure in SAS (version 9.2) with 95% confidence intervals from the regression models. Jones et al. (2015) found that $PM_{2.5}$ and its chemical constituents showed significant associations between total $PM_{2.5}$ mass and hospital admissions in the year-round model and for all exposure lags (0-4 days). Of all the $PM_{2.5}$ chemical constituents, sulfate had the strongest association with respiratory hospital admissions, particularly during the summer months. Additionally, sulfate was the largest contributor to the $PM_{2.5}$ total mass (49.9%).

### *Hospital Admissions, Respiratory-1 (ICD-9 Codes 491, 492, 493, 496)*

In a year-round single-pollutant model, the coefficient and standard error were estimated from a hazard ratio of 1.006 (95% CI: 1.003-1.008) for a 7.48 $\mu g/m^3$ increase in daily mean $PM_{2.5}$ concentrations, lagged by 4 days. The model was adjusted for season. (Jones et al. 2015, Figure 2).

### E.7.3  McConnell et al. (2010)

McConnell et al. (2010) examined the association between long-term traffic-related air pollution ($PM_{2.5}$, $PM_{10}$, $O_3$, and $NO_2$) exposure and incident asthma in children. The authors collected data for three years from a cohort of 2,497 kindergarten and first-grade children who entered the Southern California Children's Health Study without asthma or wheeze. McConnell et al. (2010) defined new-onset asthma as physician-diagnosed asthma reported by parents on a yearly questionnaire. While the primary focus of the study was traffic-related air pollution from local vehicle emissions, the authors also utilized ambient air pollution exposure data from central site monitors in each of the 13 communities in the Southern California Children's Health Study. The authors used a multilevel Cox proportional hazards model to estimate the association between ambient air pollution exposure and new-onset asthma, controlling for race/ethnicity, secondhand smoke exposure, and pets in the home. The authors concluded that traffic-related pollution exposure may contribute to an increased risk of new-onset asthma in children.

***Incidence, Asthma***

In a single-pollutant model, the coefficient and standard error were estimated from a hazard ratio of 1.66 (95% CI: 0.91-3.05) for a 17.4 µg/m$^3$ (range of exposure in the 13 communities) increase in annual average $PM_{2.5}$ exposure (McConnell et al. 2010, Table 4).

## E.7.4  Nishimura et al. (2013)

Nishimura et al. (2013) investigated the relationship between long-term early-life pollution exposure ($PM_{2.5}$, $PM_{10}$, $O_3$, $NO_2$, and $SO_2$) and asthma onset in Latino and African American children in five urban areas (Chicago, IL; Bronx, NY; Houston, TX; San Francisco, CA; Puerto Rico). The authors obtained data from the Genes–environments and Admixture in Latino Americans (GALA II) Study and the Study of African Americans, Asthma, Genes and Environments (SAGE II). GALA II and SAGE II are case-control studies that enrolled children with and without asthma. The studies defined case subjects as children with physician-diagnosed asthma plus two or more symptoms of coughing, wheezing, or shortness of breath in the two years before study enrollment while control subjects were children with no reported history of asthma, lung disease, or chronic illness, and no reported symptoms of coughing, wheezing, or shortness of breath in the two years before study enrollment. The authors estimated annual average pollution exposures during the first year of life as well as the first three years of life from self-reported residential histories by calculating inverse distance-squared weighted averages from the four closest U.S. EPA Air Quality System monitoring stations within 50 km. The authors first used regional- and study-specific logistic regression models to estimate the association between asthma diagnosis and pollution exposure, controlling for demographics and socioeconomic status and subsequently combined the regression coefficients into a multi-region estimate using a random-effects meta-analysis. Nishimura et al. (2013) showed that early-life air pollution exposure may increase the risk for asthma development in later childhood for Latino and African American cohorts.

### *Incidence, Asthma*

In a single-pollutant model estimating $PM_{2.5}$ exposure during the first year of life, the coefficient and standard error were estimated from an odds ratio of 1.03 (95% CI: 0.90-1.18)

for a 1 µg/m$^3$ increase in average annual PM$_{2.5}$ levels at the residential address during the first year of life (Nishimura et al. 2013, Figure 2).

## E.7.5  Pope et al. (2015)

Pope et al. (2015) evaluated the relationship between long-term exposure to ambient $PM_{2.5}$ and risk of death from CVD and cardiometabolic disease, including effect modification of the relationship by pre-existing cardiometabolic risk factors, in the ACS Cancer Prevention Study II cohort (ages 30+). $PM_{2.5}$ exposures were estimated at home addresses based on a land use regression model with Bayesian Maximum Entropy kriging of residuals (LURBME). Pope et al. utilized a Cox proportional hazards model controlling for individual-level covariates which included variables that characterized current and former smoking habits, exposure to second -hand cigarette smoke, workplace $PM_{2.5}$ exposure in each subject's main lifetime occupation, self -reported exposure to dust and fumes in the workplace, marital status, level of education, body mass index, consumption of alcohol, and quartile ranges of dietary fat index and quartile ranges of a dietary vegetable/fruit/fiber index. Ecological covariates included median household income; percentage of people with <125% of poverty level income; percentage of unemployed individuals aged ≥16 years; percentage of adults with <12th grade education; and percentage of the population who were Black or Hispanic.

### *Mortality, All-Cause (LURBME)*

In a single-pollutant model, the coefficient and standard error are estimated from the hazard ratio (1.07) and 95% confidence intervals (95% CI: 1.06-1.09) for a 10 µg/m$^3$ increase in monthly $PM_{2.5}$ exposure levels averaged from 1999-2004 (Pope, et al., 2015, Table 1. Cox model with individual-level plus ecological covariates; exposure based on LUR-BME).

## E.7.6  Talbott et al. (2014)

Talbott et al. (2014) assessed daily $PM_{2.5}$ concentrations and hospitalizations for cardiovascular disease in Florida, Massachusetts, New Hampshire, New Jersey, New Mexico, New York, and Washington from 2001 to 2008. The authors gathered hospital discharge data from each state's respective data stewards. Talbott et al. (2014) conducted a time-stratified case-crossover study using hospitalization data for all cardiovascular disease (ICD-9 390-459) and for several specific cardiovascular diseases within the ICD-9 390-459 range. Authors used a downscaling Bayesian space-time modeling approach to combine air monitoring data and air gridded numerical outputs from the Community Multi-Scale Air Quality Model (CMAQ) to predict daily $PM_{2.5}$ concentrations. The authors gathered meteorological data from the CDC Wonder North America Land Data Assimilation System Daily Air Temperatures and Heat Index. Talbott et al. (2014) used conditional logistic regression adjusted for $O_3$ (same day as $PM_{2.5}$) and maximum apparent temperature (same day as admission).

***Hospital Admissions, All Cardiac Outcomes (ICD-9 Codes 390-459)***

In a two-pollutant multivariable model with $O_3$, the coefficient and standard error are estimated from an odds ratio of 1.005 (95% CI: 0.998-1.012) for Massachusetts; 1.011 (95% CI: 1.007-1.016) for New Jersey; 1.011 (95% CI: 0.973-1.050) for New Mexico; 1.011 (95% CI: 1.008-1.014) for New York; 0.996 (95% CI: 0.990-1.002) for Florida; 0.988 (95% CI: 0.965-1.013) for New Hampshire; and 0.991 (95% CI: 0.981-1.002) for Washington. Each odds ratio is for a 10 µg/m³ increase in the averaged daily mean $PM_{2.5}$ concentration 0-, 1-, and 2-day lags (Talbott et al. 2014, Table 3).

## E.7.7 Turner et al. (2016)

See full study description under Turner et al. (2016) in Appendix E, Section E.1.2.

***Mortality, All-Cause (Single-Pollutant Model)***

In a single-pollutant model, the coefficient and standard error for PM$_{2.5}$ are estimated from the hazard ratio (1.06) and 95% confidence interval of (1.04–1.08) associated with a change of 10.0 μg/m$^3$ in the mean PM$_{2.5}$ exposure level from 1999-2004 (Turner et al., 2016, Table E10 HBM PM2.5, 1982-2004).

## E.7.8 Zanobetti et al. (2009)

Zanobetti et al. (2009) examined the relationship between daily $PM_{2.5}$ 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 $PM_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. Overall, the authors found that $PM_{2.5}$ mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. For a 10 μg/m$^3$ increase in 2-day averaged $PM_{2.5}$, 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 $PM_{2.5}$ and cardiovascular admissions was significantly modified when the mass of $PM_{2.5}$ 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.

***Emergency Hospital Admissions, All Respiratory (ICD-9 Codes 460-519)***

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$^3$ increase in 2-day averaged $PM_{2.5}$ (Zanobetti et al. 2009, Table 3).

# E.8  Sensitivity Analysis – At-Risk Populations

Table E-8 summarizes the PM$_{2.5}$ health impacts functions considered by EPA to be sensitivity analyses that characterize risk experienced by certain subpopulations. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table E-23.    Core Health Impact Functions for Particulate Matter Sensitivity Analyses of At-Risk Populations**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|--------|--------|------|----------|-----|---------|--------|------|---------|------|-------|
| ER Visits, Asthma | Alhanti et al. | 2016 | Atlanta, Dallas, St. Louis | 0-4 | | D24HourMean | 0.0025 | 0.0019 | Log-linear | White |
| ER Visits, Asthma | Alhanti et al. | 2016 | Atlanta, Dallas, St. Louis | 0-4 | | D24HourMean | 0.0037 | 0.0012 | Log-linear | Non-White |
| ER Visits, Asthma | Alhanti et al. | 2016 | Atlanta, Dallas, St. Louis | 5-18 | | D24HourMean | 0.0025 | 0.0016 | Log-linear | White |
| ER Visits, Asthma | Alhanti et al. | 2016 | Atlanta, Dallas, St. Louis | 5-18 | | D24HourMean | 0.0049 | 0.0012 | Log-linear | Non-White |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | O$_3$ | Annual | 0.0061 | 0.0001 | Log-linear | Non-Hispanic White |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | O$_3$ | Annual | 0.0110 | 0.0008 | Log-linear | Hispanic White |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | O$_3$ | Annual | 0.0189 | 0.0004 | Log-linear | Black |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | O$_3$ | Annual | 0.0092 | (0.0010) | Log-linear | Asian |
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | O$_3$ | Annual | 0.0095 | 0.0019 | Log-linear | Native American |

# E.8.1 Alhanti et al. (2016)

Alhanti et al. (2016) examined the relationship between daily $PM_{2.5}$ concentrations and emergency room visits for asthma (ICD-9 493, 786.07) among residents of all ages in Atlanta (1993-2009), Dallas (2006-2009), and St. Louis (2001-2007). Patient-level ER visit data were obtained from hospitals in the three cities. Daily 24-hour average $PM_{2.5}$ concentrations were estimated using data from all available monitors in the region including monitors from U.S. EPA AQS in all three cities, as well as the South Eastern Aerosol Research and Characterization (SEARCH) network and Assessment of the Spatial Aerosol Composition (ASACA) network in Atlanta. The authors ran city-specific daily time-series Poisson regression models by age group (0-4, 5-18, 19-39, 40-64, 65-99) and performed additional analysis stratified by race (White, non-White) and sex. Models controlled for temperature, day of the week, holidays, race, age, and sex.

***Emergency Room Visits, Asthma***

In single-pollutant models for ages 0-4, the coefficient and standard error are estimated from the three-city weighted average rate ratio (1.02) and 95% confidence interval (0.99-1.05) for White children and (1.03) and 95% confidence interval (1.01-1.05) for non-White children for a 8 µg/m³ increase in three-day moving average $PM_{2.5}$ concentrations (Alhanti et al. 2016, Supplemental Table 4).

In single-pollutant models for ages 5-18, the coefficient and standard error are estimated from the three-city weighted average rate ratio (1.02) and 95% confidence interval (1.00-1.05) for White children and (1.04) and 95% confidence interval (1.02-1.06) for non-White children for a 8 µg/m³ increase in three-day moving average $PM_{2.5}$ concentrations (Alhanti et al. 2016, Supplemental Table 4).

## E.8.2 Di et al. (2017)

See full study descriptionunder Di et al. (2017) in Appendix E, Section E.1.1.

***Mortality, All-Cause***

In multi-pollutant models, the coefficient and standard error are estimated from a hazard ratio of 1.063 (95% CI: 1.060, 1.065) for White; 1.208 (95% CI: 1.199, 1.217) for Black; 1.096 (95% CI: 1.075, 1.117) for Asian; 1.116 (95% CI: 1.100, 1.133) for Hispanic; and 1.100 (95% CI: 1.060, 1.140) for Native Americans. Each odds ratio is for a 10 μg/m$^3$ increase in annual mean PM$_{2.5}$ exposure (Di et al. 2017, Supplementary Table S3 (GEE, By Race)).

# Appendix F.    Core Ozone Health Impact Functions in BenMAP

In this Appendix, we present the core health impact functions used to estimate ozone-related adverse health effects, i.e., the functions that, as of the current release, U.S. EPA routinely uses in its regulatory analyses. Each sub-section has a table with a brief description of each health impact function and the underlying parameters. Following each table, we present a brief summary of each of the studies and any items that are unique to the study.

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

## F.1   Short-term Mortality

Table F-1 summarizes the core health impacts functions used to estimate the relationship between ozone and mortality. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-24.    Core Health Impact Functions for Ozone and Mortality\***

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|--------|--------|------|----------|-----|---------|--------|------|---------|------|-------|
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | | D1HourMax | 0.000727 | 0.000567 | Log-linear | Warm season. |
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | | D8HourMax | 0.000727 | 0.000567 | Log-linear | Warm season. 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.000822. |
| Long-term Mortality, Respiratory | Turner et al. | 2016 | Nationwide | 30-99 | PM2.5, NO2 | Annual (D8HourMax) | 0.007696 | 0.001176 | Log-linear | Warm season. |
| Mortality, Respiratory | Zanobetti and Schwartz | 2008 | 48 U.S. Cities | 0-99 | | D8HourMax | 0.000827 | 0.000228 | Log-linear | D8HourMean approximated as D8HourMax |

\*Unless otherwise stated, mortality is short-term.

## F.1.1  Katsouyanni et al. (2009)

Katsouyanni et al. (2009) used time series methods to examine the relationship between short-term $O_3$ exposures and mortality across the U.S for all ages. The study utilized mortality data from the National Center for Health Statistics (www.cdc.gov/nchs) for years 1987 through 1996, excluding accidental deaths (i.e., International Classification of Diseases (ICD)-9 800). 90 U.S. cities with population sizes varying from about 250,000 to above 9 million with the largest populations were included. Daily number of deaths ranged from 5 to 198. All 90 cities had daily summer $O_3$ measurements. Investigators conducted extensive simulation studies to test 1) the choice of the smoothing method and basic functions used to estimate the smooth function of time in the city-specific models, and 2) the number of degrees of freedom to be used in the smooth function of time. The investigators also evaluated whether each city should be assigned the same model specification or whether each city-specific model should depend on city-specific characteristics. For the former, the same degrees of freedom (ranging from 1 to 20 df/year of data) were assigned to the smooth function of time for every city. The range was determined by choosing the minimum possible degrees of freedom per year up to a maximum degrees of freedom per year that essentially removed all variation in the data beyond time scales of one week. Also, the collective experience of the investigators indicated that using more than 20 df/year does not substantially affect the risk estimates. For the latter approach, the degrees of freedom for the smooth function of time were chosen separately for each city using a fit criterion, such as the Akaike Information Criterion (AIC), or by minimizing the partial autocorrelation function (PACF) of the residuals. Nonparametric methods underestimated the standard error of the air pollution regression coefficient, penalized splines gave relatively small bias, and PACF in combination with penalized splines performed relatively well in terms of bias. Therefore, the identified risk estimate was a summer-only penalized spline estimate of respiratory mortality.

***Short-term Mortality, Respiratory***

In a single pollutant model, the coefficient and standard error are based on the summer-only penalized spline estimate of respiratory mortality of 0.73% (-0.39, 1.85%) per 10 ppb increase in $O_3$ from distributed lag days (Katsouyanni et al. 2009, Table 24: Distributed Lags; Penalized splines; $O_3$ Results).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

## F.1.2  Turner et al. (2016)

Turner et al. (2016) examined the relationship between long-term $O_3$ exposure (1982-2004) and mortality (all-cause, cause-specific) in American Cancer Society Cancer Prevention Study-II participants (aged 30-99 years). A hierarchal Bayesian space-time model based on National Air Monitoring Stations, State and Local Air Monitoring Stations, and Community Multi-Scale Air Quality model data estimated daily eight-hour maximum ozone concentrations at the participant's address. The models considered meteorological data and levels of other ambient pollutants ($PM_{2.5}$, both regional and near-source, and $NO_2$). Turner et al. (2016) utilized Cox proportional hazard models adjusted *a priori* for individual, socio-demographic, and ecological variables. Notably, the study compared annual mortality with warm-season $O_3$ exposures, so full-year baseline incidence rates will be used with risk estimates from this study.

***Long-term Mortality, Respiratory***

In a multi-pollutant model, the coefficient and standard error are based on the warm-season specific hazard ratio of 1.08 (1.06-1.11) per 10 ppb increase in seasonal average of daily 8-hour maximum $O_3$ concentrations (Turner et al. 2016, Table E9: Diseases of the respiratory system (cause of death), HBM $O_3$ (multipollutant model data, fully adjusted HR)).

### F.1.3 Zanobetti and Schwartz (2008)

Zanobetti and Schwartz (2008) investigated the effects of short-term $O_3$ exposure on mortality (all-cause, cardiovascular, stroke, and respiratory) in an unrestricted population of children, adults, and older adults (aged 0-99 years). Between 1998 and 2000, the authors collected mortality data from the National Center for Health Statistic in 48 cities across the United States. Along with eight-hour ozone concentrations and meteorological data obtained from US EPA's Air Quality System Technology Transfer Network, the authors utilized a generalized linear model with quasi Poisson link functions to estimate the effects of short-term ozone on respiratory mortality. The model adjusted for season, day of the week, and temperature. Since ozone concentrations vary between seasons, the authors decided to restrict their analysis to ozone warm season (June - August).

***Short-term Mortality, Respiratory***

In a single pollutant model, the coefficient and standard error are based on the warm season excess risk estimate of 0.83% (95% CI: 0.38-1.28%) for an increase of 10 ppb in daily 8-hour mean $O_3$ concentrations over a summed lag structure of zero to three days (Zanobetti and Schwartz, 2008, Table 1).

The D8HourMean metric is approximated as D8HourMax in this function.

# F.2 Hospital Admissions

Table F-2 summarizes the core health impact functions used to estimate the relationship between ozone and hospital admissions. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-25.   Core Health Impact Functions for Ozone and Hospital Admissions**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| All Respiratory | Katsouyanni et al. | 2009 | 14 U.S. Cities | 65-99 | | D1HourMax | 0.000280 | 0.000176 | Log-linear | Warm season |
| All Respiratory | Katsouyanni et al. | 2009 | 14 U.S. Cities | 65-99 | | D8HourMax | 0.000280 | 0.000176 | Log-linear | Warm season. 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.000316. |

## F.2.1  Katsouyanni et al. (2009)

Katsouyanni et al. (2009) used time series methods to examine the relationship between daily $O_3$ concentrations and hospital admissions in North America. For U.S. benefits estimation purposes, we focus on analyses performed using the U.S hospital admission datasets. These datasets included 14 cities with populations between 291,000 and 5,377,000 between 1987-1996 with city-wide daily 1-hour maximum $O_3$ concentrations ranging from ~34-60 ppb. The authors used a first stage analysis protocol that used generalized linear models with either penalized or natural splines to adjust for seasonality, with varying degrees of freedom. The number of degrees of freedom were also chosen by minimizing the partial autocorrelation function of the model's residuals. Model specification approach accounted for seasonal patterns, weekend and vacation effects, and epistemics of respiratory disease. Data were also analyzed to detect potential thresholds in the concentration-response relationships. The second stage analysis used pooling approaches and assessed potential effect modification by sociodemographic characteristic and indicators of the pollution mixture across study regions.

***Hospital Admissions, All Respiratory (ICD-9 Codes 460-519)***

In a two-pollutant model including $PM_{10}$, the coefficient and standard error are based on the warm season excess risk estimate of 0.28% (-0.07, 0.62%) per 10 ppb increase in $O_3$ averaged over lags 0-1 day (Katsouyanni et al., 2009, Table 40: Average of Lags 0-1 day; Penalized splines).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

# F.3   Emergency Room Visits

Table F-3 summarizes the core health impacts functions used to estimate the relationship between ozone and emergency room (ER) visits. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-26.   Core Health Impact Functions for Ozone and Emergency Room Visits**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Respiratory | Barry et al. | 2018 | Atlanta, GA | 0-99 | | D8HourMax | 0.00118 | 0.00040 | Log-linear | All year |
| Respiratory | Barry et al. | 2018 | Birmingham, AL | 0-99 | | D8HourMax | 0.00118 | 0.00059 | Log-linear | All year |
| Respiratory | Barry et al. | 2018 | Dallas, TX | 0-99 | | D8HourMax | 0.00195 | 0.00049 | Log-linear | All year |
| Respiratory | Barry et al. | 2018 | Pittsburgh, PA | 0-99 | | D8HourMax | 0.00118 | 0.00040 | Log-linear | All year |
| Respiratory | Barry et al. | 2018 | St. Louis, MO-IL | 0-99 | | D8HourMax | 0.00079 | 0.00030 | Log-linear | All year |

## F.3.1 Barry et al. (2018)

Barry et al. (2018) investigated the effects of short-term ozone exposure on emergency department visits for respiratory disease (ICD-9 493, 786.07, 460-466, 477, 491, 492, 496, 480–486, 466.1, 466.11, 466.19) in an unrestricted population of children, adults, and older adults (aged zero-99 years) within five cities (Atlanta, GA, Birmingham, AL, Dallas, TX, Pittsburgh, PA, and St. Louis, MO-IL) across the United States. Authors obtained individual-level health data from hospitals and hospital associations in each of the five cities. Models fusing air quality monitor data with Community Multi-Scale Air Quality modeled data at 12 x 12-km grids were used to estimate ozone exposure. Barry et al. (2018) assessed associations with short-term ozone exposure with daily eight-hour maximum ozone concentrations. The authors implemented Poisson log-linear models to estimate risk values with three day moving averages.

***ER Visits, Respiratory (ICD-9 Codes 493, 786.07, 460-466, 477, 491, 492, 496, 480–486, 466.1, 466.11, 466.19)***

In single-pollutant models, the coefficient and standard error are based on rate ratios of 1.03 (95% CI: 1.01-1.05) in Atlanta, GA, 1.03 (95% CI: 1.00-1.06) in Birmingham, AL, 1.05 (95% CI: 1.02-1.07) in Dallas TX, 1.03 (95% CI: 1.01-1.05) in Pittsburgh, PA, and 1.02 (95% CI: 1.01-1.04) in St. Louis, MO-IL for an increase of 25 ppb in full-year 8-hour daily maximum $O_3$ concentrations (three day moving average) (Barry et al. 2018, Table 3).

## F.4 Minor Effects

Table F-4 summarizes the core health impacts functions used to estimate the relationship between ozone and minor effects. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-27.    Core Health Impact Functions for Ozone and Minor Effects**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| School Loss Days, All Cause | Gilliland et al. | 2001 | Southern California | 5-17 | | D8HourMax | 0.007824 | 0.004445 | Log-linear | All year, 8-hour max from 8-hour mean. |
| Minor Restricted Activity Days | Ostro and Rothschild | 1989 | Nationwide | 18-64 | $PM_{2.5}$ | D1HourMax | 0.002200 | 0.000658 | Log-linear | |
| Minor Restricted Activity Days | Ostro and Rothschild | 1989 | Nationwide | 18-64 | $PM_{2.5}$ | D8HourMax | 0.002200 | 0.000658 | Log-linear | 8-hour max from 1-hour max using adjustment factor of 1.14, resulting in effective beta of 0.002508. |
| Hay Fever/ Rhinitis | Parker et al. | 2009 | Nationwide | 3-17 | $PM_{2.5}$, $PM_{2.5-10}$, $NO_2$, $SO_2$ | Annual (D24HourMean) | 0.01655 | 0.00390 | Logistic | Warm season; long term |
| Hay Fever/ Rhinitis | Parker et al. | 2009 | Nationwide | 3-17 | $PM_{2.5}$, $PM_{2.5-10}$, $NO_2$, $SO_2$ | Annual (D8HourMax) | 0.01655 | 0.00390 | Logistic | Warm season; long term; 8-hour max from 24-hour mean using adjustment factor of 0.654, resulting in effective beta of 0.010818. |

## F.4.1 Gilliland et al. (2001)

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

### *School Loss Days*

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

### Equation F-1

$$Duration = \frac{TotalAbsences}{NewAbsences}$$

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

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

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

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

In addition, not all children are at-risk for a new school absence, as defined by the study. On average, 5.5% of school children are absent from school on a given day (U.S. Department of Education, 1996, Table 42-1). Only those who are in school on the previous day are at risk for

a new absence (1-0.055 = 94.5%). As a result, a factor of 94.5% is used in the function to estimate the population of school children at-risk for a new absence.

**Incidence Rate:** daily school absence rate = 0.055 (U.S. Department of Education, 1996, Table 42-1)

**Population:** population of children ages 9-10 not absent from school on a given day = 94.5% of children ages 9-10 (The proportion of children not absent from school on a given day (5.5%) is based on 1996 data from the U.S. Department of Education (1996, Table 42-1).)

**Scaling Factor:** Proportion of days that are school days in the ozone season = 0.393.

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

## F.4.2 Ostro and Rothschild (1989)

Ostro and Rothschild (1989) estimated the impact of $PM_{2.5}$ and ozone on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The study population is based on the Health Interview Survey (HIS), conducted by the National Center for Health Statistics. In publications from this ongoing survey, non-elderly adult populations are generally reported as ages 18-64. From the study, it is not clear if the age range stops at 65 or includes 65 year olds. We apply the C-R function to individuals ages 18-64 for consistency with other studies estimating impacts to non-elderly adult populations. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average ozone had a highly variable association with RRADs and MRADs. Controlling for ozone, two-week average $PM_{2.5}$ was significantly linked to both health effects in most years. The C-R function for ozone is based on the co-pollutant model with $PM_{2.5}$.

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

***Minor Restricted Activity Days***

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

**Incidence Rate:** daily incidence rate for minor restricted activity days (MRAD) = 0.02137 (Ostro and Rothschild, 1989, p. 243)

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

The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight. The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro (1987).

**Equation F-2**

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

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

**Equation F-3**

$$\sigma_\beta^2 = \mathrm{var}\left( \frac{\sum_{i=1976}^{1981} \dfrac{\beta_i}{\sigma_{\beta_i}^2}}{\sum_{i=1976}^{1981} \dfrac{1}{\sigma_{\beta_i}^2}} \right) = \mathrm{var}\left( \frac{\sum_{i=1976}^{1981} \dfrac{\beta_i}{\sigma_{\beta_i}^2}}{\gamma} \right) = \sum_{i=1976}^{1981} \mathrm{var}\left( \frac{\beta_i}{\sigma_{\beta_i}^2 \times \gamma} \right)$$

This reduces down to:

**Equation F-4**

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

### F.4.3 Parker et al. (2009)

Parker et al. (2009) investigated the associations between long-term $O_3$ exposure and respiratory allergies in an unrestricted population of children (aged 3-17 years) sampled from the United States National Health Interview Survey. Authors obtained symptom data from participant parents, who reported respiratory allergies on annual surveys. Parker et al. (2009) placed all study participants reporting symptoms of respiratory allergies or hay fever into a combined rhinitis group. Parker et al. (2009) linked annual averages of $SO_2$, $NO_2$, $PM_{2.5}$, and $PM_{2.5-10}$ and warm season (May to September) $O_3$ averages to participant's addresses through ambient air pollution and meteorological data collected from US EPA Air Quality System monitors. The authors adjusted their logistic regression models for survey year, poverty-level, race/ethnicity, age, family structure, insurance coverage, usual source of care, education of adult, urban-rural status, region, and median county-level income.

***Incidence, Hay Fever/Rhinitis***

In a multi-pollutant model, the coefficient and standard error are based on the odds ratio of 1.18 (95% CI: 1.09-1.27) for a 10 ppb increase in 24-hour mean, warm season $O_3$ (Parker et al., 2009, Table 4).

The Health Impact Function was adjusted from the daily 24-hour mean metric to the daily 8-hour max metric using a ratio of 1/1.53 = 0.65359 (inverse of the ratio of 8-hour max to 24-hour mean ozone) (Anderson and Bell 2010, Table 2).

# F.5 Asthma–Related Effects

Table F-5 summarizes the core health impacts functions used to estimate the relationship between ozone and asthma exacerbation. Below, we present a brief summary of each of the studies and any items that are unique to the study. Based on advice from the SAB-HES (U.S. EPA-SAB 2004), regardless of the age ranges included in the source epidemiology studies, we extend the applied population to ages 6 to 18, reflecting the common biological basis for the effect in children in the broader age group.

**Table F-28.    Core Health Impact Functions for Ozone and Asthma-Related Effects**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Asthma Symptoms, Cough | Lewis et al. | 2013 | Detroit, MI | 5-12 | | D8HourMax | 0.00708 | 0.00372 | Logistic | All year |
| Asthma Symptoms, Wheeze | Lewis et al. | 2013 | Detroit, MI | 5-12 | | D8HourMax | 0.00764 | 0.00410 | Logistic | All year |
| Asthma Symptoms, Chest tightness | Lewis et al. | 2013 | Detroit, MI | 5-12 | | D8HourMax | 0.01140 | 0.00505 | Logistic | All year |
| Asthma Symptoms, Shortness of breath | Lewis et al. | 2013 | Detroit, MI | 5-12 | | D8HourMax | 0.00423 | 0.00386 | Logistic | All year |
| Asthma Onset | Tétreault et al. | 2016 | Québec, Canada | 0-17 | | Annual (D8HourMax) | 0.02075 | 0.00146 | Log-linear | Warm season; separate HIFs for ages 0-4; 5-17 |

## F.5.1 Lewis et al. (2013)

Lewis et al. (2013) studied the effects of short-term $O_3$ exposure on frequency of asthma symptoms in an asthmatic population of primarily lower-income, African American and Latino children (aged five-12 years) in East and Southwest Detroit, MI. Authors obtained health and demographic data through questionnaires filled out by parents or guardians for 14 consecutive days in each studied season. Questionnaires highlighted participant's asthma symptoms (cough, wheeze, shortness of breath, chest tightness), demographic information, medication use, and presence of second-hand smoke. The authors acquired maximum one-hour and maximum 8-hour $O_3$ concentrations and meteorological data from two community-level monitors placed on East and Southwest Detroit, MI school rooftops. Lewis et al. (2013) implemented a combination of generalized estimating equations and alternative logistic regression models to estimate the associations between short-term $O_3$ exposure and rate of asthma symptoms. Models adjusted for age, sex, location (Eastside or Southwest), race, household income, smoker in the home, season, and variables for companion home intervention study (control or intervention), time (pre- or post-intervention), and the interaction between intervention group status and time. Lewis et al. (2013) observed positive associations between short-term $O_3$ exposure and asthma symptoms.

### *Asthma Symptoms*

In single-pollutant models, the coefficient and standard error are based on the all year odds ratios of 1.12 (95% CI: 0.99-1.25) for cough, 1.13 (95% CI: 0.99-1.28) for wheeze, 1.20 (95% CI: 1.02-1.40) for chest tightness, and 1.07 (95% CI: 0.95-1.21) for shortness of breath, all for a 16 ppb (interquartile range) increase in 8-hour maximum $O_3$ concentrations (five-day average lag) (Lewis et al. 2013, Figure 1C).

## F.5.2 Tétreault et al. (2016)

Tétreault et al. (2016) investigated the effects of long-term $O_3$ exposure on asthma onset in children (aged zero-12 years) from Québec, Canada. The study followed participants from the Québec Integrated Chronic Disease Surveillance System open birth cohort between 1999 and 2011. The authors defined new cases of asthma based on hospital discharge reports and physician diagnoses (two diagnoses within a two-year span). Monitor data (Canadian National Air Pollution Surveillance network) and land-use mixed effect models estimated warm season (June to August) $O_3$ exposures. Authors assessed associations with asthma onset with both time of birth and time-varying exposure models and adjusted for year of birth, sex, and indices of social and material deprivation. Tétreault et al. (2016) used Cox proportional hazard models to observe associations between long-term $O_3$ exposure and asthma onset in children.

As the physiology and etiology of lung development in children is similar in children 6-17 (Baena-Cagnani et al., 2007, Guerra et al., 2004, Ochs et al., 2004, Sparrow et al., 1991, Trivedi and Denton, 2019), we apply the 4-12 year age-stratified effect estimate from Tétreault et al. (2016) to children ages 4-17.

### *Incidence, Asthma*

In a single-pollutant time-varying model, the coefficient and standard error were estimated from a warm-season hazard ratio of 1.07 (95% CI: 1.06-1.08) for a 3.26 ppb (interquartile range) increase in annual $O_3$ concentrations (Tétreault et al. 2016, Table 5).

# F.6 Sensitivity Analysis – General

Table F-6 summarizes the ozone health impact functions considered by EPA to be sensitivity analyses. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-29.  Core Health Impact Functions for Ozone Sensitivity Analyses**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| Mortality, All Cause | Di et al. | 2017 | Nationwide | 65-99 | $PM_{2.5}$ | Annual (D8HourMax) | 0.001094 | 0.000050 | Log-linear | All Cause, warm season |
| Incidence, Asthma | Garcia et al. | 2019 | 12 Southern California communities | 9-18 | | Annual (D8HourMax) | 0.016946 | 0.010941 | Log-linear | All year |
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | $PM_{10}$ | D1HourMax | 0.000985 | 0.000667 | Log-linear | Multi-pollutant, lag 1, warm season |
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | $PM_{10}$ | D8HourMax | 0.000985 | 0.000667 | Log-linear | Multi-pollutant, lag 1, warm season, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.001113. |
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | | D1HourMax | 0.000767 | 0.000304 | Log-linear | Single-pollutant, lag 1, warm season |
| Mortality, Respiratory | Katsouyanni et al. | 2009 | 90 U.S. Cities | 0-99 | | D8HourMax | 0.000767 | 0.000304 | Log-linear | Single-pollutant, lag-1, warm season, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.000867. |
| Mortality, All Cause | Turner et al. | 2016 | Nationwide | 30-99 | $PM_{2.5}$ | Annual (D8HourMax) | 0.001980 | 0.000500 | Log-linear | All Cause, warm season, multi-pollutant |
| Mortality, Respiratory | Turner et al. | 2016 | Nationwide | 30-99 | | Annual (D8HourMax) | 0.013103 | 0.001791 | Log-linear | Single-pollutant |
| Mortality, Respiratory | Turner et al. | 2016 | Nationwide | 30-99 | $PM_{2.5}$ | Annual (D8HourMax) | 0.011333 | 0.001823 | Log-linear | Multi-pollutant |

## F.6.1  Di et al. (2017)

Di et al. (2017) evaluated the relationship between long-term ozone exposure and all-cause mortality in nearly 61 million U.S. Medicare enrollees (over the age of 64) through 460 million person-years of follow-up and roughly 22 million observed deaths. This cohort comprised approximately 15% of the total U.S. population, included people living in rural areas, and is one of the largest cohort studies published to date. The authors modeled warm season ozone exposure across the contiguous U.S. using a hybrid methodology that included land use regression, satellite data, and monitor data, and resolved estimations to 1 x 1-kilometer areas. Di et al. (2017) used two-pollutant Cox proportional-hazards models with a generalized estimating equation. The authors controlled for demographic characteristics, Medicaid eligibility, and area-level covariates.

***All-Cause Mortality***

In a two-pollutant model, the coefficient and standard error for ozone are estimated from the hazard ratio (1.011) and 95% confidence interval of (1.010-1.012) associated with a change in annual mean ozone exposure of 10.0 ppb (Di et al., 2017, Table 2 Main Analysis).

## F.6.2  Garcia et al. (2019)

Garcia et al. (2019) examined the associations between long-term ozone exposure and asthma onset in children (aged nine-18 years) with no history of asthma in Southern California. The authors followed three waves of participants from the Children's Health Study for eight years between 1993 and 2014. Garcia et al. (2019) obtained health and demographic data from parents, guardians, or participants, who completed questionnaires annually. In order to calculate annual mean, community-level ozone exposure, the authors acquired daily eight-hour mean ozone concentrations through ambient air pollution monitors. Multi-level Poisson regression models with one-year lag showed no statistically significant associations between long-term ozone exposure and asthma onset in children. Models adjusted for demographic variables as well as factors pertaining to family medical history, environmental factors, and near-roadway pollution.

***Incidence, Asthma***

In a single-pollutant, all year model, the coefficient and standard error were estimated from an incidence rate ratio of 0.86 (95% CI: 0.71-1.04) for an 8.9 ppb decrease in eight-hour mean ozone concentrations (Garcia et al. 2019, Table 2). For consistency with the other HIFs, we convert this to a rate ratio for an increase in ozone, by taking the inverse of the reported incidence rate ratio, giving a rate ratio of 1.163 (95% CI: 0.962-1.408) for an 8.9 ppb increase in eight-hour mean ozone concentrations.

## F.6.3  Katsouyanni et al. (2009)

See full study descriptionunder Katsouyanni et al. (2009) in Appendix F, Section F.1.1.

***Short-term Mortality, Respiratory (Multi-Pollutant)***

In a multi-pollutant model, the coefficient and standard error are based on the summer-only penalized spline estimate of respiratory mortality of 0.99% (-0.33, 2.31%) per 10 ppb increase in $O_3$ lagged by 1 day (Katsouyanni et al. 2009, Table 24: Lag 1; Penalized splines; Controlling for $PM_{10}$).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

***Short-term Mortality, Respiratory (Single-Pollutant)***

In a single-pollutant model, the coefficient and standard error are based on the summer-only penalized spline estimate of respiratory mortality of 0.77% (0.17, 1.37%) per 10 ppb increase in $O_3$ lagged by 1 day (Katsouyanni et al. 2009, Table 24: Lag 1; Penalized splines; $O_3$ Results).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

## F.6.4 Turner et al. (2016)

See full study description under Turner et al. (2016) in Appendix F, Section F.1.2.

### *Long-term Mortality, All Cause*

In a multi-pollutant model, the coefficient and standard error are based on the warm-season specific hazard ratio of 1.02 (1.01-1.03) per 10 ppb increase in seasonal average of daily 8-hour maximum $O_3$ concentrations (Turner et al. 2016, Table E9: HBM $O_3$, multipollutant model data, fully adjusted).

### *Long-term Mortality, Respiratory (Single-Pollutant)*

In a single-pollutant model, the coefficient and standard error are based on the warm-season specific hazard ratio of 1.14 (1.10-1.18) per 10 ppb increase in seasonal average of daily 8-hour maximum $O_3$ concentrations (Turner et al. 2016, Table E5: HBM $O_3$, 1982-2004, fully adjusted plus ecological covariate).

### *Long-term Mortality, Respiratory (Multi-Pollutant)*

In a multi-pollutant model, the coefficient and standard error are based on the warm-season specific hazard ratio of 1.12 (1.08-1.16) per 10 ppb increase in seasonal average of daily 8-hour maximum $O_3$ concentrations (Turner et al. 2016, Table E7: HBM $O_3$, 1982-2004, multipollutant, fully adjusted).

# F.7  Sensitivity Analysis − At‑Risk Populations

Table F-7 summarizes the ozone health impact functions considered by EPA to be sensitivity analyses that characterize risk experienced by certain subpopulations. Below, we present a brief summary of each of the studies and any items that are unique to the study.

**Table F-30.   Core Health Impact Functions for Ozone Sensitivity Analyses of At-Risk Populations**

| Effect | Author | Year | Location | Age | Co-Poll | Metric | Beta | Std Err | Form | Notes |
|---|---|---|---|---|---|---|---|---|---|---|
| HA, All Respiratory | Cakmak et al. | 2006 | 10 Canadian Cities | 0-99 | | D24HourMean | 0.002033 | 0.000580 | Logistic | Female |
| HA, All Respiratory | Cakmak et al. | 2006 | 10 Canadian Cities | 0-99 | | D8HourMax | 0.002033 | 0.000580 | Logistic | Female, 8-hour max from 24-hour mean using adjustment factor of 0.654, resulting in effective beta of 0.001328. |
| HA, All Respiratory | Cakmak et al. | 2006 | 10 Canadian Cities | 0-99 | | D24HourMean | 0.002530 | 0.000519 | Logistic | Male |
| HA, All Respiratory | Cakmak et al. | 2006 | 10 Canadian Cities | 0-99 | | D8HourMax | 0.002530 | 0.000519 | Logistic | Male, 8-hour max from 24-hour mean using adjustment factor of 0.654, resulting in effective beta of 0.001653. |
| Mortality, Respiratory | Jerrett et al. | 2009 | Nationwide US, 96 MSAs | 30-99 | | Annual (D1HourMax) | 0.003922 | 0.000972 | Log-linear | Female |
| Mortality, Respiratory | Jerrett et al. | 2009 | Nationwide US, 96 MSAs | 30-99 | | Annual (D8HourMax) | 0.003922 | 0.000972 | Log-linear | Female, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.004432. |
| Mortality, Respiratory | Jerrett et al. | 2009 | Nationwide US, 96 MSAs | 30-99 | | Annual (D1HourMax) | 0.000995 | 0.001257 | Log-linear | Male |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Mortality, Respiratory | Jerrett et al. | 2009 | Nationwide US, 96 MSAs | 30-99 | | Annual (D8HourMax) | 0.000995 | 0.001257 | Log-linear | Male, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.001124. |
| Mortality, All Cause | Katsouyanni et al. | 2009 | Nationwide US, 90 cities | 0-74 | | D1HourMax | 0.000698 | 0.000213 | Logistic | Age <75 |
| Mortality, All Cause | Katsouyanni et al. | 2009 | Nationwide US, 90 cities | 0-74 | | D8HourMax | 0.000698 | 0.000213 | Logistic | Age <75, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.000788. |
| Mortality, All Cause | Katsouyanni et al. | 2009 | Nationwide US, 90 cities | 75-99 | | D1HourMax | 0.000618 | 0.000233 | Logistic | Age ≥75 |
| Mortality, All Cause | Katsouyanni et al. | 2009 | Nationwide US, 90 cities | 75-99 | | D8HourMax | 0.000618 | 0.000233 | Logistic | Age ≥75, 8-hour max from 1-hour max using adjustment factor of 1.13, resulting in effective beta of 0.000698. |
| HA, Lower Respiratory Infection | Lin et al. | 2005 | Toronto, Canada | 0-14 | | D1HourMax | 0.007592 | 0.005232 | Logistic | Female |
| HA, Lower Respiratory Infection | Lin et al. | 2005 | Toronto, Canada | 0-14 | | D8HourMax | 0.007592 | 0.005232 | Logistic | Female, 8-hour max from 1-hour max using adjustment factor of 1.14, resulting in effective beta of 0.008655. |
| HA, Lower Respiratory Infection | Lin et al. | 2005 | Toronto, Canada | 0-14 | | D1HourMax | 0.003530 | 0.004524 | Logistic | Male |
| HA, Lower Respiratory Infection | Lin et al. | 2005 | Toronto, Canada | 0-14 | | D8HourMax | 0.003530 | 0.004524 | Logistic | Male, 8-hour max from 1-hour max using adjustment factor of 1.14, resulting in effective beta of 0.004025. |
| Emergency Room | Mar and Koenig | 2009 | Seattle, Washington | 0-17 | | D8HourMax | 0.010436 | 0.004358 | Log-linear | Age <18 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Visits, Asthma | | | | | | | | | | |
| Emergency Room Visits, Asthma | Mar and Koenig | 2009 | Seattle, Washington | 18-99 | | D8HourMax | 0.003922 | 0.002688 | Log-linear | Age ≥18 |
| Mortality, All Cause | Medina-Ramon & Schwartz | 2008 | Nationwide US, 48 cities | 0-64 | | D8HourMean | -0.000130 | 0.000102 | Logistic | Age <65 |
| Mortality, All Cause | Medina-Ramon & Schwartz | 2008 | Nationwide US, 48 cities | 65-99 | | D8HourMean | 0.000965 | 0.000235 | Logistic | Age ≥65 |
| Mortality, All Cause | Medina-Ramon & Schwartz | 2008 | Nationwide US, 48 cities | 0-99 | | D8HourMean | 0.000936 | 0.00024 | Logistic | Female, Age 0-99 |
| Mortality, All Cause | Medina-Ramon & Schwartz | 2008 | Nationwide US, 48 cities | 0-99 | | D8HourMean | 0.000359 | 0.000038 | Logistic | Male, Age 0-99 |
| Emergency Room Visits, Asthma | Paulu and Smith | 2008 | Maine | 2-14 | | D8HourMax | 0.010436 | 0.005027 | Logistic | Age 2-14 |
| Emergency Room Visits, Asthma | Paulu and Smith | 2008 | Maine | 15-34 | | D8HourMax | 0.014842 | 0.003524 | Logistic | Age 15-34 |
| Emergency Room Visits, Asthma | Paulu and Smith | 2008 | Maine | 2-99 | | D8HourMax | 0.011333 | 0.002736 | Logistic | Female, Age 2-99 |
| Emergency Room Visits, Asthma | Paulu and Smith | 2008 | Maine | 2-99 | | D8HourMax | 0.010436 | 0.003222 | Logistic | Male, Age 2-99 |
| Emergency Room Visits, Asthma | Villeneuve et al. | 2007 | Edmonton, Canada | 2-4 | | D8HourMax | 0.003237 | 0.003342 | Logistic | Age 2-4 |
| Emergency Room Visits, Asthma | Villeneuve et al. | 2007 | Edmonton, Canada | 5-14 | | D8HourMax | 0.007279 | 0.002357 | Logistic | Age 5-14 |
| Emergency Room Visits, Asthma | Villeneuve et al. | 2007 | Edmonton, Canada | 15-14 | | D8HourMax | 0.005798 | 0.00179 | Logistic | Age 15-14 |
| Emergency Room Visits, Asthma | Villeneuve et al. | 2007 | Edmonton, Canada | 45-64 | | D8HourMax | 0.006296 | 0.003275 | Logistic | Age 45-64 |
| Emergency Room Visits, Asthma | Villeneuve et al. | 2007 | Edmonton, Canada | 65-74 | | D8HourMax | 0.007279 | 0.005544 | Logistic | Age 65-74 |
| Emergency Room | Villeneuve et al. | 2007 | Edmonton, Canada | 75-99 | | D8HourMax | -0.000558 | 0.006684 | Logistic | Age 75-99 |

| Visits, Asthma | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 0-20 | | D8HourMean | 0.00008 | 0.000252 | Logistic | Age 0-20 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 21-30 | | D8HourMean | 0.0001 | 0.000392 | Logistic | Age 21-30 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 31-40 | | D8HourMean | 0.00007 | 0.000229 | Logistic | Age 31-40 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 41-50 | | D8HourMean | 0.00008 | 0.000178 | Logistic | Age 41-50 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 51-60 | | D8HourMean | 0.000539 | 0.000178 | Logistic | Age 51-60 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 61-70 | | D8HourMean | 0.000379 | 0.000114 | Logistic | Age 61-70 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 71-80 | | D8HourMean | 0.000499 | 0.000089 | Logistic | Age 71-80 |
| Mortality, All Cause | Zanobetti and Schwartz | 2008 | Nationwide US, 48 cities | 81-99 | | D8HourMean | 0.00029 | 0.000079 | Logistic | Age 81-99 |

## F.7.1 Cakmak et al. (2006)

Cakmak et al. (2006) examined the relationship between daily average $O_3$ concentrations and hospital admissions for respiratory causes (ICD-9 466, 480-486, 490, 491, 492, 493, 494, and 496) among residents of 10 Canadian cities (Calgary, Edmonton, Halifax, London, Ottawa, Saint John, Toronto, Vancouver, Windsor, and Winnipeg). Data on 215,544 respiratory hospitalizations were obtained from the Canadian Institute for Health Information. Daily 24-hour average $O_3$ concentrations in all seasons were estimated using the average of data from all available monitors within each city. The authors ran city-specific multi-pollutant Poisson regression models by sex, education level, and income quartile using time lags of 0 to 5 days. Models controlled for day of the week, temperature, humidity, and barometric pressure. Pooled estimates across all 10 cities were calculated by using a random-effects model.

***Hospital Admissions, All Respiratory***

In single-pollutant models, the coefficient and standard error are estimated from a percentage increase of 3.6% (95% CI: 1.6-5.7%) for females and 4.5% (95% CI: 2.6-6.3%) for males for a 17.4 ppb increase in daily 24-hour average $O_3$ concentrations (Cakmak et al. 2006, Table 3).

The Health Impact Function was adjusted from the daily 24-hour mean metric to the daily 8-hour max metric using a ratio of 1/1.53 = 0.65359 (inverse of the ratio of 8-hour max to 24-hour mean ozone) (Anderson and Bell 2010, Table 2).

## F.7.2 Jerrett et al. (2009)

Jerrett et al. (2009) examined the potential contribution of long-term ozone exposure to the risk of death from cardiopulmonary causes and specifically to death from respiratory causes. Data from the study cohort of the American Cancer Society Cancer Prevention Study II were correlated with air-pollution data from 96 metropolitan statistical areas in the United States. Associations between warm season ozone concentrations and the risk of death were evaluated with the use of standard and multilevel Cox regression models. In single-pollutant models, increased concentrations of either $PM_{2.5}$ or ozone were significantly associated with an increased risk of death from cardiopulmonary causes. In two-pollutant models, $PM_{2.5}$ was associated with the risk of death from cardiovascular causes, whereas ozone was associated with the risk of death from respiratory causes. The estimated relative risk of death from respiratory causes that was associated with an increment in ozone concentration of 10 ppb was 1.040 (95% confidence interval, 1.010 to 1.067). The association of ozone with the risk of death from respiratory causes was insensitive to adjustment for confounders and to the type of statistical model used. The authors concluded that they were not able to detect an effect of ozone on the risk of death from cardiovascular causes when the concentration of $PM_{2.5}$ was taken into account. But they did demonstrate a significant increase in the risk of death from respiratory causes in association with an increase in ozone concentration.

***Mortality, Respiratory (ICD-9 code 460-519)***

In single-pollutant models, the coefficient and standard error are estimated from a relative risk of 1.04 (95% CI: 1.03-1.07) for females and 1.01 (95% CI: 0.99-1.04) for males for a 10 ppb change in ambient ozone concentration measured from April to September during the years from 1977 to 2000 (Jerrett et al. 2009, Table 4).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

## F.7.3 Katsouyanni et al. (2009)

See full study description under Katsouyanni et al. (2009) in Appendix F, Section F.1.1.

### *Short-term Mortality, All Cause*

In single pollutant models, the coefficient and standard error are based on the summer-only penalized spline estimate of all-cause mortality of 0.70% (0.28, 1.12%) for ages <75 and 0.62% (0.16, 1.08%) for ages ≥75 per 10 ppb increase in $O_3$ from distributed lag days (Katsouyanni et al. 2009, Table 24: Distributed Lags; Penalized splines; $O_3$ Results).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

## F.7.4 Lin et al. (2005)

Lin et al. (2005) examined the relationship between short term $O_3$ exposures and hospital admissions for lower respiratory infections (ICD-9 464, 466, 480-487) in a case-crossover study among Toronto residents under the age of 15 between 1998 and 2001. Data on 6,782 hospitalizations were obtained from the Discharge Abstract Database. Daily 1-hour maximum $O_3$ concentrations in all seasons were obtained from seven monitoring stations in the National Air Pollution Surveillance system. The authors ran conditional logistic regression models controlling for temperature and weather conditions using 1- to 7-day average lags.

### *Hospital Admissions, All Respiratory*

In multi-pollutant models adjusted for $PM_{2.5}$ and $PM_{10-2.5}$, the coefficient and standard error are estimated from an odds ratio of 1.18 (95% CI: 0.94-1.47) for females and 1.08 (95% CI: 0.89-1.31) for males for a 21.8 ppb increase in daily 1-hour maximum $O_3$ concentrations with a 4-day average lag (Lin et al. 2005, Table 3 (Adjusted B)).

The Health Impact Function was adjusted from the daily 1-hour max metric to the daily 8-hour max metric using a ratio of 1.13 (ratio of 1-hour max to 8-hour max ozone) (Anderson and Bell 2010, Table 2).

## F.7.5 Mar and Koenig (2009)

Mar and Koenig (2009) evaluated the relationship between outdoor ozone in the summer and asthma aggravation. The authors used hospital data on daily asthma cases from 1998 to 2002 in Seattle with local monitored $PM_{2.5}$ and ozone concentrations to assess the association between asthma visits to the emergency department and air pollutants. They analyzed 1-hour and 8-hour max ozone concentrations at two monitors in Greater Seattle. Asthma ED visits were analyzed at 0 through 5-day lags. The authors found that ozone exposure exacerbates asthma in people in the Seattle area, especially in children. Authors found that in adults during the warmer months between May and September, a 10 ppb increase in 8-hour maximum ozone concentration is associated with relative risk of asthma-related ED visits of 1.08 (1.02, 1.14) with a 4-day lag. In children, during the warmer months, a 10 ppb increase in 8-hour maximum ozone concentration is associated with relative risk of asthma-related ED visits of 1.11 (1.02, 1.21) with a 3-day lag. The difference in lag and relative risk between children and adults suggests that children are more immediately responsive to the adverse effects of ozone exposure.

***Emergency Room Visits, Asthma***

In single-pollutant models, the coefficient and standard error are estimated from a relative risk of 1.11 (95% CI: 1.02-1.21) for age <18 and 1.04 (95% CI: 0.99-1.10) for age ≥18 for a 10 ppb increase in daily 8-hour maximum summer ozone concentration with a 3-day average lag (Mar and Koenig, 2009, Table 5 and Table 6).

## F.7.6  Medina-Ramon & Schwartz (2008)

Medina-Ramon & Schwartz (2008) evaluated short-term $O_3$ exposure and all-cause mortality among residents of all ages in 48 U.S. cities from 1989-2000 using a case-only approach. Data on 2,729,640 non-accidental deaths was obtained from the National Center for Health Statistics. The authors estimated 8-hour daily mean ozone concentrations (warm season, May to September) for each city using daily ozone levels reported by the U.S. EPA Aerometric Retrieval System. The authors ran city-specific conditional logistic regressions controlling for seasonality, temperature, and day of the week and pooled the results across cities. Results were presented by socio-demographic characteristics and chronic conditions.

***Mortality, All Cause***

The $O_3$-mortality risk estimates for at-risk subpopulations reported in Medina-Ramon & Schwartz (2008) required additional modification in order to use those results to develop health impact functions.  The authors presented excess risk estimates for each subpopulation as the additional percent change in mortality for persons who have the condition, compared to persons without the condition. For our populations of interest, these subgroups were persons age 65 or older compared to those younger than 65, and females relative to males. However, they did not report the risk estimate for these comparison groups, so in order to estimate the total excess risk for each exposed at-risk group, we needed to first back-calculate the excess risk for the comparison group without the factor of interest. We accomplished this by assuming that the authors' overall reported excess risk for the full sample of 0.65% (95% confidence interval = 0.38% to 0.93%) could be expressed as a weighted average of the unreported excess risk ("x") and the full excess risk for the at-risk group, which would be expressed as the sum of x and the reported excess risk from Medina-Ramon & Schwartz (2008) Table 2, where the weights are calculated using the total and at-risk group sample sizes in Table 1 of that paper. For example, to calculate the total excess risks for the females in the sample, we used the following equation:

**Equation F-5**

$$ER_{Total} = \frac{ER_{Male}(Pop_{Male}) + ER_{Female}(Pop_{Female})}{Pop_{Total}}$$

where $ER_{Total}$ is the full sample excess risk of 0.65%; $ER_{Female}$ is the excess risk of ozone exposures for females; $ER_{Male}$ is the excess risk of ozone exposures for males; $Pop_{Total}$ is the total sample population; and $Pop_{Female}$ and $Pop_{Male}$ are the size of the female and male subsets of the sample population, respectively. We also know from Table 2 of that paper that:

**Equation F-6**

$$ER_{Female} = ER_{Male} + 0.58\%$$

Substituting and using the available information from Medina-Ramon & Schwartz (2008) Tables 1 and 2, we can solve for $ER_{Male}$ and then $ER_{Female}$:

**Equation F-7**

$$0.65\% = \frac{ER_{Male}(1,365,937) + (0.58\% + ER_{Male})(1,363,703)}{2,729,640}$$

$$ER_{Male} = 0.36\%$$

and

$$ER_{Female} = 0.36\% + 0.58\% = 0.94\%$$

We then used the full excess risk value for the female subpopulation to derive a health impact function for ozone-related mortality for females. Final calculated excess risks are 0.94% (0.47-1.42%) for females aged 0-99; 0.36% (0.29-0.44%) for males aged 0-99, -0.13% (-0.33-0.07%) for both sexes aged 0-64, and 0.97% (0.51-1.44%) for both sexes aged 65-99.

## F.7.7 Paulu and Smith (2008)

Paulu and Smith (2008) conducted a case-crossover analysis to evaluate the relationship between daily ozone concentrations and emergency room visits for asthma (ICD-9 493) among Maine residents aged 2 and older from 2000 to 2003. Data on 8,020 asthma-related ER visits was obtained from the Maine Health Data Organization. Daily 8-hour maximum $O_3$ concentrations were computed from two monitor sites situated in or near Portland, ME and data was obtained from the Maine Department of Environmental Protection, Bureau of Air Quality. The authors defined the warm season as May 22-September 10 (2000) and May 23-September 11 (2001-2003). The authors ran conditional logistic regression models stratified by sex and age groups (2–14, 15–34, 35–64, and ≥65 years) controlling for temperature, humidity, and day after major holidays as well as $PM_{2.5}$ in co-pollutant models. Paulu and Smith (2008) found that excess risk was concentrated among females aged 15 to 34 and males younger than 15.

***Emergency Room Visits, Asthma***

In single-pollutant models for both sexes, the coefficient and standard error are estimated from an excess risk of 11% (1-23%) for ages 2-14 and 16% (8-24%) for ages 15-34 for a 10 ppb increase in average daily 8-hour maximum ozone (lags 0-3 days) (Paulu and Smith, 2008, Figure 1 text).

In single-pollutant models for ages 2 and above, the coefficient and standard error are estimated from an excess risk of 12% (6-18%) for females and 11% (4-18%) for males for a 10 ppb increase in average daily 8-hour maximum ozone (lags 0-3 days) (Paulu and Smith, 2008, Figure 1 text).

## F.7.8  Villeneuve et al. (2007)

Villeneuve et al. (2007) evaluated the relationship between short-term ozone exposure and emergency room visits for asthma (ICD-9 493) among residents of Edmonton, Canada aged 2 and above from 1992 to 2002 in a case-crossover study. Data on 57,912 asthma-related ER visits was provided by Capital Health. Daily 8-hour maximum $O_3$ concentrations were obtained from automated fixed-site monitoring stations maintained by Environment Canada as part of the National Air Pollution Surveillance Network. The monitors measured both warm season (April-September) and cold season (October-March) ozone concentrations. The authors ran conditional logistic regression models controlling for temperature, humidity, influenza, and aeroallergens. Villeneuve et al. (2007) found associations between ozone and asthma emergency room visits in the warm season and observed the strongest effects in young children.

***Emergency Room Visits, Asthma***

In single-pollutant models, the coefficient and standard error are estimated from odds ratios of 1.06 (0.94-1.19) for ages 2-4; 1.14 (1.05-1.24) for ages 5-14; 1.11 (1.04-1.18) for ages 15-44; 1.12 (1.00-1.26) for ages 45-64; 1.14 (0.94-1.39) for ages 65-74; and 0.99 (0.78-1.25) for ages 75-99 for a 18 ppb increase in average daily 8-hour maximum warm season ozone (5-day average lag) (Villeneuve et al., 2007, Tables 4, 5, 6, 7, 8, 9).

## F.7.9  Zanobetti and Schwartz (2008b)

Zanobetti and Schwartz (2008b) evaluated the relationship between short-term ozone exposure and all-cause mortality across 48 U.S. cities (1989-2000) for all ages in a case-crossover study. Data on 6,951,395 total deaths was provided by the National Center for Health Statistics. Daily 8-hour mean $O_3$ concentrations were obtained from the U.S. EPA Air Quality System Technology Transfer Network for all seasons. The authors ran conditional logistic regression models by season, month, and age group (0-20, 21-30, 31-40, 41-50, 51-60, 61-70, 71-80, 80+) controlling for temperature, dew point, and day of the week.

### *Mortality, All Cause*

In single-pollutant models, the coefficient and standard error are estimated from an excess risk of 0.08% (-0.42-0.57%) for ages 0-20; 0.1% (-0.67-0.87%) for ages 21-30; 0.07% (-0.38-0.52%) for ages 31-40; 0.08% (-0.27-0.43%) for ages 41-50; 0.54% (0.19-0.89%) for ages 51-60; 0.38% (0.16-0.61%) for ages 61-70; 0.5% (0.32-0.67%) for ages 71-80; and 0.29% (0.13-0.44%) for ages 81-99 for a 10 ppb increase in average daily 8-hour mean all season ozone (Zanobetti and Schwartz 2008b, Table 2).

# Appendix G.   Additional Health Impact Functions in BenMAP

In this Appendix, we present additional health impact functions for estimating $PM_{2.5}$ and Ozone-related adverse health effects. Unlike Appendices E and F, these functions are not currently used by the U.S. EPA in regulatory impact analyses.  **PLACEHOLDER: Information on additional functions will be included here as they are added to the tool by EPA.**

# Appendix H.   Core Health Valuation Functions in BenMAP

This appendix presents the core unit values that are available in BenMAP for each of the health effects included in the current suite of health impact functions. Specifically, this appendix includes the values currently used by U.S. EPA in regulatory impact analyses. For the .apvx files summarizing current EPA practices, see:

https://www.epa.gov/benmap/benmap-community-edition

Wherever possible, we present a distribution of the unit value, characterizing the uncertainty surrounding any point estimate. The mean of the distribution is taken as the point estimate of the unit value, and the distribution itself is used to characterize the uncertainty surrounding the unit value, which feeds into the uncertainty surrounding the monetary benefits associated with reducing the incidence of the health effect. Below we give detailed descriptions of the derivations of unit values and their distributions, as well as tables listing the unit values and their distributions, available for each health effect. The definitions of the distributions and their parameters are given in Table H-1.

**Table H-31.    Unit Value Uncertainty Distributions and Their Parameters**

| Distribution* | Parameter 1 (P1) | Parameter 2 (P2) |
|---|---|---|
| Normal | Standard deviation | – |
| Triangular | Minimum value | Maximum value |
| Lognormal ** | Mean of corresponding normal distribution | Standard deviation of corresponding normal distribution |
| Uniform | Minimum value | Maximum value |
| Weibull *** | $\alpha$ | $\beta$ |

*In all cases, BenMAP calculates the mean of the distribution, which is used as the "point estimate" of the unit value.

** If Y is a normal random variable, and Y = logeX, then X is lognormally distributed. Equivalently, X is lognormally distributed if X = eY, where Y is normally distributed.

*** The Weibull distribution has the following probability density function:

**Equation H-1**

$$\left(\frac{\beta}{\alpha}\right)\left(\frac{x}{\alpha}\right)^{\beta-1} e^{-(x/\alpha)^{\beta}}$$

This appendix also presents EPA methods for developing income growth adjustment factors that allow you to adjust the WTP estimates to account for the growth in income over time.

# H.1  Mortality

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economics and public policy analysis communities. Issues such as the appropriate discount rate and whether there are factors, such as age or the quality of life, that should be taken into consideration when estimating the value of avoided premature mortality are still under discussion. BenMAP currently offers a variety of options reflecting the uncertainty surrounding the unit value for premature mortality.

## H.1.1 Value of a Statistical Life Based on 26 Studies

The current undiscounted VSL used by EPA is $8.7 million (2015$). This estimate is the mean of a distribution fitted to 26 "value of statistical life" (VSL) estimates that appear in the economics literature and that 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. Accounting for the cessation lag, or the delay between pollutant exposure and death, this VSL equates to $7.8 million (2015$) using a 3% discount rate and $7.1 million (2015$) using a 7% discount rate (U.S. EPA, 2014).

The VSL approach mirrors that of Viscusi (1992), and uses the same criteria as Viscusi in his review of value-of-life studies. The $8.7 million estimate is consistent with Viscusi's conclusion (updated to 2015$) that "most of the reasonable estimates of the value of life are clustered in the $5.2 to $12.3 million range." Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. Because this VSL-based unit value does not distinguish among people based on the age at their death or the quality of their lives, it can be applied to all premature deaths. Table H-2 presents the unit values for the 26 value-of-life studies, the 3% and 7% discounted unit values represent the core EPA values for this effect while the undiscounted rate represents an additional valuation function.

**Table H-32.    Core Unit Values for VSL based on 26-value-of-life studies**

| Basis for Estimate * | Age Range at Death | | Unit Value (VSL) (2015$) | Distribution of Unit Value | Parameters of Distribution | |
|---|---|---|---|---|---|---|
| | Min | Max | | | P1 | P2 |
| VSL, based on 26 value-of-life studies | 0 | 99 | 8,705,114 | Weibull | 9,648,168 | 1.509588 |
| VSL, based on 26 value-of-life studies, 3% discount rate | 0 | 99 | 7,887,356 | Weibull | 8,741,819 | 1.367777 |
| VSL, based on 26 value-of-life studies, 7% discount rate | 0 | 99 | 7,103,778 | Weibull | 7,873,354 | 1.231894 |

\* The original value of a statistical life was calculated in 1990$.  We have used a factor of 1.8134, based on the All-Items CPI-U.

# H.2  Hospital Admissions & Emergency Room Visits

This section presents the core values for avoided hospital admissions, as well as avoided emergency room visits. We assume that hospital admissions due to acute exposure to air pollution pass through the emergency room. However, the value of hospital admissions that we have calculated here does not account for the cost incurred in the emergency room visit.

## H.2.1 Hospital Admissions

As suggested above, the total value to society of an individual's avoidance of a hospital admission can 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 available for use in BenMAP 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 downward bias with an upward bias. Based on Science Advisory Board (SAB) advice, the COI values currently available for use in BenMAP are not adjusted.

Unit values are based on ICD-code-specific estimated hospital charges and 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.

For all hospital admissions effects available in BenMAP, 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 (2016). 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 2016 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 were used to define the health effects.

Since most pollution-related hospital admissions are likely unscheduled, the unit values of avoided hospital admissions used in BenMAP 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.

County-specific median annual income divided by (52*5) was used to estimate county-specific median daily wage. The data source for median annual income is the 2015 American Community Survey (ACS). ACS provided data for median annual income for all individuals over 16 years old in 819 counties. For all other counties, ACS provided a five-year estimate of median annual income for the years 2010-2014. We calculated the ratio of state-specific median annual income in 2015 to state-specific median annual income during this five-year interval (2010-2014). This ratio was then applied to the 2010-2014 county-specific median annual income to obtain an estimate of 2015 county-specific income for the 2,323 counties without 2015 one-year estimates from ACS. Because wage data used in BenMAP are county-specific, the unit value for a hospital admission varies from one county to another.

Although the data for hospital charges are from year 2016, the default hospital admission unit values in BenMAP are in year 2015 dollars to be consistent with the unit values of other health effects in BenMAP. This was done by inflating the medical costs (2016 dollars) to 2015 dollars using BenMAP's medical inflation index.

The hospital admission outcomes that the EPA uses in its regulatory analyses are given in Table H-3. Although unit values available for use in BenMAP are county-specific, the national median daily wage was used to calculate opportunity costs and total costs.

**Table H-33.  Core Unit Values Available for Hospital Admissions**

| Health Effect | ICD Codes | Age Range Min | Age Range Max | Mean Hospital Charge (2015 $) | Mean Length of Stay (days) | Total Cost of Illness (Unit Value in 2015$)* |
|---|---|---|---|---|---|---|
| HA, All Cardiac Outcomes | 390-459 | 0 | 99 | $16,045 | 5.05 | $16,918 |
| HA, All Respiratory | 460-519 | 0 | 18 | $9,075 | 3.49 | $9,678 |
| HA, All Respiratory | 460-519 | 65 | 99 | $35,402 | 6.07 | $36,451 |
| HA, Alzheimer's Disease | 331.0 | 65 | 99 | $10,696 | 7.95 | $12,070 |
| HA, Cardio-, Cerebro- and Peripheral Vascular Disease | 410- 414, 429, 426- 427, 428, 430-438, 440-449 | 65 | 99 | $14,665 | 4.82 | $15,498 |
| HA, Respiratory-1 | 491, 492, 493, 496 | 0 | 99 | $7,676 | 3.86 | $8,343 |
| HA, Respiratory-2 | 464-466, 480-487, 490-492, 493 | 65 | 99 | $9,003 | 4.66 | $9,808 |
| HA, Parkinson's Disease | 332 | 18 | 99 | $12,190 | 3.83 | $12,852 |

* The opportunity cost of a day spent in the hospital was estimated, for the above exhibit, at the median daily wage of all workers, regardless of age. The median daily wage was calculated by dividing the median weekly wage ($864 in 2015$) by 5. The median weekly wages for 2015 were obtained from the U.S. Census Bureau's 2015 American Community Survey, "Selected Economic Characteristics: 2015 American Community Survey 1-Year Estimates."

For two hospital admission effects, Alzheimer's and Parkinson's Disease, we calculated the lifetime COI in addition to calculating the costs associated with the initial hospitalization.

Valuation sources of Alzheimer's disease lifetime medical costs were available from the Alzheimer's Association (2020) report and Jutkowitz et al. (2017). Using Alzheimer's Association (2020), we first developed an estimate of incremental annual medical expenses for Medicare beneficiaries living with Alzheimer's Disease. Then, using the estimated life expectancy duration of 5 years from Jutkowitz et al. (2017), we estimated total discounted present values for a five-year stream of costs using 3 and 7 percent discount rates (Table H-4). We note that the median age of Alzheimer's disease onset is after the age of 65. As such, we exclude any potential lost wages given the low rate of labor force participation in this age group. Lifetime medical costs, excluding initial hospitalization, are estimated at $156,920 using a 3% discount rate or $145,946 using a 7% discount rate in 2015.

**Table H-34.   Alzheimer's Disease Valuation (2015$)**

| Year | 3% Discount Rate | 7% Discount Rate |
|---|---|---|
| 0 | $33,266 | $33,266 |
| 1 | $32,297 | $31,090 |
| 2 | $31,357 | $29,056 |
| 3 | $30,443 | $27,155 |
| 4 | $29,557 | $25,379 |
| Total Lifetime Costs | $156,920 | $145,946 |

Estimates of lifetime costs for Parkinson's Disease were provided by Yang et al. (2020), including direct, indirect, and non-medical costs. Using Yang et al. (2020), we first developed an annual estimate of excess costs associated with living with Parkinson's Disease. Then, using the estimated life expectancy duration of 14.6 years from De Pablo-Fernandez et al., 2017, we calculated the present value of lifetime costs over this period using 3 and 7 percent discount rates (Table H-5). Lifetime medical costs are estimated at $567,285 using a 3% discount rate or $445,792 using a 7% discount rate in 2015$.

**Table H-35.   Lifetime Parkinson's Disease Valuation Estimate Calculation**

| Year | 3% Discount Rate | 7% Discount Rate |
|------|------------------|------------------|
| 0 | $44,718 | $44,718 |
| 1 | $43,416 | $41,793 |
| 2 | $42,151 | $39,059 |
| 3 | $40,924 | $36,503 |
| 4 | $39,732 | $34,115 |
| 5 | $38,574 | $31,883 |
| 6 | $37,451 | $29,798 |
| 7 | $36,360 | $27,848 |
| 8 | $35,301 | $26,026 |
| 9 | $34,273 | $24,324 |
| 10 | $33,275 | $22,732 |
| 11 | $32,305 | $21,245 |
| 12 | $31,364 | $19,855 |
| 13 | $30,451 | $18,556 |
| 14 | $29,564 | $17,343 |
| 14.6 | $17,427 | $9,992 |
| Total Lifetime Costs (14.6 yr survival) | $567,285 | $445,792 |

## H.2.2 Emergency Room Visits

As with hospital admissions, to value emergency room visits we develop primary COI estimates using data from the Healthcare Cost and Utilization Project (HCUP). The 2016 Nationwide Emergency Department Sample (NEDS) provides recent, nationally representative information on medical treatment in emergency departments. In the case of emergency department visits, valuation estimates include only the medical costs.

The NEDS dataset includes discharge-level observations. That is, each data point represents one individual being discharged from the emergency department (NEDS). Because

individuals are treated in these settings for a variety of reasons, we use medical billing codes to extract observations related to each health effect. The epidemiological studies described in Appendix E, F and G provide ICD-9 codes for each illness; however, recent HCUP datasets (including NEDS) use ICD-10 codes. Thus, we first crosswalk the relevant ICD-9 codes to associated ICD-10 codes using a mapping provided by the U.S. Centers for Disease Control. We then identify all discharges in the HCUP datasets with ICD-10 codes that match to a study's ICD-9 code(s). Because HCUP datasets often include multiple ICD-10 codes for each discharge, we focus on the principal diagnosis (i.e., the first-listed ICD-10 code).

In the NIS dataset, we convert total charges (i.e., the amount billed to patients, employers, or insurance providers) into estimates of total costs (i.e., the final reimbursements for medical treatment). Unadjusted charges are not suitable for use in regulatory analysis because posted prices generally do not reflect actual medical costs due, in part, to negotiation between medical providers and payers (e.g., insurance companies). We assume that adjusted charges reflect the actual revenue the hospital receives and thus the actual cost of providing care. This conversion is completed using hospital-specific cost-to-charge (CCR) ratios provided with NIS. Because CCRs are not available for NEDS, we apply average CCRs for each health effect in NIS to the same set of ICD-10 codes in NEDS.

For each health effect, mean estimates are calculated using estimation commands for survey data to account for the sampling design and sample discharge weights of the HCUP data. This results in estimates of mean costs and a 95% confidence interval, which represents uncertainty in our valuation estimates of medical costs. The resulting estimates are presented in Table H-6.

**Table H-36.   Core Unit Values Available for Emergency Department Visits**

| Health Effect | ICD Codes | Age Range | | Mean Unit Value (2015 $) |
|---|---|---|---|---|
| | | Min | Max | |
| ER visits, All Cardiac Outcomes | 390-459 | 0 | 99 | $1,161 |
| ER visits, respiratory | 491-493, 460-466, 477, 480-486, 496, 786.07, 786.09 | 0 | 99 | $875 |

## H.2.3 Emergency Room Visits for Asthma

Two unit values are currently available for use in BenMAP for asthma emergency room (ER) visits. One is $533.69, 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 $533.69 in 2015$ (using the CPI for medical care to adjust to 2015$). The uncertainty surrounding this estimate, based on the uncertainty surrounding the number of ER visits and the total cost of all visits reported by Smith et al. is characterized by a triangular distribution centered at $533.69, on the interval [$395.14, $738.19].

A second unit value is $446.52 from Stanford et al. (1999). This study considered asthmatics in 1996-1997, in comparison to the Smith et al. (1997) study, which used 1987 National Medical Expenditure Survey (NMES) data. In comparing their study, the authors note that the 1987 NMES, 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 (see above for a discussion of costs versus charges).

The unit values and the corresponding distributions available in BenMAP for asthma-related ER visits are summarized in Table H-7.

**Table H-37.    Core Unit Values Available for Asthma-Related ER Visits**

| Basis for Estimate | Age Range | | Unit Value (2015$) | Distribution of Unit Value | Parameters of Distribution | |
|---|---|---|---|---|---|---|
| | Min | Max | | | P1 | P2 |
| COI: Smith et al. (1997) | 0 | 99 | $534 | Triangular | $395 | $738 |
| COI: Standford et al. (1999) | 0 | 99 | $447 | Normal | 8.95 | -- |

# H.3  Other Health Effect Occurrence

Monetary valuation estimates for health effect occurrences other than hospital admissions or emergency department visits are described below, listed in alphabetical order.

### H.3.1 Lung Cancer

The unit value for non-fatal lung cancer incidence is derived from the direct medical costs of lung cancer treatment estimated by Kaye et al. (2018). Lost earnings are assumed to be negligible because of the low labor force participation rate among the age groups at highest risk of developing lung cancer (average age of diagnosis is approximately 70 years). Lung cancer treatment costs depend to a large extent on the phase of care, with costs in the initial year of treatment ($17,422 for males) far exceeding the continuing costs of treatment in subsequent years ($3,269 for males). We calculated costs over a five-year span, beginning with the initial onset which occurs with a delay after exposure.  The initial year's treatment cost is summed with four years of continuing annual costs discounted by 3% and 7%.

Furthermore, Kaye et al. (2018) provides separate treatment cost estimates for men and women. The distribution of new lung cancer cases by sex in the United States from Siegel et

al. (2019) is approximately 51% male and 49% female. This distribution of new lung cancer cases was used to weight the sex-specific cost estimates from Kaye et al. (2018) to obtain a combined five-year cost estimate for both sexes. In order to adjust the cost estimate to 2015$ using a medical cost index, we assume that costs presented by Kaye et al. (2018) are in 2010$ as an approximate midpoint of the data years 2007-2012. Altogether, the cost of non-fatal lung cancer incidence over a five-year period is estimated to be $33,809 using a 3% discount rate or $32,548 using a 7% discount rate (Table H-8).

**Table H-38.   Core Unit Values Available for non-fatal Lung Cancer**

| Health Effect | Basis for Estimate | Age Range | | Unit Value (2015$) |
| | | Min | Max | |
|---|---|---|---|---|
| Lung Cancer | COI: 5 yrs med, 3% DR, Kaye (2018) | 65 | 99 | $33,809 |
| | COI: 5 yrs med, 7% DR, Kaye (2018) | 65 | 99 | $32,548 |

For an outcome such as lung cancer, there is an expected time lag between changes in pollutant exposure in a given year and the total realization of health effect benefits, commonly referred to in regulatory analyses as the "cessation lag." The time between exposure and diagnosis can be quite long, on the order of years to decades, to realize the full benefits of the air quality improvements. This latency period is important in order to properly discount the economic value of these health benefits.

To estimate the latency period, we performed a literature search using the keywords "non-fatal lung cancer," "lung cancer," "$PM_{2.5}$," "latency," and "incidence." Five papers that estimate the risk of lung cancer incidence from $PM_{2.5}$ exposure using a latency period were identified. The latency period length and country of the identified papers are summarized in Table H-9. Based on estimates of lung cancer latency from the literature, 10 years was the most common latency period estimate found in the literature (i.e., the mode).

**Table H-39.   Latency Periods Used in Lung Cancer Risk Assessment Papers**

| Study | Latency Period (years) | Location |
|---|---|---|
| Gogna et al., 2019 | 5 | Canada |
| Bai et al., 2020 | 4; 10 | Canada |
| Kulhanova et al., 2018 | 10 | France |
| Coleman et al., 2020 | 10; 15 | US |
| Harrison et al., 2004 | 20 | US |

To account for the latency period between air pollution reductions and avoided lung cancer diagnoses in our economic valuation estimates, we developed an age-at-diagnosis **cessation lag** distribution method based on an approach previously used to estimate avoided cases of kidney cancer in analyses of water quality rules (U.S. EPA, 2017). The method uses lung and bronchus cancer diagnosis age-distribution from the Surveillance, Epidemiology, and End Results Program (SEER). For this model, we assumed that the case reduction distribution would follow the age-pattern of cancer diagnosis between the age at which the exposure change occurs and 99 years. Table H-10 shows an example case reduction distribution calculation for an exposure change experienced at 55. SEER estimates 92.2% of lung and bronchus cancer cases occur in individuals 55 years and older. Dividing the percentages in the remaining age bins by 92.2% (the percent of lung and bronchus cancer diagnoses between the age of exposure change and end of lifetime), we find that there is a 24% chance that the risk reductions for a 55-year-old occur between ages 55 and 64, a 37% chance that the case reductions occurs between ages 65 and 74, etc. For distributing avoided cases within an age bin, we assume an equal incidence distribution across years within each bin.

**Table H-40.** **Percent Lung and Bronchus Cancer Incidence by Age and Distribution of Risk Reduction by Age for an Exposure Change at 55**

| Age Group | Percent New Cases per Year by Age* | Percent of New Cases Occurring at or After Age 55[1] |
|---|---|---|
| 0-20 | 0 | NA |
| 20-34 | 0.2 | NA |
| 35-44 | 0.9 | NA |
| 45-54 | 6.6 | NA |
| 55-64 | 21.8 | 24 |
| 65-74 | 34.1 | 37 |
| 75-84 | 26.6 | 29 |
| 85-99 | 9.7 | 11 |
| 55-99 | 92.2 | 100 |

*May not sum to 100% due to rounding

[1]Calculated as the percentage in column 2 divided by 92.2%, where 92.2% is the percentage of lung and bronchus incidence between age 55 and 99.

## H.3.2 Out of Hospital Cardiac Arrest

The COI for cardiac arrests occurring outside of the hospital is derived from O'Sullivan et al. (2011), who estimate three-year medical costs associated with cardiovascular disease events among adults ages 35 and older in the U.S. The authors rely on administrative claims data from a large U.S. health plan and develop econometric models to predict medical costs for 15 different cardiovascular events, including cardiac arrest, referred to as resuscitated cardiac arrest. The dataset includes over 20 million commercial and Medical Advantage members between 2002 and 2006. Cardiac arrests are identified using the ICD-9 code 427.5. The authors use propensity score matching to develop a control group with which to compare costs versus individuals that suffered cardiac arrest. Medical costs occurring within the month of the event were excluded to avoid double counting hospitalization costs, which are separately captured by the hospitalization valuation functions. Over three years, the total medical costs, excluding hospitalization, are $36,142 (undiscounted, inflated to 2015$), or $35,753 using a 3% discount rate and $35,282 for a 7% discount rate (Table H-11).

**Table H-41.    Valuation Estimate for Cardiac Arrests (2015$)**

| Costs | Cumulative Costs | Annual Costs | | |
|---|---|---|---|---|
| | | Undiscounted | 3% Discount Rate | 7% Discount Rate |
| Month of Event* | $43,904 | $43,904 | $43,904 | $43,904 |
| Year 1 | $71,901 | $27,997 | $27,997 | $27,997 |
| Year 2 | $74,701 | $2,800 | $2,718 | $2,617 |
| Year 3 | $80,046 | $5,345 | $5,038 | $4,668 |
| Years 1-3 | $80,046 | $36,142 | $35,753 | $35,282 |

*Excluded to avoid double-counting with hospitalization costs.

## H.3.3 Stroke

The COI of non-fatal stroke incidence is calculated from Mu et al. (2017) estimates of direct medical costs incurred during initial hospitalization and the 360 days following hospital discharge. The study identifies individuals experiencing a first-time stroke using ICD-9 codes 434 and 436. The authors analyze medical claims from January 2006 to March 2015 utilizing the retrospective IMS LifeLink PharMetrics Plus database for individuals ages 18 to 65, and Medicare Advantage and Medicare Supplemental Claims for individuals above the age of 65. The authors present acute care and long-term care costs stratified by three discharge classifications: dead at discharge, discharged with disability, and discharged without disability. We estimated the average costs for non-fatal cases by weighting the costs for individuals discharged with disability and without disability by their prevalence (23 and 77 percent, respectively). The resulting COI for non-fatal stroke incidence is $33,962 (2015$). This value reflects one-year of medical costs following stroke and does not include hospitalization costs, as these costs are separately captured by hospitalization valuation functions. We reviewed several studies that estimate longer-term medical costs (Goodwin et al., 2011, Lee et al., 2007, Luengo-Fernandez et al., 2012, Nicholson et al., 2016) and concluded that roughly three quarters of costs are incurred in first year after stroke occurrence.

# H.4 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 effects which are associated with one or more of these acute symptoms and illnesses, such as minor restricted activity days, school loss days, and work loss days, have also been associated with air pollution. We briefly discuss the derivation of the unit values for acute respiratory symptoms (minor restricted activity days), asthma exacerbation, and school loss days. Tables H-12 and H-13 summarize the values used by EPA in their regulatory impact analyses.

**Table H-42.   Additional Unit Values Available for Myocardial Infarction**

| Basis of Estimate | Age Range | | Medical Cost | Opportunity Cost | Total Cost |
|---|---|---|---|---|---|
| | Min | Max | | | |
| COI: 3 yrs med, 5 yrs wages, 3% DR, O'Sullivan (2011) | 0 | 24 | $48,796 | $0 | $48,796 |
| | 25 | 44 | $48,796 | $13,301 | $62,097 |
| | 45 | 54 | $48,796 | $19,604 | $68,400 |
| | 55 | 65 | $48,796 | $113,316 | $162,112 |
| | 66 | 99 | $48,796 | $0 | $48,796 |
| COI: 3 yrs med, 5 yrs wages, 7% DR, O'Sullivan (2011) | 0 | 24 | $47,623 | $0 | $47,623 |
| | 25 | 44 | $47,623 | $11,908 | $59,531 |
| | 45 | 54 | $47,623 | $17,552 | $65,175 |
| | 55 | 65 | $47,623 | $101,451 | $149,074 |
| | 66 | 99 | $47,623 | $0 | $47,623 |

**Table H-43.  Core Unit Values Available for Acute Symptoms and Illnesses**

| Health Effect | Basis for Estimate | Age Range Min | Age Range Max | Unit Value (2015$) | Distribution of Unit Value | Parameters of Distribution P1 | Parameters of Distribution P2 |
|---|---|---|---|---|---|---|---|
| Minor Restricted Activity Days | WTP: 1 day, CV studies | 18 | 99 | $70 | Triangular | 28.51 | 110.62 |
| New Onset Asthma | COI: lifetime med, lifetime productivity, 3% DR | 0 | 17 | $17,232 | Normal | 441.62 | 0 |
| | | 4 | 21 | $16,425 | Normal | 464.38 | 0 |
| | | 35 | 99 | $16,741 | Normal | 639.75 | 0 |
| | COI: lifetime med, lifetime productivity, 7% DR | 0 | 17 | $10,187 | Normal | 277.28 | 0 |
| | | 4 | 21 | $9,644 | Normal | 294.27 | 0 |
| | | 35 | 99 | $12,594 | Normal | 505.97 | 0 |
| Cough; Chest Tightness; Shortness of Breath; Wheeze | WTP: 1 symptom-day, Dickie and Mesmen (2004) | 0 | 17 | $219 | LogNormal | 5.390 | 0.078 |
| | | 18 | 99 | $115 | LogNormal | 5.390 | 0.078 |
| Albuterol Use | COI: use of inhaler | 0 | 99 | $0.35 | None | 0 | 0 |
| Allergic Rhinitis | COI: 1 yr med costs | 0 | 17 | $600 | None0 | 0 | 0 |
| Work Loss Days * | Median daily wage, county-specific | 18 | 65 | $173 | None | N/A | N/A |
| School Loss Days | Described in text | 0 | 17 | $106 | None | N/A | N/A |

\* Unit values for work loss days are county-specific, based on county-specific median wages. The unit value shown here is the national median daily wage, given for illustrative purposes only.

## H.4.1 Non-Fatal Myocardial Infarctions (Heart Attacks)

In the absence of a suitable WTP value for reductions in the risk of non-fatal heart attacks, there are a variety of cost-of-illness unit values available for use in BenMAP. These cost-of-illness unit values incorporate 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, the unit values include costs incurred over five years.

Economic values for acute myocardial infarctions (AMIs, also known as heart attacks) have been derived from O'Sullivan et al. (2011), which estimate three-year medical costs associated with cardiovascular disease events among adults ages 35 and older in the U.S. The authors rely on administrative claims data from a large U.S. health plan and develop econometric models to estimate medical costs for 15 different cardiovascular events, including AMIs. The dataset includes over 20 million commercial and Medical Advantage members between 2002 and 2006. AMIs are identified using the ICD-9 code 410. The authors use propensity score matching to develop a control group with which to compare costs versus individuals that suffered AMIs. We exclude medical costs within the month of the event in an attempt avoid double counting hospitalization costs, which would be captured separately in the hospitalization valuation. Over three years, the total medical costs, excluding initial hospitalization, are $49,758 (undiscounted, inflated to 2015$), or $48,796 using a 3% discount rate and $47,623 for a 7% discount rate (Table 25). This study analyzed costs associated with individuals ages 35 and older. We apply the total medical costs to all ages from zero to 99, although only a small portion (<10%) of annual AMI incidence occurs in the age range below 35.

We supplement AMI medical costs with estimates of lost earnings using age-specific estimates from Cropper and Krupnick (1990). Using a 3% discount rate, we estimated the following present discounted values in lost earnings over 5 years due to a heart attack: 0.219 times annual earnings for someone between the ages of 25 and 44, 3.534 times annual earnings for someone between the ages of 45 and 54, and 1.245 times annual earnings for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings using a 7% discount rate are 0.203, 3.287, and 1.158 times annual earnings, respectively. Cropper and Krupnick (1990) does not provide lost earnings for populations under 25 or over 65. As such we do not include lost earnings in the cost estimates for these age groups. These costs, along with the total valuation estimates for AMIs, are presented in Table H-9.

## H.4.2 Minor Restricted Activity Days (MRADs)

Two unit values are currently available in BenMAP for MRADs associated with acute respiratory symptoms. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). Although Ostro and Rothschild (1989) 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. The original unit value, then, assumes that MRADs associated with PM exposure may be more specifically defined as MRRADs, and uses the estimate of mean WTP to avoid a MRRAD.

IEc (1993) derived an estimate of WTP to avoid a 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 $).

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 effect 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.

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on

such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $20.03 (1999 $), for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 2015$, this is a triangular distribution centered at $69.58, ranging from $29 to $111.

The estimate for the MRADs that is used in EPA benefits analyses can be found in Table H-10.

### H.4.3 New Onset Asthma

The unit value of new onset asthma is reported by Belova et al. (2020), who estimate the lifetime costs of asthma using data from the 2002 to 2010 Medical Expenditure Panel Survey (MEPS). The authors identify all individuals with current asthma (9,409 out of 158,867 respondents) using the ICD-9 code 493 in the MEPS Medical Conditions Files. Additionally, they identify the date of asthma onset for these individuals. Using the MEPS Medical Events files, which capture most types of medical expenditures (e.g., hospitalizations, emergency room visits, outpatient visits, prescriptions), Belova et al., 2020 estimated annual expenditures by asthma duration and age at onset. The annual healthcare costs for asthma— as measured by healthcare expenditures by all paying parties—vary from $800 to $2,100 for children and $900 to $2,500 for adults (2015$). They extrapolate these values to a lifetime cost stream for an incident chronic asthma case to generate present value estimates by onset age using discount rates of 3% and 7%. Additionally, the authors consider productivity impacts

that capture 1) the probability of not being able to work due to health reasons, 2) the impact of asthma on occupational choice, and 3) impact of asthma on weekly earnings.

We adapted the Belova et al. (2020) estimates to align with the age groups 0 to 17, 4 to 21, and 35 to 99. This calculation entails weighting the Belova et al. (2020) age groups by their relative prevalence and propagating the standard errors to derive new uncertainty bounds. Confidence intervals are not provided for productivity losses because BenMAP is currently only capable of reflecting uncertainty in one parameter (in this case, medical costs) (Table H-10).

## H.4.4 Asthma Symptoms

The valuation estimates for cough, wheeze, chest tightness, and shortness of breath were estimated from the Dickie and Messman (2004) analysis of parents' WTP to relieve asthma symptoms in children and adults. The authors derive the WTP estimates from an attribute-based, stated-choice question assessing preferences to avoid acute illness as part of a survey performed in Hattiesburg, Mississippi in 2000. Survey respondents are asked to identify whether they or their child have experienced the following asthma symptoms in the past year: cough with phlegm, shortness of breath with wheezing, chest pain on deep inspiration, and fever with muscle pain and fatigue. Respondents were then assigned one of sixteen illness profiles varying by symptom, symptom duration, in days, as well as discomfort level. Dickie and Messman (2004) calculate the WTP for children ages zero to seventeen as $219, for one avoided mild symptom-day (2015$). The authors also provide WTP estimates by symptom, however, they represent six avoided symptom-days. Therefore, we apply the same WTP value, for one avoided mild symptom-day, to each asthma symptom effect.

We calculated the economic value for albuterol use associated with asthma symptoms through prescription costs for albuterol inhalers. Epocrates and GoodRx provide cost and actuation information for four common types of albuterol inhalers in 2020 dollars. Both

online resources utilize published price lists, purchases, claim records, and pharmaceutical data to provide clinical statistics. Epocrates and the FDA provide cost and actuation information for one additional, less common, albuterol inhaler.70 We divide the cost of inhalers by the actuations per inhaler to calculate an average cost per actuation across all inhaler types. We then adjust the values to 2015$ using the Consumer Price Index (CPI) for medical care. Since medical cost index data were unavailable for 2020 at the time of these calculations, we used the most recently available index (2019). The resulting value for asthma symptoms, albuterol use is $0.35 per actuation (2015$).

Table H-10 summarizes the unit values utilized by EPA for asthma related health effects.

## H.4.5 Allergic Rhinitis

We derived COI estimates for allergic rhinits (also referred to as hay fever) from the 2005 unit cost data presented by Soni (2008). The study utilizes data from the Medical Expenditure Panel Survey (MEPS) and identifies allergic rhinitis using ICD-9 code 477. Soni (2008) analyzes medical expenditures stratified by age group for the years 2000 and 2005, and calculate the cost-of-illness as the mean expenditures per person for ambulatory care, in-patient services, and prescription medications. The resulting COI for allergic rhinitis is $600 for ages zero to seventeen (2015$; Table H-10). The COI estimate represents mean annual medical costs for patients with hay fever. Given that the health impact function for this effect relates to allergic rhinitis prevalence, these estimates are more applicable than values representing only first-year costs.

## H.4.6 Work Loss Days (WLDs)

Work loss days are valued at a day's wage. BenMAP calculates county-specific median daily wages from county-specific annual wages by dividing by (52*5), on the theory that a worker's

vacation days are valued at the same daily rate as work days. The unit value for WLDs can be found in Table H-10.

## H.4.7 School Loss Days

We include two costs of school loss days: caregiver costs and loss of learning. We calculate each separately and then sum them. Caregiver costs are valued at their employers' average cost for employed caregivers. For unemployed caregivers, the opportunity cost of their time is calculated as the average take-home pay. The loss of learning is calculated based on the impact of absences on learning multiplied by the impact of school learning on adult earnings. The loss of learning estimate is currently only available for middle and high school students. The two costs are summed.

The caregiver costs assumes that an adult caregiver stays home with the child and loses any wage income they would have earned that day. For working caregivers, we follow EPA guidance and value their time at the average wage including fringe benefits and overhead costs (U.S. EPA, 2020a). The average daily wage in 2021 was $195 (2015$, assumed to be the average weekly wage divided by 5, US Bureau of Labor Statistics, 2021a), which yields an average daily labor cost of $340 for employed parents applying average multiplier of 1.46 for fringe benefits and 1.2 for overhead. For nonworking caregivers, we assume that the opportunity cost of time is the average after tax earnings. We estimate the income tax rate for a median household to be 7%, yielding net earnings of $195*0.93 or $181 ($2015). The income tax rate of 7% is the percentage difference in median post-tax income and median income from (U.S. Census Bureau, 2021).

The probability that a parent is working is measured with the employment population ratio among people with their own children under 18 and is 77.2% (US Bureau of Labor Statistics, 2021b). Combining the cost of working and nonworking caregivers yields a caregiver cost of $305 per school loss day.

To measure the loss of learning, we update the Liu et al., 2021 estimate. Liu et al., 2021 estimated the impact of a school absence on learnings as measured by an end-of-course test score. We multiply by an estimate of the impact of learning as measured by end-of-course test scores on adult income from Chetty et al., 2014. This approach yields an estimated learning loss of $2,230 per school absence (discounted at 3%) or $975 per school absence (discounted at 7%).

We updated the Chetty et al., 2014 estimate to use 2010 income and to estimate lifetime incomes discounted at 3% and 7%. Liu et al., 2021 provide an estimate that a school absence leads to a $1,200 reduction in lifetime earnings, based on the Chetty et al., 2014 estimate that lifetime earnings are $522,000 (2010$). We use 2010 ACS data from IPUMS to calculate expected lifetime earnings of $892,579 (discounting at 3%) and $390,393 (discounting at 7%). We then multiply the Liu et al., 2021 estimate of $1200 by (892,579/522,000) and (390,393/522,000) and convert from 2010 dollars to 2015 dollars based on the Consumer Price Index for All Urban Consumers.

We use caregiver costs for preschool and elementary school children and the sum of caregiver costs and loss of learning for middle school and high school students. We calculate that 31% of children under 18 are middle school and high school ages 13-18, assuming each bin distributed equally), so the combined average effect is ($305 + $2,230*0.31) or $1000 with 3% discounting, or ($305 + $975*0.31) or $610 with 7% discounting (U.S. Census Bureau, 2010).

A unit value based on the approach described above is likely to understate the value of a school loss day in at least four ways:

1. It omits WTP to avoid the symptoms/illness which resulted in the school absence
2. It omits the opportunity cost of time for non-working caregivers' day
3. The approach omits other aspects of school attendance such as social and emotional development or meals

4. It does not account for deleterious effects on student learning in other subjects.

The unit value for school loss days can be found in Table H-10.

# H.5 Developing Income Growth Adjustment Factors

Chapter 4 of the BenMAP User Manual provides instructions for formatting and adding income growth data. These values are used to adjust WTP estimates for growth in real income. As discussed in that chapter, evidence and theory suggest that WTP should increase as real income increases. When reviewing the economic literature to develop income growth adjustment factors, it is important to have an economist assist. For an overview of valuation, see Chapter 4: Valuation and Discounting.

Adjusting WTP to reflect growth in real income requires three steps:

1. *Identify relevant income elasticity estimates from the peer-reviewed literature.*
2. *Calculate changes in future income.*
3. *Calculate adjustments to WTP based on changes in future income and income elasticity estimates.*

1. Identifying income elasticity estimates

Income elasticity estimates relate changes in demand for goods to changes in income. Positive income elasticity suggests that as income rises, demand for the good also rises. Negative income elasticity suggests that as income rises, demand for the good falls. BenMAP does not adjust Cost-of-Illness (COI) estimates according to changes in income elasticity due to the fact that COI estimates the direct cost of a health outcome; instead we adjust this metric using inflation factors described above. BenMAP includes income elasticity estimates specific to the type of health effect associated with the WTP estimate. BenMAP contains elasticity estimates for three types of health effects: minor, severe and mortality. Minor health

effects are those of short duration. Severe, or chronic, health effects are of longer duration. Consistent with economic theory, the peer reviewed literature indicates that income elasticity varies according to the severity of the health effect. A review of the literature revealed a range of income elasticity estimates that varied across the studies and according to the severity of health effect. Table H-14 summarizes the income elasticity estimates for minor health effects, severe health effects and mortality. Here we have provided a lower-, upper- and central-elasticity estimate for each type of health effect.

**Table H-44.    Income Elasticity Estimates**

| Health Effect | Lower Bound | Central Estimate | Upper Bound |
|---|---|---|---|
| Minor Health Effect | 0.04 | 0.15 | 0.30 |
| Severe and Chronic Health Effects | 0.25 | 0.45 | 0.60 |
| Mortality | 0.08 | 0.40 | 1.00 |

2. Calculating changes in future income

The next input to the WTP adjustment is annual changes in income. Historical US Gross Domestic Product (GDP) data (1990-2016) comes from the U.S. Bureau of Commerce's Bureau of Economic Analysis (BEA). GDP values were adjusted for inflation using the BEA's price index for GDP. We divided historical GDP values by populations provided by the BEA to estimate GDP per capita to maintain internal consistency in the calculation. Future changes in annual income are based on data presented in the Annual Energy Outlook (AEO) 2020, a report prepared by the U.S. Energy Information Administration (EIA) (AEO, 2020). AEO published annual GDP projections through the year 2050, which were adjusted for inflation using the GDP Chain-type Price Index reported by AEO. We divided projected GDP values by AEO's population projections to estimate per capita GDP, again maintaining internal consistency in the calculation.

3. Calculating changes in WTP

The income elasticity estimates from Table H-11 and the estimated changes in future income may then be used to estimate changes in future WTP for each health effect. The adjustment formula follows four steps:

**Equation H-2**

1. $\varepsilon = \dfrac{\frac{\Delta WTP}{WTP}}{\frac{\Delta I}{I}} = \dfrac{(WTP_2 - WTP_1) \times (I_2 + I_1)}{(I_2 - I_1) \times (WTP_2 + WTP_1)}$

2. $\varepsilon I_2 WTP_2 + \varepsilon I_2 WTP_1 - \varepsilon I_1 WTP_2 - \varepsilon I_1 WTP_1 = I_2 WTP_2 + I_1 WTP_2 - I_2 WTP_1 - I_1 WTP_1$

3. $WTP_2 \times (\varepsilon I_2 - \varepsilon I_1 - I_2 - I_1) = WTP_1 \times (\varepsilon I_1 - \varepsilon I_2 - I_1 - I_2)$

4. $WTP_2 = WTP_1 \times \dfrac{\varepsilon I_1 - \varepsilon I_2 - I_1 - I_2}{\varepsilon I_2 - \varepsilon I_1 - I_2 - I_1}$

Table H-15 summarizes the income-based WTP adjustments used within BenMAP for minor health effects, severe health effects, and premature mortality. BenMAP applies the "mid" income growth adjustment to the WTP for each corresponding health effect. The "low" and "upper" are provided for bounding the "mid" estimate.

## Table H-45. Income-Based WTP Adjustments by Health Effect and Year

| Year | Minor Health Endpoint | | | Severe Health Endpoint | | | Mortality | | |
|---|---|---|---|---|---|---|---|---|---|
| | Low | Mid | Upper | Low | Mid | Upper | Low | Mid | Upper |
| 1990 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1991 | 0.999425 | 0.997845 | 0.995695 | 0.996411 | 0.99355 | 0.991409 | 0.99885 | 0.994264 | 0.985722 |
| 1992 | 1.000278 | 1.001043 | 1.002086 | 1.001738 | 1.003131 | 1.004177 | 1.000556 | 1.002783 | 1.006971 |
| 1993 | 1.000845 | 1.003171 | 1.006353 | 1.005291 | 1.009545 | 1.012746 | 1.00169 | 1.00848 | 1.021335 |
| 1994 | 1.001941 | 1.007299 | 1.014651 | 1.012194 | 1.022057 | 1.029519 | 1.003886 | 1.019583 | 1.049686 |
| 1995 | 1.002529 | 1.009516 | 1.019122 | 1.01591 | 1.028821 | 1.038614 | 1.005064 | 1.025578 | 1.065196 |
| 1996 | 1.003545 | 1.01336 | 1.026899 | 1.022366 | 1.040622 | 1.054532 | 1.007103 | 1.036027 | 1.092569 |
| 1997 | 1.004809 | 1.018155 | 1.036642 | 1.030442 | 1.055472 | 1.074652 | 1.009642 | 1.049157 | 1.127596 |
| 1998 | 1.006098 | 1.023062 | 1.046661 | 1.038734 | 1.070818 | 1.095552 | 1.012234 | 1.062703 | 1.164493 |
| 1999 | 1.007498 | 1.02841 | 1.057639 | 1.047803 | 1.087723 | 1.1187 | 1.015052 | 1.077598 | 1.205983 |
| 2000 | 1.008676 | 1.032928 | 1.066959 | 1.055489 | 1.102148 | 1.138556 | 1.017428 | 1.090286 | 1.242108 |
| 2001 | 1.008675 | 1.032924 | 1.06695 | 1.055482 | 1.102134 | 1.138537 | 1.017426 | 1.090274 | 1.242075 |
| 2002 | 1.008984 | 1.03411 | 1.069403 | 1.057504 | 1.105943 | 1.143797 | 1.018048 | 1.093621 | 1.251727 |
| 2003 | 1.009739 | 1.037019 | 1.075433 | 1.062468 | 1.115325 | 1.15678 | 1.019574 | 1.101858 | 1.275708 |
| 2004 | 1.010863 | 1.041352 | 1.084451 | 1.069884 | 1.129408 | 1.176348 | 1.021844 | 1.114208 | 1.312269 |
| 2005 | 1.011864 | 1.045228 | 1.092549 | 1.076534 | 1.142112 | 1.194079 | 1.02387 | 1.125332 | 1.345838 |
| 2006 | 1.012602 | 1.04809 | 1.09855 | 1.081456 | 1.151559 | 1.207315 | 1.025363 | 1.133594 | 1.371175 |
| 2007 | 1.012957 | 1.049471 | 1.101452 | 1.083835 | 1.156139 | 1.213647 | 1.026084 | 1.137596 | 1.383575 |
| 2008 | 1.012532 | 1.047821 | 1.097984 | 1.080992 | 1.150667 | 1.206063 | 1.025223 | 1.132814 | 1.368769 |
| 2009 | 1.011166 | 1.042525 | 1.086897 | 1.071894 | 1.13324 | 1.181689 | 1.022457 | 1.117566 | 1.322336 |
| 2010 | 1.011843 | 1.045146 | 1.092377 | 1.076393 | 1.141841 | 1.1937 | 1.023827 | 1.125095 | 1.345117 |
| 2011 | 1.012165 | 1.046395 | 1.094994 | 1.07854 | 1.145958 | 1.199463 | 1.024479 | 1.128697 | 1.356115 |
| 2012 | 1.012764 | 1.04872 | 1.099873 | 1.082541 | 1.153646 | 1.210245 | 1.025692 | 1.135418 | 1.376817 |
| 2013 | 1.013213 | 1.050467 | 1.103547 | 1.085551 | 1.159447 | 1.2184 | 1.026602 | 1.140487 | 1.392581 |
| 2014 | 1.013911 | 1.053184 | 1.109275 | 1.090241 | 1.168516 | 1.231181 | 1.028017 | 1.148402 | 1.417472 |
| 2015 | 1.014754 | 1.056473 | 1.116227 | 1.095927 | 1.179559 | 1.246797 | 1.029727 | 1.158031 | 1.4482 |
| 2016 | 1.015112 | 1.057873 | 1.119194 | 1.098352 | 1.184283 | 1.253496 | 1.030454 | 1.162147 | 1.461489 |
| 2017 | 1.015291 | 1.058571 | 1.120676 | 1.099562 | 1.186645 | 1.25685 | 1.030817 | 1.164204 | 1.468167 |
| 2018 | 1.016092 | 1.061712 | 1.127353 | 1.105013 | 1.197312 | 1.272029 | 1.032446 | 1.173487 | 1.498591 |
| 2019 | 1.016945 | 1.06506 | 1.134495 | 1.110837 | 1.208762 | 1.288383 | 1.03418 | 1.183439 | 1.531756 |
| 2020 | 1.017494 | 1.067221 | 1.139119 | 1.114604 | 1.216197 | 1.299038 | 1.035298 | 1.189894 | 1.553577 |
| 2021 | 1.017858 | 1.068652 | 1.142185 | 1.1171 | 1.221138 | 1.306201 | 1.036037 | 1.194181 | 1.568204 |
| 2022 | 1.018224 | 1.070097 | 1.145287 | 1.119624 | 1.226144 | 1.313335 | 1.036784 | 1.198523 | 1.583131 |
| 2023 | 1.018585 | 1.071523 | 1.148352 | 1.122117 | 1.231098 | 1.320474 | 1.03752 | 1.202817 | 1.598006 |
| 2024 | 1.018991 | 1.073127 | 1.151804 | 1.124923 | 1.236689 | 1.328546 | 1.038347 | 1.20766 | 1.61492 |
| 2025 | 1.019446 | 1.074925 | 1.155682 | 1.128074 | 1.242981 | 1.337648 | 1.039273 | 1.213106 | 1.634116 |
| 2026 | 1.019915 | 1.076785 | 1.159702 | 1.131337 | 1.249515 | 1.347122 | 1.040231 | 1.218759 | 1.654237 |
| 2027 | 1.02041 | 1.078749 | 1.163955 | 1.134787 | 1.256443 | 1.357191 | 1.041242 | 1.224747 | 1.675778 |
| 2028 | 1.020937 | 1.080842 | 1.168495 | 1.138469 | 1.263858 | 1.367993 | 1.042318 | 1.231152 | 1.699074 |
| 2029 | 1.021436 | 1.082827 | 1.17281 | 1.141964 | 1.270919 | 1.378307 | 1.043337 | 1.237246 | 1.721494 |
| 2030 | 1.021959 | 1.08491 | 1.17735 | 1.145639 | 1.278367 | 1.389214 | 1.044406 | 1.243669 | 1.745395 |
| 2031 | 1.022479 | 1.086985 | 1.181879 | 1.149303 | 1.285815 | 1.400148 | 1.045469 | 1.250087 | 1.76956 |
| 2032 | 1.022969 | 1.088943 | 1.186166 | 1.152768 | 1.292879 | 1.410546 | 1.046472 | 1.256169 | 1.792727 |
| 2033 | 1.023487 | 1.091016 | 1.190711 | 1.15644 | 1.300389 | 1.421627 | 1.047532 | 1.262629 | 1.817622 |
| 2034 | 1.024004 | 1.093088 | 1.195265 | 1.160115 | 1.307929 | 1.432783 | 1.048592 | 1.269111 | 1.842903 |
| 2035 | 1.024493 | 1.095052 | 1.199589 | 1.163603 | 1.315106 | 1.443429 | 1.049594 | 1.275275 | 1.867234 |
| 2036 | 1.02498 | 1.097006 | 1.203902 | 1.16708 | 1.322282 | 1.454101 | 1.050592 | 1.281434 | 1.891828 |
| 2037 | 1.025478 | 1.099009 | 1.208332 | 1.170647 | 1.329668 | 1.465113 | 1.051613 | 1.287768 | 1.917424 |
| 2038 | 1.025984 | 1.101051 | 1.212857 | 1.174289 | 1.33723 | 1.476418 | 1.052652 | 1.294248 | 1.943931 |
| 2039 | 1.026492 | 1.103099 | 1.217405 | 1.177947 | 1.34485 | 1.487839 | 1.053694 | 1.300771 | 1.970954 |
| 2040 | 1.027004 | 1.105171 | 1.222018 | 1.181654 | 1.352597 | 1.499482 | 1.054748 | 1.307398 | 1.998757 |
| 2041 | 1.027508 | 1.107212 | 1.226569 | 1.185309 | 1.36026 | 1.511031 | 1.055784 | 1.313948 | 2.026591 |
| 2042 | 1.02803 | 1.109326 | 1.231295 | 1.189101 | 1.368236 | 1.523084 | 1.056857 | 1.320759 | 2.055917 |
| 2043 | 1.028555 | 1.111457 | 1.236071 | 1.19293 | 1.376316 | 1.535329 | 1.057937 | 1.327653 | 2.086006 |
| 2044 | 1.029075 | 1.113573 | 1.240821 | 1.196736 | 1.384373 | 1.547576 | 1.059008 | 1.334521 | 2.116397 |
| 2045 | 1.029605 | 1.115728 | 1.245672 | 1.20062 | 1.392623 | 1.560152 | 1.060099 | 1.341548 | 2.147923 |
| 2046 | 1.030116 | 1.117812 | 1.250373 | 1.20438 | 1.400637 | 1.572403 | 1.061152 | 1.348368 | 2.178951 |
| 2047 | 1.030638 | 1.119946 | 1.255197 | 1.208236 | 1.408882 | 1.585045 | 1.06223 | 1.355378 | 2.211295 |
| 2048 | 1.031135 | 1.121978 | 1.259801 | 1.211913 | 1.416771 | 1.597176 | 1.063255 | 1.36208 | 2.242655 |
| 2049 | 1.03162 | 1.123965 | 1.264312 | 1.215513 | 1.424519 | 1.609122 | 1.064256 | 1.368656 | 2.273847 |
| 2050 | 1.032088 | 1.125883 | 1.268677 | 1.218994 | 1.432035 | 1.620744 | 1.065222 | 1.37503 | 2.304491 |

# H.6 Inflation Indices

Chapter 4 of the BenMAP User Manual provides instructions for formatting and adding inflation data. These values are used to adjust economic values to express monetary units in a consistent dollar year. As discussed in that chapter, BenMAP includes inflation factors for three different types of values. The source for these values is included in Table H-16. These values were re-indexed to $2015 prior to import in BenMAP.

**Table H-46.    Inflation Factors**

| Name | Description | Years | Source |
|------|-------------|-------|--------|
| All Goods Index | Value of generic goods and services | | BLS, Data Series CUUR0000SA0 at http://data.bls.gov/cgi-bin/surveymost?cu |
| Medical Cost Index | Value of medical expenses | 1980-2020 | BLS, Data Series CUUR0000SAM at http://data.bls.gov/cgi-bin/surveymost?cu |
| Wage Index | Value of wages | | BLS, Employment Cost Trends. Table 5 at http://www.bls.gov/web/eci/ecicois.txt |

# Appendix I.  Additional Health Valuation Functions in BenMAP

In this Appendix, we present additional health valuation functions that are included in BenMAP but are not currently used regularly in U.S. EPA regulatory analyses. **PLACEHOLDER: Information on additional functions will be included here as they are added to the tool by EPA.**

# Appendix J.    Population & Other Data in BenMAP

This section describes the population, monitor, and demographic data for the United States included in BenMAP. It consists of the following three subsections:

Population Data. This describes how BenMAP forecasts population; the block-level and county-level data underlying the forecasts; and the PopGrid software application, which aggregates block-level population data to whatever grid definition might be needed. The population data currently in the web tool through year 2050 relies on the same procedures described in this Appendix. The web tool adds a population projection for 2055 based on a linear extrapolation of the projection trend through 2050 from the desktop tool. Implementation of user-input population data and projections, e.g., using PopGrid tool, will be included in a future release of the web tool.

Monitor Data. The current version of the web tool does not support the use of monitor data for air quality surfaces.  Support for monitor data will be included in a future release.

Demographic Datasets. This subsection describes the various U.S. datasets in BenMAP related to demography: household size, poverty rates, and educational attainment. The web tool currently includes the full set of data described in this appendix; however the variables used by the tool are limited to those included in the default set of Health Impact Functions. Broader user access to these variables and more complete integration with user-specified health impact functions will be implemented in a future version.

# J.1   Population Data for the U.S.

BenMAP calculates health impacts for any desired grid definition, so long as you have a shapefile for that grid definition and population data for that grid definition. In this description, we use the term "population grid cell" to refer to a cell (e.g., county) within a grid definition. The foundation for calculating the U.S. population in the population grid-cells is 2010 Census block data. A separate application called "PopGrid," described below, combines the Census block data with any user-specified set of population grid- cells, so long as they are defined by a GIS shape file. Unfortunately, PopGrid relies on extremely large census files that are too large to include with BenMAP – hence the need for the separate application. The PopGrid program is available on the BenMAP website here: www.epa.gov/benmap

Within any given population grid-cell, BenMAP has 304 unique race-ethnicity-gender-age groups: 19 age groups by 2 ethnic groups by gender by 4 racial groups (19*2*2*4=304). Table J-1 presents the 304 population variables available in BenMAP. As discussed below, these variables are available for use in developing age estimates in whatever grouping you require.

**Table J-47.   Demographic Groups and Variables Available in BenMAP**

| Racial Group | Ethnicity | Age | Gender |
|---|---|---|---|
| White, African American, Asian, American Indian | Hispanic, Non-Hispanic | <1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+ | Male, Female |

In this section on U.S. population data in BenMAP, we describe:

Forecasting Population. This describes how BenMAP forecasts population.

Data Needed. This section describes the block-level and county-level data underlying the forecasts.

PopGrid. This section reviews the PopGrid software application, which aggregates block-level population data to whatever grid definition might be need.

## J.1.1  How BenMAP Forecasts Population

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

**Equation J-1**

$$age_{3-12} = \frac{1}{2} \times age_{1-4} + age_{5-9} + \frac{3}{5} \times age_{10-14}$$

To estimate population levels for the years after the last Census in 2010, BenMAP scales the 2010 Census-based estimate with the ratio of the county-level forecast for the future year of interest over the 2010 county-level population level. Woods & Poole (2015) provides the county-level population forecasts used to calculate the scaling ratios; these data are discussed in detail below.

In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2020, BenMAP calculates:

**Equation J-2**

$$age_{4-9,g,2020} = age_{4-9,g,2010} \times \frac{age_{4-9,county,2020}}{age_{4-9,county,2010}}$$

Where the gth population grid-cell is wholly located within a given county.

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

**Equation J-3**

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

Multiplying this fraction with the number of individuals ages 4 to 9 in the year 2010 gives an estimate of the number of individuals ages 4 to 9 that reside in the fraction of the county within the gth grid-cell in the year 2010:

**Equation J-4**

$$age_{4-9, g \ in \ county_c, 2010} = age_{4-9, g, 2010} \times fraction \ age_{4-9, g \ in \ county_c}$$

To then forecast the population in 2020, we scale the 2010 estimate with the ratio of the county projection for 2020 to the county projection for 2010:

**Equation J-5**

$$age_{4-9, g \ in \ county_c, 2020} = age_{4-9, g \ in \ county_c, 2010} \times \frac{age_{4-9, county_c, 2020}}{age_{4-9, county_c, 2010}}$$

Combining all these steps for "n" counties within the gth grid-cell, we forecast the population of persons ages 4 to 9 in the year 2020 as follows:

**Equation J-6**

$$age_{4-9, g, 2020} = \sum_{c=1}^{n} age_{4-9, g, 2010} \times \frac{total \ pop_{g \ in \ county_c}}{total \ pop_g} \times \frac{age_{4-9, county_c, 2020}}{age_{4-9, county_c, 2010}}$$

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

**Equation J-7**

$$age_{4-9,g,2020} = \sum_{c=1}^{n} age_{4-9,g,2010} \times \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \times \frac{age_{4-9,\ county_c,2020}}{age_{4-9,\ county_c,2010}}$$

$$age_{10-14,g,2020} = \sum_{c=1}^{n} age_{10-14,g,2010} \times \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \times \frac{age10-14_{10-14,\ county_c,2020}}{age_{10-14,\ county_c,2010}}$$

$$age_{4-12,g,2020} = age_{4-9,\ g,2020} + \frac{3}{5} \times age_{10-14,\ g,2020}$$

To estimate population for 2055, we extrapolate Woods and Poole projections from the 2045 to 2050 period:

**Equation J-8**

$$W_{2055,i} = W_{2050,i} * \frac{W_{2050,i}}{W_{2045,i}}$$

where *W* is the growth weight (relative to 2010) and *i* is each demographic cell (i.e., unique combinations of county, gender, ethnicity, race, and age range).

## J.1.2  Data Needed for Forecasting

Underlying the population forecasts in BenMAP there are block-level databases used to provide year 2010 population estimates and a county-level database of forecast ratios. Both files have the same set of 304 race-ethnicity-gender-age population groups.

The block-level data is typically not used directly in BenMAP, and instead is used with the PopGrid software (described below) to provide year 2010 estimates for a grid definition of interest (e.g., 12 kilometer CMAQ grid). The output from PopGrid with the year 2010 population estimates can then be loaded into BenMAP.

The county-level data comes pre-installed and is not something that you need to load yourself. These data are simply county-level ratios of a year (2009, 2011-2050) and year 2010 population data for each county and each of the 304 race-ethnicity-gender-age population groups.

We describe the development of each databases below.

### J.1.2.1 Block-Level Census 2010

There are about five million "blocks" in the United States, and for each block we have 304 race-ethnicity-gender-age groups. The block-level population database is created separately for each state, in order to make the data more manageable. (A single national file of block data would be about six gigabytes.)

The initial block file from the U.S. Census Bureau is not in the form needed. The block data has 7 racial categories and 23 age groups, as opposed to the 4 and 19 used in BenMAP. Table J-2 summarizes the initial set of variables and the final desired set of variables.

**Table J-48.    Race, Ethnicity and Age Variables in 2010 Census Block Data**

| Type | Race | Ethnicity | Gender | Age |
|------|------|-----------|--------|-----|
| Initial Variables (SF1 file) | White Alone, Black Alone, Native American Alone, Asian Alone, Pacific Islander/Hawaiian Alone, Other Alone, Two or More Alone | -- | Male, Female | 0-4, 5-9, 10-14, 15-17, 18-19, 20, 21, 22-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-61, 62-64, 65-66, 67-69, 70-74, 75-79, 80-84, 85+ |
| Final Desired Variables | White, African-American, Asian-American, Native-American | Hispanic, Non-Hispanic | Female, Male | <1,1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+ |

The initial set of input files are as follows.

Census 2010 block-level and tract-level files (Summary File 1)

Data: http://www2.census.gov/census_2010/04-Summary_File_1/

Docs: http://www.census.gov/prod/cen2010/doc/sf1.pdf

Census 2000 MARS national-level summary

Docs: http://www.census.gov/popest/archives/files/MRSF-01-US1.pdf

The SF1 tract-level and MARS data, as described below, are needed to reorganize the variables that come initially in the block-levelSF1 file. (For the sake of completeness, we note that there exists a county-level Census 2000 MARS file; however, due to major population count discrepancies between the county-level MARS file and block-level SF1 file, we used only the nation-level summary table. Tables in MARS documentation file did not have the discrepancies that the county-level file had. We were unable to get an adequate explanation of this from the U. S. Census.)

The steps in preparing the data are as follows:

**1. Adjust Age-classifications:**

We combined some age groups in the block-level SF1 data to match the age groups wanted for BenMAP. For example, we combined age groups 15-17 and 18-19 to create the 15-19 age group used in BenMAP. Then, in the case of the 0-4 age group, we split it into <1 and 1-4 using the tract-level SF1 data, which gave us the fraction of 0-4 year-olds who are <1.

**2. Fill in Missing Racial-Ethnic Interactions:**

We used the tract-level SF1 data to calculate the fraction of Hispanics in each ethnically-aggregated subpopulation from the block-level data, by age and sex. We used these fractions to distribute each age-sex-race-block-level datum into Hispanics and non-Hispanics.

**3. Assign "Other" and "Multi-Racial" to the Remaining Four Racial Categories:**

We assign the "Other" race category in two steps. First, based on the national MARS data, we estimated how many people in the "multi-racial" category checked off "some other race" as one of their races, for Hispanics and non-Hispanics separately. In each age-sex-race-block-level datum, we added those people to "other race" category to create the re-distribution pool, analogously to the method implemented by Census while creating MARS data (see U.S. Census Bureau, 2002a, Table 1, below). Second, based on the national re-allocation fractions for Hispanics and non-Hispanics (derived from the MARS data), we assigned the "Other" race into the four races of interest and "multi-race".

After the assignment of the "Other" race category, we then assigned "multi-racial" category to the four racial categories, using state fractions of these races in each age-sex-race-block-level datum.

Exhibit J-3: Summary of Modified Race and Census 2000 Race Distributions for the United States

| Subject | Modified Race Number | Percent | Census 2000 Number | Percent |
|---|---|---|---|---|
| **TOTAL POPULATION** | 281,421,906 | 100.00 | 281,421,906 | 100.00 |
| One race | 277,524,226 | 98.62 | 274,595,678 | 97.57 |
| Specified race only | 277,524,226 | 98.62 | 259,236,605 | 92.12 |
| White | 228,104,485 | 81.05 | 211,460,626 | 75.14 |
| Black or African American | 35,704,124 | 12.69 | 34,658,190 | 12.32 |
| American Indian and Alaska Native | 2,663,818 | 0.95 | 2,475,956 | 0.88 |
| Asian | 10,589,265 | 3.76 | 10,242,998 | 3.64 |
| Native Hawaiian and Other Pacific Islander | 462,534 | 0.16 | 398,835 | 0.14 |
| | | | | |
| Non-specified race only | (X) | (X) | 15,359,073 | 5.46 |
| | | | | |
| **Two races** | 3,578,053 | 1.27 | 6,368,075 | 2.26 |
| Specified race only | 3,578,053 | 1.27 | 3,366,517 | 1.20 |
| Specified and non-specified races | (X) | (X) | 3,001,558 | 1.07 |
| | | | | |
| **Three or more races** | 319,627 | 0.11 | 458,153 | 0.16 |
| Specified race only | 319,627 | 0.11 | 297,298 | 0.11 |
| Specified and non-specified races | (X) | (X) | 160,855 | 0.06 |
| **HISPANIC OR LATINO AND RACE** | 35,305,818 | 100.00 | 35,305,818 | 100.00 |
| One race | 34,814,386 | 98.61 | 33,081,736 | 93.70 |
| Specified race only | 34,814,386 | 98.61 | 18,190,433 | 51.52 |
| White | 32,529,000 | 92.13 | 16,907,852 | 47.89 |
| Black or African American | 1,391,117 | 3.94 | 710,353 | 2.01 |
| American Indian and Alaska Native | 566,378 | 1.60 | 407,073 | 1.15 |
| Asian | 232,461 | 0.66 | 119,829 | 0.34 |
| Native Hawaiian and Other Pacific Islander | 95,430 | 0.27 | 45,326 | 0.13 |
| | | | | |
| Non-specified race only | (X) | (X) | 14,891,303 | 42.18 |
| | | | | |
| **Two races** | 433,726 | 1.23 | 2,110,965 | 5.98 |
| Specified race only | 433,726 | 1.23 | 315,611 | 0.89 |
| Specified and non-specified races | | | 1,795,354 | 5.09 |
| | | | | |
| **Three or more races** | 57,706 | 0.16 | 113,117 | 0.32 |
| Specified race only | 57,706 | 0.16 | 48,933 | 0.14 |
| Specified and non-specified races | (X) | (X) | 64,184 | 0.18 |
| **NOT HISPANIC OR LATINO AND RACE** | 246,116,088 | 100.00 | 246,116,088 | 100.00 |
| One race | 242,709,840 | 98.62 | 241,513,942 | 98.13 |
| Specified race only | 242,709,840 | 98.62 | 241,046,172 | 97.94 |
| White | 195,575,485 | 79.46 | 194,552,774 | 79.05 |
| Black or African American | 34,313,007 | 13.94 | 33,947,837 | 13.79 |
| American Indian and Alaska Native | 2,097,440 | 0.85 | 2,068,883 | 0.84 |
| Asian | 10,356,804 | 4.21 | 10,123,169 | 4.11 |
| Native Hawaiian and Other Pacific Islander | 367,104 | 0.15 | 353,509 | 0.14 |
| | | | | |
| Non-specified race only | (X) | (X) | 467,770 | 0.19 |
| | | | | |
| **Two races** | 3,144,327 | 1.28 | 4,257,110 | 1.73 |
| Specified race only | 3,144,327 | 1.28 | 3,050,906 | 1.24 |
| Specified and non-specified races | (X) | (X) | 1,206,204 | 0.49 |
| | | | | |
| **Three or more races** | 261,921 | 0.11 | 345,036 | 0.14 |
| Specified race only | 261,921 | 0.11 | 248,365 | 0.10 |
| Specified and non-specified races | (X) | (X) | 96,671 | 0.04 |
| (X) Not applicable. | | | | |

U.S. Census Bureau, Census 2000                    Procedure 3-3

## J.1.2.2       County-Level Forecasts

Woods & Poole (2015) developed county-level forecasts for each year from 2000 through 2050, by age and gender for non-Hispanic White, African-American, Asian-American, and

Native-American and for all Hispanics. The detailed documentation can be found at
http://www. woodsandpoole.com/pdfs/CED15.pdf. As discussed below, the adjustments
necessary to prepare the data for use in BenMAP are relatively straightforward.

For each non-Hispanic subset of the population and each year from 2000-2050, we divided
the Woods and Poole population for that year by the Woods and Poole population for that
subset in 2010. These serve as the growth coefficients for the non-Hispanic subsets of each
race. We used a similar calculation to determine the growth rates for the Hispanic population.
We assume that each Hispanic race grows at the same rate, and use these growth rates for the
Hispanic subsets of each race.[14]

**Matching Age Groups Used in BenMAP**

There are 86 age groups, so it is a simple matter of aggregating age groups to match the 19
used in BenMAP.

**Matching Counties Used in U.S. Census**

The county geographic boundaries used by Woods & Poole are somewhat more aggregated
than the county definitions used in the 2010 Census and those in BenMAP, and the FIPS codes
used by Woods and Poole are not always the standard codes used in the Census. To make the
Woods and Poole data consistent with the county definitions in BenMAP, we disaggregated
the Woods and Poole data and changed some of the FIPS codes to match the U.S. Census.

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[14] Previous versions of the BenMAP program used a different methodology whereby population estimates for 2000
– 2009 were adjusted using the ratio of 2000 Woods & Poole estimated population and 2000 Census population.

**Calculating Growth Ratios with Zero Population in 2010**

There are a small number of cases were the 2010 county population for a specific demographic group is zero, so the ratio of any future year to the year 2010 data is undefined. In these relatively rare cases, we prepared statewide and national totals and used ratios at the higher levels of geographic aggregation when the more local ratios caused divide-by-zero errors.

# J.2   U.S. Demographic Datasets in BenMAP

BenMAP includes county-level data on household size, poverty status, educational attainment, unemployment, health insurance coverage, and occupational status. We describe the data sources and processing methodology for each dataset below. All estimates were generated at the county level for 3,109 counties in the contiguous United States.[15]

## J.2.1  Household Size

To generate average household size for each county, we utilize ACS 5-year estimates for 2012 to 2016. Average household size was provided by ACS at the county level for all counties except for two, for which data were not available.[16] For these counties, we applied the state level average household size.

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[15] In 2013, Bedford city, Virginia was removed from the list of counties in the U.S. Due to BenMAP's grid definition, we continue to include Bedford city (FIPS code 51515) in this update. We impute the value for this county using the value for the county with which it was combined (Bedford County, FIPS code 51019). In 2015, Oglala Lakota County, South Dakota (46102) changed name and code from Shannon County (46113). To match BenMAP's grid definition, we use the old FIPS code (46113) for this county. For further information, please see the Census website: https://www.census.gov/programs-surveys/geography/technical-documentation/county-changes.html

[16] The two counties without data were Shannon County, South Dakota (FIPS Code 46113) and Bedford, Virginia (FIPS Code 51515).

## J.2.2 Educational Attainment

We use data from the ACS to provide county-level summaries of educational attainment. These data represent 5-year average ACS estimates from 2015 to 2019. Specifically, the data included in BenMAP span two broad education categories: no high school diploma (termed "no_hs_degree"), and high school diploma (or equivalency) and above (termed "hs_degree_plus"). The latter category includes individuals with a high school diploma (or equivalency), some college, college degree, or post-graduate degree and ranges from 53-98%.

For both education groups (with/without HS diploma), we estimate the fraction of the total county population (ages 25 years and above) in each education group. Thus, the two estimates sum to one for each county.

For each estimate, we generate a coefficient of variation (CV) equal to the ratio of the standard error to the point estimate. For counties with a CV greater than 0.3 (1.7% percent of all counties), we impute the county-level estimate with a state-level estimate following Census guidance, which defines any estimate with a CV greater than 0.3 as low reliability and to be used with extreme caution (King et al. 2015).

## J.2.3 Poverty Status

To determine the poverty status at the county level, we utilize ACS 5-year estimates from 2015 to 2019. The resulting datasets represent the fraction of the total population in the county that falls below the federal poverty line (termed "below_poverty_line") and the fraction of the population that falls above the poverty line (termed "above_poverty_line"). The EPA Standard Variables dataset also includes two variables (termed "below_2x_poverty_line" and "above_2x_poverty_line") representing the fraction of the county-level population below and above 200% of the poverty line. The county-level

proportions below the poverty line range from 3-55% and below 200% of the poverty line range from 8-75%.

For each estimate, we generate a coefficient of variation (CV) equal to the ratio of the standard error to the point estimate. For counties with a CV greater than 0.3 (1.7% percent of all counties), we impute the county-level estimate with a state-level estimate following Census guidance, which defines any estimate with a CV greater than 0.3 as low reliability and to be used with extreme caution (King et al. 2015).

## J.2.4 Unemployment Rates

BenMAP includes county-level employment variables representing average rates from 2017 to 2021. Importantly, the employment variables are adjusted for use in BenMAP to use *total population* as the denominator rather than *labor force*. This allows you to multiply the rates by the total population (in a health impact function) to assess populations that are (a) employed, (b) unemployed, and (c) not part of the labor force (e.g., retirees, students, discouraged workers).

County-level unemployment rates are from the Bureau of Labor Statistics. We adjusted these rates using county-level population estimates from the U.S. Census Bureau from 2017 to 2021. The calculations were done for each year individually and then averaged together to create a five-year average. The value calculated in BenMAP represents the average rate across all months within this period. The rate of residents not in the labor force was calculated by subtracting the labor force from the total population and then dividing the remainder by the total population.

## J.2.5  Health Insurance

We use data from the Small Area Health Insurance Estimates (SAHIE) collected by the U.S. Census Bureau from 2015 to 2019 to calculate the percentage of individuals with and without health insurance in each county. The SAHIE date provides the number of individuals with and without health insurance by county. Calculations were done for each year individually and then averaged together to create a five-year average.

## J.2.6  Blue Collar Workers

We use five-year estimates (2012-2016) from the ACS to estimate the fraction of each county's labor force employed in white collar and blue collar occupations. The dataset includes the number of employed individuals over 16 that work within five occupation categories. We assign each of these five occupations to either the blue collar or white collar designation, as shown in Table J-3.

**Table J-49.    Mapping Occupations to Blue Collar and White Collar Designations**

| Occupation | Designation |
|---|---|
| Management, business, science and arts | White collar |
| Service | White collar |
| Sales and office | White collar |
| Natural resources, construction and maintenance | Blue collar |
| Production, transportation and material moving | Blue collar |

We calculate the fraction of each county in blue collar professions by dividing the total number of individuals employed in blue collar jobs by the total number of employed individuals within each county. The same calculation is done for white collar professions. We adjust FIPS codes to match the BenMAP county grid definition, as described in section J.3.5.

# Appendix K.   Uncertainty & Pooling

This Appendix discusses the treatment of uncertainty in BenMAP, both for incidence changes and associated dollar benefits. Some background is then given on pooling methodology. **Pooling functionality is not implemented in the current version of the tool, though we expect it to be addressed in a future version.**

## K.1  Uncertainty

Although there are several sources of uncertainty affecting estimates of incidence changes and associated benefits, the sources of uncertainty that are most readily quantifiable in benefit analyses are uncertainty surrounding the health impact functions and uncertainty surrounding unit dollar values. The total dollar benefit associated with a given health effect depends on how much the health effect will change in the control scenario (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a statistical death avoided is worth).

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by distributions. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies. Although such an "uncertainty distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy 1990, pp. 168-172). Unlike a sampling distribution (which describes the possible values that an estimator of an unknown value might take on), this uncertainty distribution describes our beliefs about what values the unknown value itself might be.

Such uncertainty distributions can be constructed for each underlying unknown (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknowns (such as the total dollar benefit of a regulation). In either case, an uncertainty distribution is a characterization of our beliefs about what the unknown (or the function of unknowns) is likely to be, based on all the available relevant information. Uncertainty statements based on such distributions are typically expressed as 90 percent credible intervals. This is the interval from the fifth percentile point of the uncertainty distribution to the ninety-fifth percentile point. The 90 percent credible interval is a "credible range" within which, according to the available information (embodied in the uncertainty distribution of possible values), we believe the true value to lie with 90 percent probability. The uncertainty surrounding both incidence estimates and dollar benefits estimates can be characterized quantitatively in BenMAP. Each is described separately below.

## K.1.1 Characterization of Uncertainty Surrounding Incidence Changes

To calculate point estimates of the changes in incidence of a given adverse health effect associated with a given set of air quality changes, BenMAP performs a series of calculations at each grid-cell. First, it accesses the health impact functions needed for the analysis, and then it accesses any data needed by the health impact functions. Typically, these include the grid-cell population, the change in population exposure at the grid-cell, and the appropriate baseline incidence rate. BenMAP then calculates the change in incidence of adverse health effects for each selected health impact function. The resulting incidence change is stored, and BenMAP proceeds to the next grid-cell, where the above process is repeated.

In *Latin Hypercube* mode, BenMAP reflects the uncertainty surrounding estimated incidence changes (resulting from the sampling uncertainty surrounding the pollutant coefficients in the health impact functions used) by producing a *distribution* of possible incidence changes rather than a single point estimate. To do this, it uses the distribution (*Dist Beta*) associated with the pollutant coefficient (*Beta*, or β), and potentially the point estimate (*Beta*) and two

parameters (*P1Beta, P2Beta*). Typically, pollutant coefficients are normally distributed, with mean Beta and standard deviation *P1Beta*.

BenMAP uses an N-point Latin Hypercube to represent the underlying distribution of β and to create a corresponding distribution of incidence changes in each population grid cell, where N is specified by you. The Latin Hypercube method represents an underlying distribution by N percentile points of the distribution, where the *n*th percentile point is equal to:

### Equation K-1

$$(n-1) \times \frac{100}{N} + \frac{100}{2N}$$

The Latin Hypercube method is used to enhance computer processing efficiency. It is a sampling method that divides a probability distribution into intervals of equal probability, with an assumption value for each interval assigned according to the interval's probability distribution. Compared with conventional Monte Carlo sampling, the Latin Hypercube approach is more precise over a fewer number of trials because the distribution is sampled in a more even, consistent manner (Decisioneering, 1996, pp. 104-105).

Suppose, for example, that you elect to use a 20-point Latin Hypercube. BenMAP would then represent the distribution of β by 20 percentile points, specifically the 2.5th, 7.5th, ..., 97.5th. To do this, the inverse cumulative distribution function specified by the distribution of β is called with the input probability equal to each the 20 percentile points. BenMAP then generates an estimate of the incidence change in a grid-cell for each of these values of β, resulting in a distribution of N incidence changes. This distribution is stored, and BenMAP proceeds to the next population grid-cell, where the process is repeated.

## K.1.2 Characterization of Uncertainty Surrounding Dollar Benefits

The uncertainty distribution of the dollar benefits associated with a given health or welfare effect is derived from the two underlying uncertainty distributions - the distribution of the change in incidence of the effect (number of cases avoided) and the distribution of the value of a case avoided (the "unit value"). The derivation of the uncertainty distribution for incidence change is described above. The distributions used to characterize the uncertainty surrounding unit values are described in detail in the appendix on the Economic Value of Health Effects. As noted in that Appendix, a variety of distributions have been used to characterize the uncertainty of unit values, including uniform, triangular, normal, and Weibull.

To represent the underlying distribution of uncertainty surrounding unit values, a 100-point Latin Hypercube is generated in the same way described in the previous section for the distribution of $\beta$. That is, the unit value distribution is represented using the 0.5th, 1.5th, ..., and 99.5th percentile values of its distribution.

A distribution of the uncertainty surrounding the dollar benefits associated with a given health effect is then derived from Latin Hypercube values generated to represent the change in incidence and the Latin Hypercube values generated to represent the unit value distribution. To derive this new distribution, each of the 100 unit values is multiplied by each of the N incidence change values, yielding a set of 100 * N dollar benefits. These values are sorted low to high and binned down to a final distribution of N dollar benefit values.

# Appendix L.    Batch Run Approach

**Placeholder: Future versions of the tool will include batch processing functionality, but this is not yet implemented.**

# Appendix M.   Function Editor

The function editor is used to develop both health impact functions and valuation functions. This appendix describes the syntax of this editor. **This functionality is not implemented in the current version of the tool, though we expect it to be present in a future version.**

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