



**Environmental Benefits Mapping and Analysis Program –
Community Edition**

User's Manual

March 2023

Updated for BenMAP-CE Version 1.5.8

Table of Contents

<i>Chapter 1</i>	Welcome to BenMAP-CE	1-1
1.1	Overview of BenMAP-CE & Benefits Assessment	1-1
1.2	How to Use this Manual.....	1-6
1.3	Computer Requirements.....	1-7
1.4	Installing BenMAP-CE	1-8
1.5	Uninstalling BenMAP-CE.....	1-9
1.6	Contacts for Comments, Questions & Bug Reporting.....	1-10
1.7	Sources for More Information	1-11
1.8	Frequently Asked Questions (General)	1-12
<i>Chapter 2</i>	Terminology	2-1
<i>Chapter 3</i>	Overview of BenMAP-CE Features	3-1
3.1	Core Program.....	3-4
3.1.1	Create Air Quality Surfaces	3-5
3.1.2	Estimate Health Impacts	3-7
3.1.3	Aggregate, Pool, and Value	3-9
3.2	Menus	3-11
3.2.1	Tools Menu.....	3-11
3.2.2	Help Menu.....	3-13
3.3	Outputs	3-14
3.3.1	Results.....	3-14
3.3.2	Maps.....	3-15
3.3.3	Audit Trail Report.....	3-15
3.3.4	Dataset Validation Reports	3-17
3.3.5	File Types.....	3-17
3.4	Frequently Asked Questions	3-19
<i>Chapter 4</i>	Loading Data.....	4-1
4.1	Add, Modify, and Delete a Setup	4-3
4.1.1	Grid Definitions	4-5
4.1.2	Pollutants.....	4-13
4.1.3	Monitor Datasets	4-24
4.1.4	Incidence and Prevalence Rates Data.....	4-30
4.1.5	Population Data.....	4-36
4.1.6	Health Impact Functions	4-43
4.1.7	Variable Data.....	4-53
4.1.8	Inflation Data.....	4-58

4.1.9	Valuation Data	4-62
4.1.10 Income Growth Data	4-68
4.2	Export and Import Setups	4-71
4.3	Frequently Asked Questions	4-72
<i>Chapter 5</i>	<i>Creating Air Quality Surfaces</i>	<i>5-1</i>
5.1	Model Data	5-4
5.2	Monitor Data	5-6
5.2.1	Closest Monitor for Monitor Data	5-7
5.2.2	Voronoi Neighborhood Averaging (VNA) for Monitor Data	5-8
5.2.3	Fixed Radius for Monitor Data.....	5-10
5.2.4	Custom Monitor Filtering for EPA Standard Monitors	5-10
5.3	Monitor Rollback.....	5-15
5.3.1	Example: A Single Rollback in One Region.....	5-16
5.3.2	Example: Combining Three Rollback Approaches in Different Regions ...	5-20
5.4	Open *.aqgx File	5-24
5.5	Frequently Asked Questions	5-25
<i>Chapter 6</i>	<i>Estimating Incidence</i>	<i>6-1</i>
6.1	Introduction to Estimating Health Incidence.....	6-3
6.2	Create a Health Impact Configuration.....	6-4
6.2.1	Air Quality Surfaces – Pollutants and Air Quality Grids	6-4
6.2.2	Estimate Health Impacts – Population and Health Impact Functions.....	6-7
6.2.3	Advanced Configuration Settings	6-12
6.3	Run Health Impact Configuration	6-14
6.4	View and Export Health Impact Results.....	6-15
6.5	Frequently Asked Questions	6-19
<i>Chapter 7</i>	<i>Aggregating, Pooling, and Valuing</i>	<i>7-1</i>
7.1	Introduction to Valuation, Discounting, and Pooling	7-2
7.1.1	Overview of Economic Valuation	7-2
7.1.2	Overview of Discounting.....	7-5
7.1.3	Overview of Pooling	7-10
7.2	Create Aggregation, Pooling, and Valuation (APV) Configuration.....	7-17
7.2.1	Selecting Aggregation Levels.....	7-17
7.2.2	Pooling Incidence Results.....	7-18
7.2.3	Valuing Pooled Incidence Results	7-28
7.2.4	APV Configuration Advanced Settings	7-33
7.3	Open & Modify Existing APV Configuration	7-37
7.4	Run APV Configuration.....	7-37

7.5 View and Export Pooled Incidence and Valuation Results	7-38
7.5.1 Pooled Incidence Results	7-38
7.5.2 Pooled Valuation Results	7-38
7.6 Frequently Asked Questions	7-38
<i>Chapter 8 GIS/Mapping</i>	8-1
8.1 Overview of Mapping	8-2
8.2 Results Panel.....	8-2
8.3 GIS Mapping Panel.....	8-4
8.3.1 GIS Map Tab.....	8-4
8.3.2 Data Tab	8-11
8.3.3 Chart Tab.....	8-12
8.3.4 Audit Trail Report.....	8-12
8.4 Frequently Asked Questions	8-14
<i>Chapter 9 Tools Menu</i>	9-1
9.1 Air Quality Surface Aggregation	9-3
9.2 Database Export.....	9-5
9.3 Database Import.....	9-9
9.4 Online Database Export.....	9-10
9.5 Online Database Import	9-11
9.6 Export Air Quality Surface.....	9-12
9.7 GBD Rollback.....	9-13
9.7.1 Create New Scenario.....	9-15
9.7.2 Select Regions or Countries	9-15
9.7.3 Choose Rollback Settings	9-16
9.7.4 Choose Mortality Valuation Settings.....	9-17
9.7.5 Execute Scenarios and Save Results.....	9-20
9.8 Monitor Data Conversion.....	9-22
9.9 Neighbor File Creator.....	9-22
9.10 PopSim	9-24
9.11 Options.....	9-26
9.12 Compute Grid Crosswalk.....	9-26
<i>Appendix A. Monitor Rollback Algorithms</i>	A-1
A.1 Percentage Rollback.....	A-1
A.2 Incremental Rollback	A-2
A.3 Rollback to a Standard.....	A-2
A.3.1 Interday Rollback – Generating Target Metric Values	A-3
A.3.2 Intraday Rollback - Adjusting Hourly Observations	A-6
A.3.3 Intraday Rollback - Incremental.....	A-11

Appendix B. Algorithms for Estimating Air Pollution Exposure.....	B-1
B.1 Direct Modeling.....	B-2
B.2 Closest Monitor	B-2
B.3 Voronoi Neighbor Averaging (VNA)	B-3
B.4 Fixed Radius	B-6
B.5 Monitor or Model Data with Missing Days.....	B-6
Appendix C. Deriving Health Impact Functions.....	C-1
C.1 Overview	C-1
C.2 Review Relative Risk and Odds Ratio.....	C-2
C.3 Linear Model	C-3
C.4 Log-linear Model	C-3
C.5 Logistic Model.....	C-5
C.6 Cox Proportional Hazards Model	C-11
Appendix D. Health Incidence & Prevalence Data in U.S. Setup	D-1
D.1 Mortality.....	D-1
D.1.1 Mortality Data for 2012-2014.....	D-1
D.1.2 Mortality Rate Projections 2015-2060	D-4
D.1.3 Ethnicity-Stratified Mortality Incidence.....	D-6
D.1.4 Ethnicity-Stratified Mortality Projections (2020-2060)	D-6
D.1.5 Race-Stratified Mortality Incidence	D-8
D.1.6 Race-Stratified Mortality Projections (2020-2060).....	D-9
D.2 Hospitalizations	D-12
D.3 Nonfatal Heart Attacks.....	D-17
D.4 Emergency Department Visits	D-18
D.5 School Loss Days	D-20
D.6 Other Acute and Chronic Effects.....	D-21
D.6.1 Acute Bronchitis.....	D-22
D.6.2 Chronic Bronchitis Incidence Rate	D-22
D.6.3 Chronic Bronchitis Prevalence Rate.....	D-22
D.6.4 Lower Respiratory Symptoms.....	D-22
D.6.5 Minor Restricted Activity Days (MRAD)	D-23
D.6.6 Work Loss Days.....	D-23
D.7 Asthma-Related Health Effects.....	D-23
D.7.1 New Onset Asthma.....	D-24
D.7.2 Shortness of Breath	D-24
D.7.3 Wheeze.....	D-24
D.7.4 Cough.....	D-24
D.7.5 Albuterol Use.....	D-25

D.7.6	Upper Respiratory Symptoms	D-25
D.7.7	Asthma Population Estimates.....	D-25
D.8	Other Health Endpoint Occurrence.....	D-26
D.8.1	Allergic Rhinitis.....	D-26
D.8.2	Lung Cancer	D-27
D.8.3	Out of Hospital Cardiac Arrest.....	D-27
D.8.4	Stroke.....	D-28
Appendix E.	Core Particulate Matter Health Impact Functions in U.S. Setup	E-1
E.1	Long-term Mortality	E-1
E.1.1	Di et al. (2017).....	E-1
E.1.2	Turner et al. (2016).....	E-2
E.1.3	Woodruff et al. (2008).....	E-2
E.1.4	Pope et al. (2019)	E-3
E.1.5	Wu et al. (2020).....	E-3
E.2	Chronic/Severe Illness.....	E-4
E.2.1	Ensor et al. (2013).....	E-5
E.2.2	Gharibvand et al. (2017)	E-5
E.2.3	Kioumourtzoglou et al. (2016)	E-6
E.2.4	Kloog et al. (2012).....	E-7
E.2.5	Peters et al. (2001)	E-7
E.2.6	Pope et al. (2006)	E-8
E.2.7	Rosenthal et al. (2008).....	E-9
E.2.8	Silverman et al. (2010).....	E-9
E.2.9	Sullivan et al. (2005).....	E-10
E.2.10	Wei et al. (2019).....	E-10
E.2.11	Zanobetti and Schwartz (2006).....	E-11
E.2.12	Zanobetti et al. (2009).....	E-11
E.3	Hospitalizations	E-12
E.3.1	Bell et al. (2015).....	E-13
E.3.2	Ostro et al. (2009)	E-13
E.4	Emergency Room Visits.....	E-14
E.4.1	Ostro et al. (2016)	E-14
E.4.2	Krall et al. (2013).....	E-15
E.5	Minor Effects	E-15
E.5.1	Ostro (1987).....	E-16
E.5.2	Ostro and Rothschild (1989).....	E-17
E.5.3	Parker et al. (2009).....	E-18
E.6	Asthma-Related Effects	E-18

E.6.1	Rabinovitch et al. (2006).....	E-19
E.6.2	Tétreault et al. (2016)	E-19
E.7	Sensitivity Analysis – General.....	E-20
E.7.1	Di et al. (2017).....	E-21
E.7.2	Jones et al. (2015)	E-22
E.7.3	McConnell et al. (2010).....	E-22
E.7.4	Nishimura et al. (2013).....	E-23
E.7.5	Pope et al. (2015)	E-23
E.7.6	Talbott et al. (2014).....	E-24
E.7.7	Turner et al. (2016).....	E-24
E.7.8	Zanobetti et al. (2009).....	E-25
E.8	Sensitivity Analysis – At-Risk Populations.....	E-25
E.8.1	Alhanti et al. (2016)	E-26
E.8.2	Di et al. (2017).....	E-27
E.8.3	Pope et al. (2019)	E-27
Appendix F.	Core Ozone Health Impact Functions in U.S. Setup	F-1
F.1	Short-term Mortality	F-1
F.1.1	Katsouyanni et al. (2009).....	F-1
F.1.2	Turner et al. (2016).....	F-2
F.1.3	Zanobetti and Schwartz (2008).....	F-3
F.2	Hospital Admissions	F-3
F.2.1	Katsouyanni et al. (2009).....	F-4
F.3	Emergency Room Visits.....	F-4
F.3.1	Barry et al. (2018).....	F-5
F.4	Minor Effects	F-5
F.4.1	Gilliland et al. (2001)	F-6
F.4.2	Ostro and Rothschild (1989).....	F-7
F.4.3	Parker et al. (2009).....	F-9
F.5	Asthma-Related Effects	F-9
F.5.1	Lewis et al. (2013)	F-10
F.5.2	Tétreault et al. (2016)	F-10
F.6	Sensitivity Analysis – General.....	F-11
F.6.3	Katsouyanni et al. (2009).....	F-13
F.6.4	Turner et al. (2016).....	F-13
F.7	Sensitivity Analysis – At-Risk Populations.....	F-14
F.7.1	Cakmak et al. (2006).....	F-17
F.7.2	Jerrett et al. (2009).....	F-17
F.7.3	Katsouyanni et al. (2009).....	F-18

F.7.4	Lin et al. (2005).....	F-18
F.7.5	Mar and Koenig (2009).....	F-19
F.7.6	Medina-Ramon & Schwartz (2008).....	F-19
F.7.7	Paulu and Smith (2008).....	F-21
F.7.8	Villeneuve et al. (2007).....	F-21
F.7.9	Zanobetti and Schwartz (2008b).....	F-22
Appendix G.	Additional Health Impact Functions in U.S. Setup	G-1
G.1	Additional PM _{2.5} Health Impact Functions	G-1
G.1.1	Long-term Mortality.....	G-1
G.1.1.1	Expert Functions.....	G-3
G.1.1.2	Baxter et al. (2017)	G-26
G.1.1.3	Global Exposure Mortality Model (Burnett et al., 2018)	G-26
G.1.1.4	Ito et al. (2013).....	G-28
G.1.1.5	Jerrett et al. (2017)	G-28
G.1.1.6	Krewski et al. (2009).....	G-29
G.1.1.7	Laden et al. (2006).....	G-30
G.1.1.8	Lepeule et al. (2012)	G-30
G.1.1.9	Pope et al. (2002)	G-31
G.1.1.10	Pope et al. (2015).....	G-32
G.1.1.11	Thurston et al. (2016).....	G-32
G.1.1.12	Turner et al. (2016)	G-33
G.1.1.13	Woodruff et al. (1997).....	G-33
G.1.1.14	Woodruff et al. (2006).....	G-33
G.1.1.15	Zanobetti et al. (2014).....	G-34
G.1.2	Chronic/Severe Illness.....	G-34
G.1.2.1	Abbey et al. (1995b).....	G-35
G.1.2.2	Peters et al. (2001).....	G-36
G.1.2.3	Pope et al. (2006)	G-36
G.1.2.4	Sullivan et al. (2005)	G-36
G.1.2.5	Zanobetti and Schwartz (2006)	G-36
G.1.2.6	Zanobetti et al. (2009).....	G-36
G.1.3	Hospitalizations	G-36
G.1.3.1	Babin et al. (2007).....	G-37
G.1.3.2	Bell et al. (2008, 2012).....	G-38
G.1.3.3	Ito (2003).....	G-38
G.1.3.4	Kloog et al. (2012).....	G-40
G.1.3.5	Moolgavkar (2000a).....	G-40
G.1.3.6	Moolgavkar (2000b), Cardiovascular	G-41

G.1.3.7 Moolgavkar (2003)	G-42
G.1.3.8 Peng et al. (2008)	G-42
G.1.3.9 Peng et al. (2009)	G-43
G.1.3.10 Sheppard (2003)	G-44
G.1.3.11 Zanobetti et al. (2009)	G-44
G.1.4 Emergency Room Visits	G-45
G.1.4.1 Glad et al. (2012)	G-45
G.1.4.2 Mar et al. (2010)	G-46
G.1.4.3 Norris et al. (1999)	G-46
G.1.4.4 Slaughter et al. (2005)	G-47
G.1.5 Minor Effects	G-47
G.1.5.1 Dockery et al. (1996)	G-47
G.1.5.2 Ostro (1987)	G-48
G.1.5.3 Ostro and Rothschild (1989)	G-48
G.1.5.4 Schwartz and Neas (2000)	G-48
G.1.6 Asthma-Related Effects	G-49
G.1.6.1 Mar et al. (2004)	G-50
G.1.6.2 Ostro et al. (2001)	G-51
G.1.6.3 Pope et al. (1991)	G-52
G.2 Additional Ozone Health Impact Functions	G-53
G.2.1 Short-term Mortality	G-53
G.2.1.1 Bell et al. (2004)	G-55
G.2.1.2 Bell et al. (2005)	G-56
G.2.1.3 Huang et al. (2005)	G-57
G.2.1.4 Ito and Thurston (1996)	G-57
G.2.1.5 Ito et al. (2005)	G-58
G.2.1.6 Jerrett et al. (2009)	G-59
G.2.1.7 Levy et al. (2005)	G-60
G.2.1.8 Moolgavkar et al. (1995)	G-60
G.2.1.9 Samet et al. (1997)	G-60
G.2.1.10 Schwartz (2005)	G-61
G.2.1.11 Smith et al. (2009)	G-61
G.2.1.12 Zanobetti and Schwartz (2008)	G-62
G.2.2 Hospital Admissions	G-62
G.2.2.1 Burnett et al. (2001)	G-63
G.2.2.2 Katsouyanni et al. (2009)	G-64
G.2.2.3 Moolgavkar et al. (1997)	G-64
G.2.2.4 Schwartz (1994a)	G-65

G.2.2.5 Schwartz (1994b).....	G-65
G.2.2.6 Schwartz (1995)	G-66
G.2.3 Emergency Room Visits	G-67
G.2.3.1 Glad et al. (2012)	G-67
G.2.3.2 Ito et al. (2007).....	G-67
G.2.3.3 Mar and Koenig (2009).....	G-68
G.2.3.4 Peel et al. (2005).....	G-68
G.2.3.5 Sarnat et al. (2013)	G-69
G.2.3.6 Wilson et al. (2005)	G-69
G.2.4 Minor Effects.....	G-70
G.2.4.1 Chen et al. (2000)	G-70
G.2.4.2 Gilliland et al. (2001)	G-72
G.2.4.3 Ostro and Rothschild (1989).....	G-72
G.2.5 Asthma-Related Effects.....	G-72
G.2.5.1 Mortimer et al. (2002).....	G-72
G.2.5.2 O'Connor et al. (2008)	G-73
G.2.5.3 Schildcrout et al. (2006).....	G-73
G.2.6 Converting Functions to 8-Hour Daily Maximum Metric.....	G-73
Appendix H. Core Health Valuation Functions in U.S. Setup.....	H-1
H.1 Mortality.....	H-1
H.1.1 Value of a Statistical Life Based on 26 Studies.....	H-2
H.2 Hospital Admissions & Emergency Room Visits.....	H-3
H.2.1 Hospital Admissions.....	H-3
H.2.2 Emergency Room Visits.....	H-6
H.2.3 Emergency Room Visits for Asthma	H-7
H.3 Other Health Endpoint Occurrence.....	H-8
H.3.1 Lung Cancer	H-8
H.3.2 Out of Hospital Cardiac Arrest.....	H-10
H.3.3 Stroke.....	H-10
H.4 Acute Symptoms and Illness Not Requiring Hospitalization	H-11
H.4.1 Non-Fatal Myocardial Infarctions (Heart Attacks)	H-12
H.4.2 Minor Restricted Activity Days (MRADs)	H-13
H.4.3 New Onset Asthma.....	H-14
H.4.4 Asthma Symptoms	H-15
H.4.5 Allergic Rhinitis.....	H-15
H.4.6 Work Loss Days (WLDs).....	H-16
H.4.7 School Loss Days.....	H-16
H.5 Developing Income Growth Adjustment Factors	H-17

H.6 Inflation Indices	H-21
Appendix I. Additional Health Valuation Functions in U.S. Setup	I-1
I.1 Mortality.....	I-1
I.1.1 Value of a Statistical Life Based on Selected Studies.....	I-1
I.2 Hospital Admissions	I-1
I.3 Chronic Illness	I-2
I.3.1 Chronic Bronchitis	I-2
I.3.2 Chronic Bronchitis Reversals.....	I-5
I.3.3 Chronic Asthma.....	I-5
I.4 Acute Symptoms and Illness Not Requiring Hospitalization	I-6
I.4.1 Non-Fatal Myocardial Infarctions (Heart Attacks)	I-7
I.4.2 Acute Bronchitis in Children	I-9
I.4.3 Minor Restricted Activity Days (MRADs)	I-10
I.4.4 Asthma Exacerbation.....	I-10
I.4.5 Upper Respiratory Symptoms (URS) in Children.....	I-12
I.4.6 Lower Respiratory Symptoms (LRS) in Children.....	I-13
I.4.7 Any of 19 Respiratory Symptoms	I-14
Appendix J. Population & Other Data in U.S. Setup.....	J-1
J.1 Population Data in U.S. Setup.....	J-1
J.1.1 How BenMAP Forecasts Population.....	J-2
J.1.2 Data Needed for Forecasting.....	J-3
J.1.3 PopGrid.....	J-7
J.2 Monitor Data in U.S. Setup.....	J-15
J.2.1 Data Processing.....	J-15
J.2.2 Output Files.....	J-16
J.3 Demographic Datasets in U.S. Setup	J-16
J.3.1 Household Size	J-17
J.3.2 Educational Attainment.....	J-17
J.3.4 Unemployment Rates	J-19
J.3.5 Health Insurance.....	J-19
J.3.6 Blue Collar Workers	J-20
J.3.7 Linguistic Isolation	J-20
J.3.8 Redlining.....	J-21
Appendix K. Uncertainty & Pooling.....	K-1
K.1 Uncertainty	K-1
K.1.1 Characterization of Uncertainty Surrounding Incidence Changes	K-2
K.1.2 Characterization of Uncertainty Surrounding Dollar Benefits	K-3
K.2 Pooling.....	K-3

K.2.1	Weights Used for Pooling.....	K-4
K.2.2	Mechanics of Pooling in BenMAP	K-8
K.2.3	Summing Distributions.....	K-9
K.2.4	Subtracting Distributions	K-10
Appendix L.	Command Line BenMAP.....	L-1
L.1	Overview.....	L-1
L.2	Variables	L-1
L.3	Commands.....	L-2
L.3.1	Set Active Setup.....	L-2
L.3.2	Create AQG	L-2
L.3.3	Run CFG	L-6
L.3.4	Run APV.....	L-7
L.3.5	Generate Report.....	L-8
L.4	Example 1	L-10
L.5	Example 2	L-12
Appendix M.	Function Editor	M-1
M.1	User Defined Variables	M-1
M.2	The Script Language.....	M-2
M.3	Operands.....	M-3
M.4	Operations.....	M-3
M.5	Arithmetic Functions.....	M-4
M.6	Aggregate Functions	M-5
References	R-1

Chapter 1

Welcome to BenMAP-CE

In this chapter, find...

- **An overview of the tool.**
- **Instructions for installing the tool.**
- **Contacts, sources for more information, and answers to frequently asked general questions.**

The environmental Benefits Mapping and Analysis Program—Community Edition (BenMAP-CE) is a powerful, yet easy-to-use program that estimates the number and economic value of health impacts resulting from changes in air pollution concentrations. The open-source BenMAP-CE tool replaces the proprietary version of the program (BenMAP) that the U.S. Environmental Protection Agency (U.S. EPA) first developed in 2003 to analyze national-scale air quality policies. These 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 Locomotive Marine Engine Rule (2008).

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

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

While the BenMAP-CE development team designed the program to be accessible to novice users, the tool includes a number of features that will appeal to advanced analysts as well. For example, analysts can add their own health impact and valuation functions, map results, and perform a suite of sensitivity analyses. Beginning users can take advantage of U.S. EPA's pre-programmed settings and reports in the core program.

1.1 Overview of BenMAP-CE & Benefits Assessment

The BenMAP-CE program estimates the human health impacts and economic value of air quality changes. That is — BenMAP-CE relates air quality changes to human health benefits. 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-CE to inform air quality management decisions.¹

BenMAP-CE estimates benefits from improvements in human health, such as reductions in the risk of premature death, heart attacks, and other adverse health effects. Other benefits of reducing air pollution (i.e., visibility and ecosystem effects) are not quantified in the current version of BenMAP-CE. After estimating the reductions in the incidence of adverse health effects, BenMAP-CE calculates the monetary benefits associated with those reductions.

How does BenMAP-CE estimate human health effects?

First, BenMAP-CE determines the change in ambient air pollution using user-specified air quality data. Because BenMAP-CE does not model air quality changes, these data

¹ For a list of peer-reviewed articles that used the BenMAP and BenMAP-CE tools, see: www.epa.gov/benmap

must be input into BenMAP-CE as modeling data or generated from air pollution monitoring data (although some monitoring data is pre-loaded in BenMAP-CE, see Chapter 5 for details). Next, BenMAP-CE relates the change in pollution concentration with certain health effects (also known as health endpoints, see Chapter 6 for details). This relationship is often referred to as the health impact function (HIF) or the concentration-response (C-R) function. As shown in Figure 1-1, these HIFs are derived from epidemiology studies that relate pollutant concentrations with health outcomes. BenMAP-CE applies that relationship to the population experiencing the change in pollution exposure to calculate health impacts.

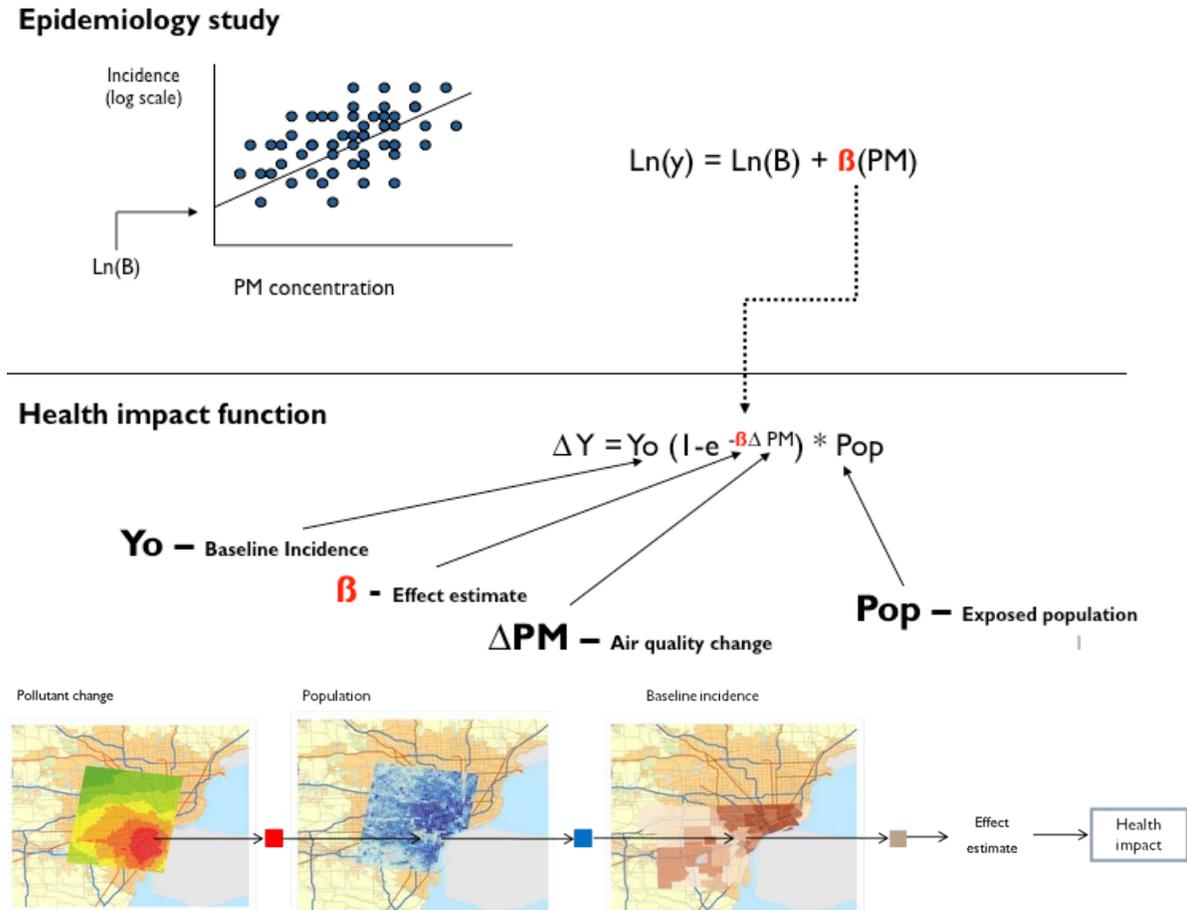


Figure 1-1. Deriving a Health Impact Function from the Epidemiology Literature

A simplified example is shown below.

Health Effect = Air Quality Change * Health Effect Estimate * Exposed Population *
Health Baseline Incidence

- **Air Quality Change.** The air quality change is the difference between the starting air pollution level (i.e., the baseline) and the air pollution level after some change, such as a new regulation (i.e., the control).
- **Health Effect Estimate.** The health effect estimate is an estimate of the percentage change in the risk of an adverse health effect due to a one unit change in ambient air pollution. Epidemiological studies are a good source for effect estimates.
- **Exposed Population.** The exposed population is the number of people affected by the air pollution reduction. The government Census office is a good source for this information. In addition, private companies may collect this information and offer it for sale.
- **Health Baseline Incidence.** The health incidence rate is an estimate of the average number of people who die (or suffer from some adverse health effect) in a given population over a given period of time. For example, the health incidence rate might be the probability that a person will die in a given year. Health incidence rates and other health data are typically collected by the government. In addition the World Health Organization is a good source for this.²

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

BenMAP-CE also calculates the economic value of avoided health effects (see Chapter 7 for details). After calculating the health changes, you can estimate the economic value by multiplying the reduction of the health effect by an estimate of the economic value per case (see Figure 1-2):

$$\text{Economic Value} = \text{Health Effect} * \text{Value of Health Effect}$$

² The World Health Organization is a good source for international health data, see: <http://www.who.int>.

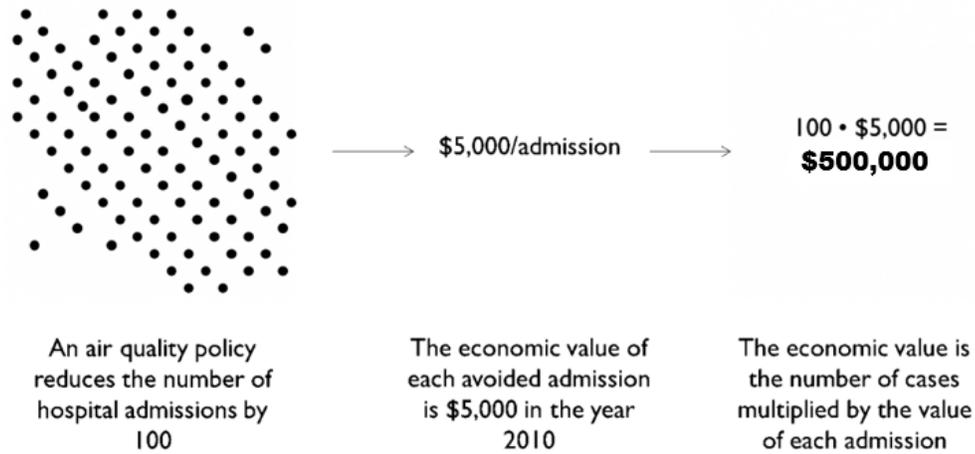


Figure 1-2. Estimating the Economic Value of Human Health Effects

There are several different ways of calculating the value of the health effect. For example, the value of an avoided premature mortality is generally calculated using the Value of Statistical Life (VSL). The value of a statistical life is the 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 medical costs of the illness may be the only valuation data available. The BenMAP-CE 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 effects³.

Figure 1-3 summarizes the BenMAP-CE inputs and outputs. This figure shows the types of choices that you make regarding the modeling of population exposure, the types of health effects to model, and how to place an economic value on these health effects. Please note that BenMAP-CE does not have air quality modeling capabilities, and therefore the user must provide externally created data in order to work with modeled data. BenMAP-CE is preloaded with limited air quality monitoring data, but externally created monitoring data may also be needed.

What else can BenMAP-CE do?

BenMAP-CE incorporates a geographic information system (GIS), allowing users to create, utilize, visualize, and export maps of air pollution, population, incidence rates, incidence rate changes, economic valuations, and other types of data (Figure 1-4).

³ See <https://www.epa.gov/benmap/benmap-community-edition>.

BenMAP Analysis: Inputs & Outputs

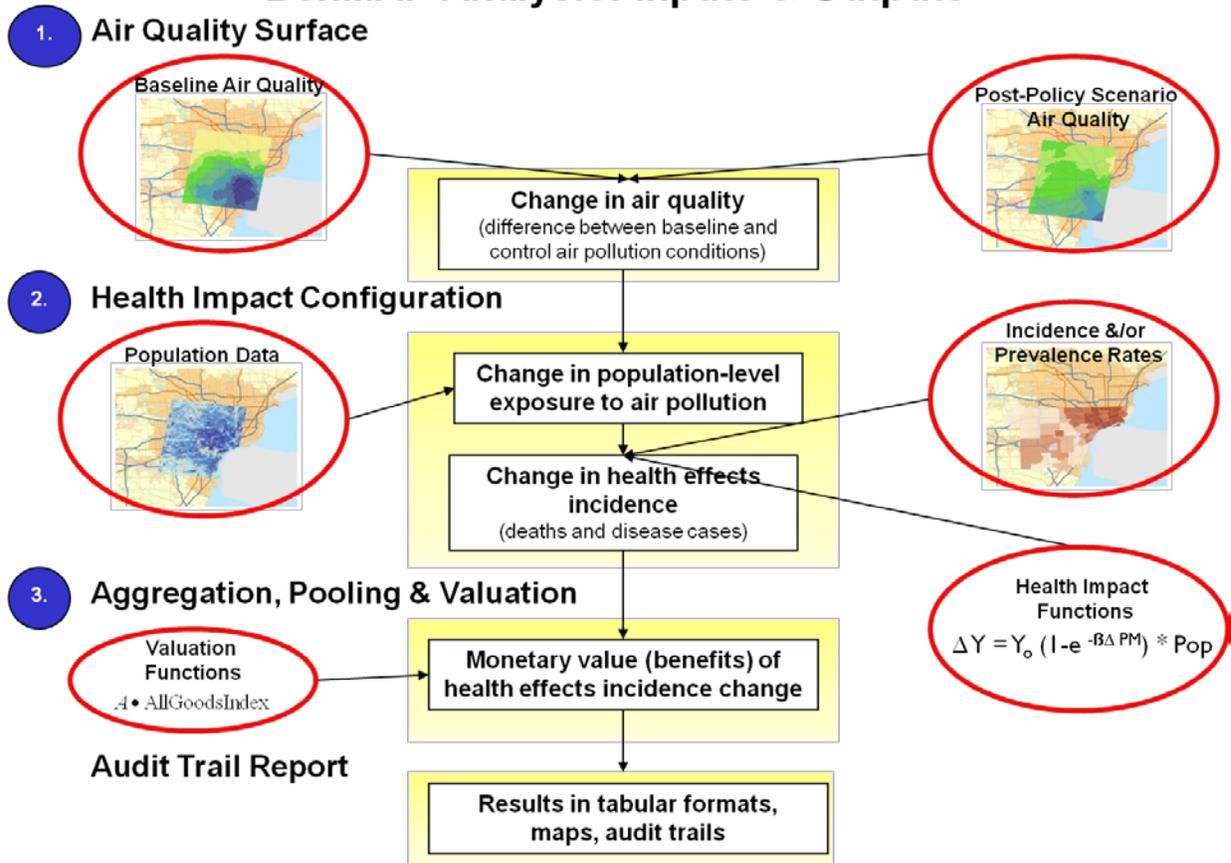


Figure 1-3. BenMAP-CE Flow Diagram

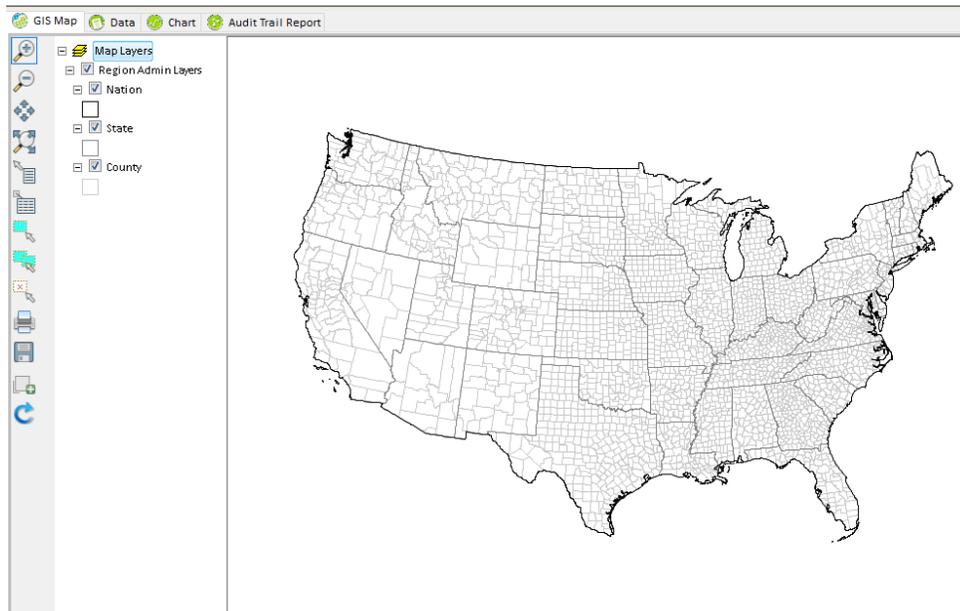


Figure 1-4. BenMAP-CE GIS Example

Analysts can use BenMAP-CE to:

- Create maps illustrating the population/community level ambient pollution levels;
- Compare benefits associated with various regulatory programs;
- Characterize the distribution of health impacts among population sub-groups;
- 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-CE 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 health impacts 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?

1.2 How to Use this Manual

Chapters 2 through 9 of this manual provide step-by-step instructions on how to use BenMAP-CE. New users should start with Chapters 2 and 3, which are both relatively short. These chapters provide a basic overview of the tool and how it works, and explain some potentially confusing terminology.⁴ Use the rest of the manual to answer any specific questions you may have, or to walk you step-by-step through the various components. Chapter 4 discusses how to enter data into BenMAP-CE, Chapters 5 through 7 cover each of the main steps in the Core Program, and Chapters 8 and 9 cover mapping, report options, and additional tools.

Each chapter is introduced by a short section that describes what you can find within the chapter and provides an outline of the chapter's contents. This is a good place to go if the Table of Contents does not provide enough detail for you to find the section you need. The end of most chapters has a series of "Frequently Asked Questions," which may also be helpful for answering specific questions. In chapters that provide instructions on navigating the tool, the following conventions are observed: tree menu items, buttons, tabs and selection box labels are in bold type; prompts and messages are enclosed in quotation marks; and drop-down menu items, options to click or check, and items that need to be filled in or selected by the user are italicized. Throughout the chapters you will also see boxes presenting common mistakes and important things to remember when working with BenMAP-CE.

⁴ Another good reference is the BenMAP-CE Quick Start Guide, see: <http://www.epa.gov/benmap>.

There is also a set of Technical Appendices to provide more detailed information on model functions, data, and underlying assumptions.

Appendix A: Monitor Rollback Algorithms

Appendix B: Air Pollution Exposure Estimation Algorithms

Appendix C: Deriving Health Impact Functions

Appendix D: Health Incidence & Prevalence Data in U.S. Setup

Appendix E: Core Particulate Matter Health Impact Functions in U.S. Setup

Appendix F: Core Ozone Health Impact Functions in U.S. Setup

Appendix G: Additional Health Impact Functions in U.S. Setup

Appendix H: Core Health Valuation Functions in U.S. Setup

Appendix I: Additional Health Valuation Functions in U.S. Setup

Appendix J: Population & Other Data in U.S. Setup

Appendix K: Uncertainty & Pooling

Appendix L: Command Line BenMAP-CE

Appendix M: Function Editor

References

1.3 Computer Requirements

The computer hardware requirements for BenMAP-CE are typically modest, though this will vary depending on the complexity of the analysis. BenMAP-CE requires a Windows platform and can be used on machines running Windows 7, Windows 8 or Windows 10. In particular, BenMAP-CE requires a computer with:

- Either a 64- or 32-bit operating system, although a 64-bit operating system is recommended
- Adobe Acrobat Reader
- Microsoft Excel or other spreadsheet program (in order to read exported .xlsx files)⁵
- Microsoft .NET Framework 4 (or 4.5)⁶

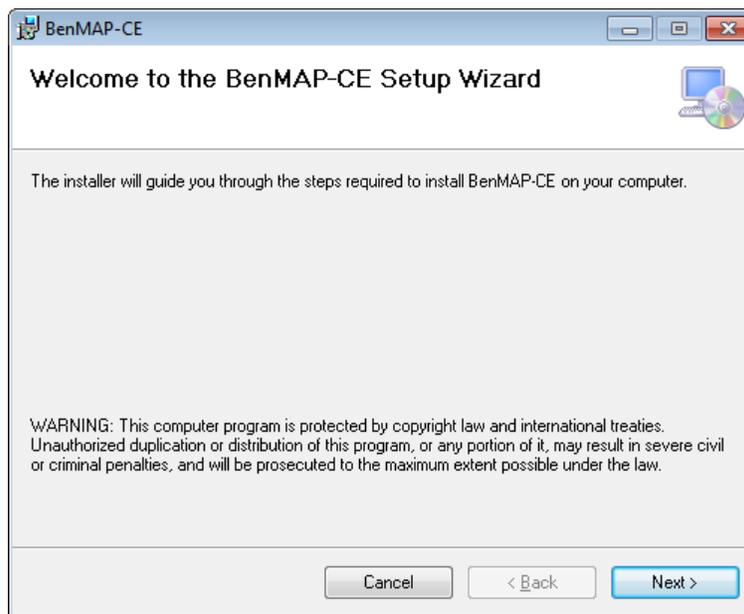
⁵ OpenOffice and LibreOffice are two open-source options for spreadsheet tools.

⁶ If .NET is not pre-installed, BenMAP-CE will provide a message advising you to install .NET. A standalone installer is available on the Microsoft website (URL: <http://www.microsoft.com/en-us/download/details.aspx?id=17718>). Install the runtime version and associated files (e.g., dotnetfx40_full_x86_x64.exe).

- At least 4 gigabytes of RAM⁷
- Intel or compatible processor, Core i5 (or better)
- At least 10 GB free disk space is necessary for installation of the BenMAP-CE database and ancillary files⁸.

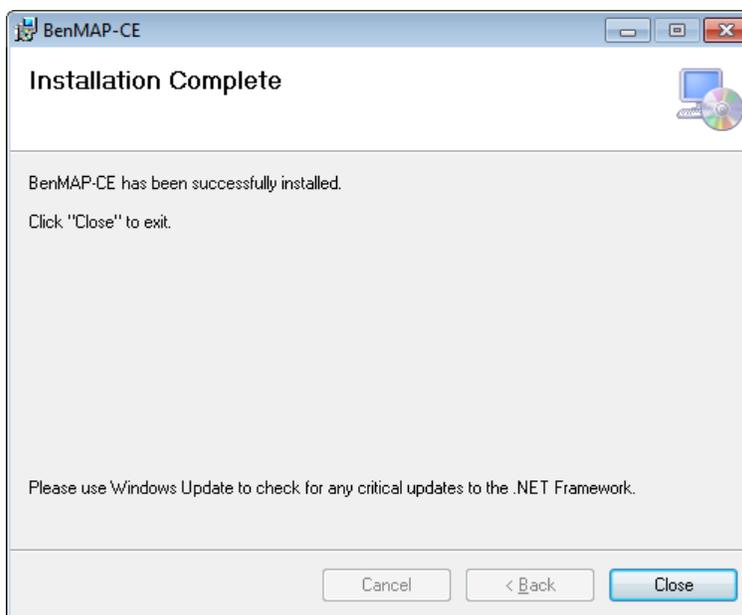
1.4 Installing BenMAP-CE

The installation of BenMAP-CE is very simple. Double click **Setup.exe** in your installation directory to bring up the setup wizard. Then follow the setup wizard by clicking 'Next' or 'OK' to complete the installation.



⁷ BenMAP-CE works best in a 64-bit Windows environment. With a 32-bit installation there are limits on the memory available to the software application; it can utilize no more than 2 GB of RAM. This will impact performance when processing large spatial datasets or numerous health impact/valuation functions. To determine whether your computer is running a 32-bit or 64-bit version of Windows, refer to this article from Microsoft: <https://support.microsoft.com/en-us/help/15056/windows-7-32-64-bit-faq>

⁸ A solid state drive (SSD) has also shown improved performance over hard disk drives (HDD).



Upgrading to a new version of BenMAP-CE

Periodically, new versions of BenMAP-CE will be made available and posted to the BenMAP-CE website: <http://www.epa.gov/benmap/>. If you are upgrading, first uninstall the previous version of the software on your computer (details provided in Section 1.5). Then extract the files from the installer package (.zip file) and run the executable ("setup.exe"). A new installer package can be relatively large (~1.2 gigabytes) because the database is embedded in the installer. However, once downloaded, the installation process is generally very fast.

Installation instructions are typically provided with each software release. Refer to these supplemental instructions for important additional information. Please note that your existing Setups will not be automatically transferred to newly installed versions of BenMAP. See instructions for exporting and importing databases in Chapter 9.

1.5 Uninstalling BenMAP-CE

To uninstall BenMAP-CE, go to **Control Panel, Programs and Features** and remove **BenMAP-CE**. Note that uninstalling BenMAP-CE does not also remove any results files that you have created with BenMAP-CE.

1.6 Contacts for Comments, Questions & Bug Reporting

For comments and questions, please contact Ali Kamal at the U.S. EPA.

Address: C539-07, U.S. EPA Mailroom, Research Triangle Park, NC 27711

Email: kamal.ali@epa.gov

Telephone: 919-541-4959

Alternatively, you can send a message at the BenMAP-CE website:

<https://www.epa.gov/benmap/forms/contact-us-about-benmap>, or by simply emailing benmap@epa.gov.

To report programming bugs or suggest additions to the software in BenMAP-CE:

- Select the **Help** menu in the main window;
- Open the **Provide Feedback** form;
- Complete the form and submit the report.

Provide Feedback

BenMAP-CE is open-source software. If you encounter an error or would like to request a feature, please consider submitting a report by filling out this form. We apologize for any issues you may have encountered and value your feedback as it will help us continue to improve BenMAP-CE.

Operating System: Microsoft Windows NT 6.2.9200.0

BenMAP-CE Version: 1.4.8.0 (June 12, 2018)

Your name (optional):

Your email address (optional):

Country where you are located (optional):

What type of report would you like to submit?

Software Error

Requested Feature

How severe is the error you experienced?

Minor - this issue has little or no impact on my ability to use BenMAP-CE

Major - this issue significantly hinders my ability to use BenMAP-CE

Blocking - this issue prevents me from using BenMAP-CE

What component of BenMAP-CE does the error affect?

Graphic User Interface: suggested changes

The audit trail report may help us to debug or understand your report better.

Include BenMAP-CE generated audit trail report.

Please describe what you were doing when you encountered the error. Can you tell us how to reproduce the error? (5000 character limit)

To submit this report to us, click the Submit button. Thank you for your time.

Submit Cancel

1.7 Sources for More Information

For supplemental information on BenMAP-CE, such as articles and presentations, manuals, and training materials go to:

- U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards (OAQPS), BenMAP-CE website: <https://www.epa.gov/benmap/>

To interact with other BenMAP users, go to:

- BenMAP-CE Discussion Forum: <https://forum.benmap.org/>

To access the BenMAP-CE source code, go to:

- BenMAP-CE on Github: <https://github.com/BenMAPCE/BenMAP-CE>

For more information on conducting benefit analysis, see the following documents:

- U.S. EPA (various years). Costs and Benefits of the Clean Air Act. Available at: <https://www.epa.gov/clean-air-act-overview/benefits-and-costs-clean-air-act>
- U.S. EPA (2006). Final Regulatory Impact Analysis: 2006 National Ambient Air Quality Standards for Particulate Matter. Office of Air Quality Planning and Standards. *See:* Chapter 5. Available at: <http://www.epa.gov/ttn/ecas/regdata/RIAs/Chapter%205--Benefits.pdf>
- U.S. EPA (2008). Final Ozone NAAQS Regulatory Impact Analysis. Office of Air Quality Planning and Standards. March. *See:* Chapter 6. Available at: <https://www3.epa.gov/ttn/ecas/regdata/RIAs/6-ozoneriachapter6.pdf>
- U.S. EPA (2008). Regulatory Impact Analysis: Control of Emissions of Air Pollution from Locomotive Engines and Marine Compression Ignition Engines Less than 30 Liters Per Cylinder. Office of Transportation and Air Quality. EPA420-R-08-001a. May. *See:* Chapter 6. Available at: <https://nepis.epa.gov/Exe/ZyPDF.cgi/P10024CN.PDF?Dockey=P10024CN.PDF>
- U.S. EPA (2010). Final Regulatory Impact Analysis (RIA) for the SO₂ National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards. June. *See:* Chapter 5. Available at: <http://www.epa.gov/ttn/ecas/regdata/RIAs/fso2ria100602ch5.pdf>
- U.S. EPA (2010). Final Regulatory Impact Analysis (RIA) for the NO₂ National Ambient Air Quality Standards (NAAQS). Office of Air Quality Planning and Standards. June. *See:* Chapter 4. Available at: https://www3.epa.gov/ttn/ecas/docs/ria/naaqs-no2_ria_final_2010-01.pdf

- U.S. EPA (2010). Guidelines for Preparing Economic Analyses. Office of the Administrator, National Center for Environmental Economics. EPA 240-R-10-001. December. Available at: [https://yosemite.epa.gov/ee/epa/erm.nsf/vwAN/EE-0568-50.pdf/\\$file/EE-0568-50.pdf](https://yosemite.epa.gov/ee/epa/erm.nsf/vwAN/EE-0568-50.pdf/$file/EE-0568-50.pdf)
- U.S. EPA (2011). Regulatory Impact Analysis (RIA) for the Final Transport Rule. Office of Air and Radiation. June. See: Chapter 5. Available at: <http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf>
- U.S. EPA (2011). Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards. Office of Air Quality Planning and Standards. December. See: Chapter 5. Available at: <http://www.epa.gov/ttnecas1/regdata/RIAs/matsriafinal.pdf>
- U.S. EPA (2012). Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter. See: Chapter 5. Available at: <http://www.epa.gov/ttnecas1/regdata/RIAs/finalria.pdf>

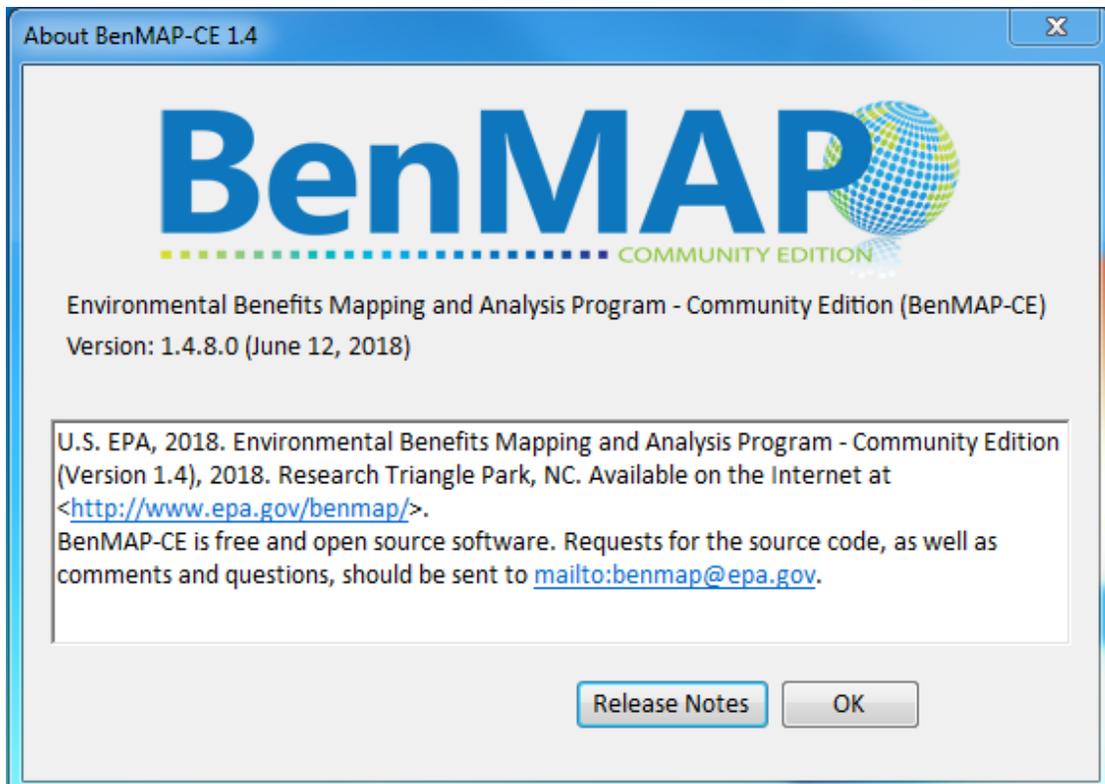
1.8 Frequently Asked Questions (General)

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

BenMAP-CE is free. There is no Terms of Use agreement and there are no restrictions on using BenMAP-CE. Feel free to share it with others.

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

You can identify the version of BenMAP-CE you are using by going to the Help menu and choosing **About**. Here you will see the version number and contact information. To determine whether you have the most recent version of BenMAP-CE, you can check the BenMAP-CE website (<http://www.epa.gov/benmap/>), which will have the latest version that is publicly available. Alternatively, you can use the contact information to inquire about any upcoming versions of the model.

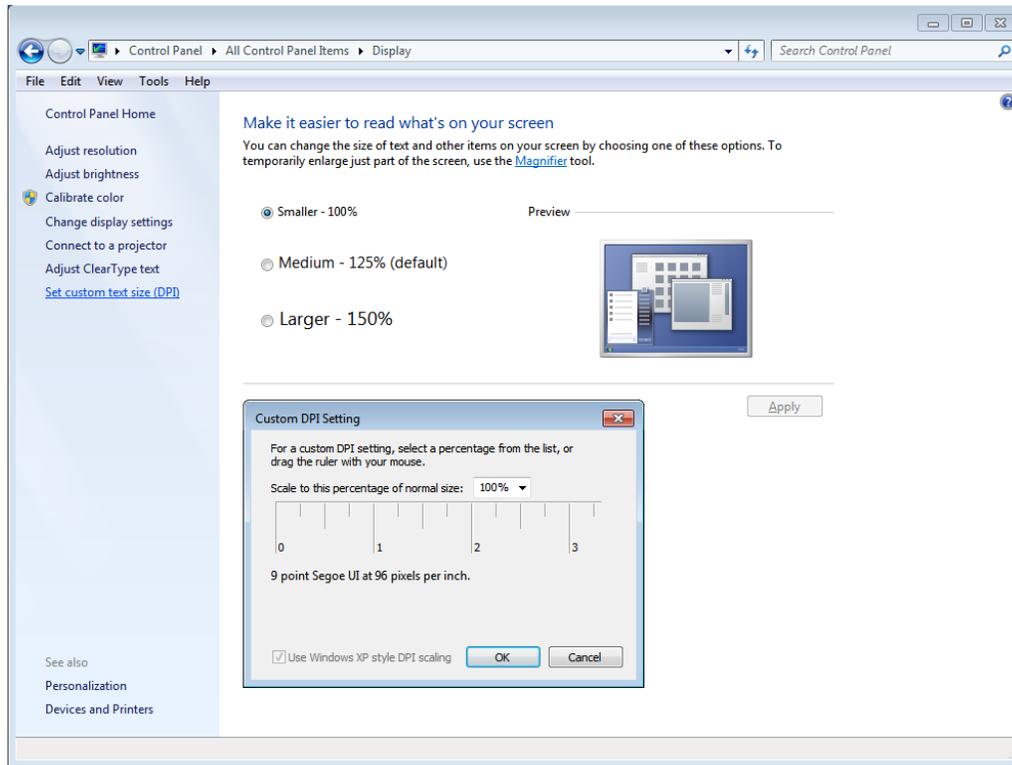


Why don't my files created with an older version of BenMAP work with BenMAP-CE?

Files created with an older version of BenMAP will not, in most cases, work with BenMAP-CE because of changes to the program. For example, later versions of BenMAP-CE have the capability to handle population data differentiated by ethnicity. For this reason, after completing an analysis with BenMAP-CE, it is always good to archive the BenMAP-CE installer along with the files used in your analysis, so that you will always be able to reproduce your work in the future.

Why are my pop-up windows too small? Why are buttons missing?

If the BenMAP-CE pop-up windows do not show the entire content (display seems cut off or buttons are missing), please check the display properties for your computer. Locate your **Control Panel**, then your **Display** settings, and choose the screen resolution associated with **96 DPI** (display pixels per inch). Here is what the screen looks like for Windows 7 Professional operating system.



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 the Audit Trail Reporting option. With the Audit Trail 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.

What do I need to be aware of if I use BenMAP-CE for a local scale analysis?

Perhaps the most important issue is to make sure that you have identified the resolution of your analysis and have the appropriate grid definitions loaded into BenMAP-CE. See Chapter 4 (Section 4.1.1) to read about grid definitions. The next key step, which is closely connected to the grid definitions, is to determine the data that you want to use. Data such as air quality modeling, incidence data, and population data need to match the grid definitions that you are using. You also need to be careful about the formatting of your data when loading it into BenMAP-CE. Chapter 4 also provides information on loading data into BenMAP-CE.

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

No. BenMAP-CE 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-CE 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.

How can I get training for BenMAP-CE?

A variety of training resources are available, including self-paced exercises, online interactive modules and instructor-led training are available at:

<https://www.epa.gov/benmap/benmap-ce-training-materials>

Where can I find the source code for BenMAP-CE?

BenMAP-CE 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/BenMAP-CE>.

Chapter 2

Terminology

In this chapter...

- **Find definitions for common terms used in the BenMAP-CE tool and in this manual.**

Active Layer. In the GIS window, the active layer is the top-most data layer. All queries or statistical analysis of the map will act upon this top-most layer.

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

Aggregation, Pooling, and Valuation (APV) Configuration. APV configurations store your preferences regarding how to aggregate your results, whether and how to pool your results, and any economic valuation functions you have applied. For example, an APV file might aggregate your estimated change in incidence to the U.S. county level, it might pool across multiple hospital admission health impact functions and it could include an economic valuation function. APV configurations are stored in files with an *.apvx* file extension. The results derived from an APV configuration have an *.apvrx* file extension. APV files are by default stored in the *APV* folder, and APV results files are by default stored in the *CFGR* folder.

Air Quality Surface. An air quality surface contains modeled or monitored air pollution data 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). These surfaces are also referred to as air quality grids. BenMAP-CE uses one air quality grid to represent the baseline scenario and a second grid to represent the control scenario. These baseline and control grids must share the same geographic structure. The program calculates the difference between baseline and control grids as an input to the health impact function. Air Quality Grids are stored in files with an *.aggx* file extension.

Air Quality Metric. The metric expresses 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 models are valuable air quality management tools. Models are mathematical descriptions of pollution transport, dispersion, and related physical and chemical processes in the atmosphere. Air quality models (like CMAQ⁹ and CAMx¹⁰) 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

⁹ Community Multi-scale Air Quality (CMAQ) Model is available at: <http://www.epa.gov/amad/Research/RIA/cmaq.html> or <https://www.cmascenter.org/cmaq/>.

¹⁰ Comprehensive Air Quality Model with Extensions (CAMx) is available at: <http://www.camx.com/>.

area where factors such as meteorology, topography, and emissions from both local and remote sources could be important. BenMAP-CE does not contain an air quality model.

Attainment. The state of meeting the National Ambient Air Quality Standard (NAAQS) for a pollutant. A geographical area that meets the NAAQS is called an "attainment area."

Audit Trail. This is a report that contains a record of all the choices involved in creating a particular file. Audit trails can be created for any file that BenMAP-CE creates.

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

Background Incidence. The incidence of a given adverse effect due to all causes including air pollution. Also called baseline incidence rates.

Baseline Scenario. The air quality levels prior to whatever policy change you are evaluating. The baseline is sometimes referred to as "Business as Usual." The baseline scenario is usually considered to be the reference scenario against which to compare a potential "control scenario", in which air quality levels are changed from the baseline levels.

Beta. The coefficient for the health impact function. The value of beta (β) typically represents the percent change in a given adverse health impact per unit of pollution.

Closest Monitor. The procedure by which data from the closest monitor is used to represent air pollutant levels in a population grid cell. BenMAP-CE can also scale the data from the closest monitor with air pollution modeling data. BenMAP-CE includes two types of scaling - "temporal" and "spatial". See "Scaling" for additional information.

Community Multi-scale Air Quality (CMAQ) Model. An open-source photochemical grid air quality model that the U.S. EPA and others rely upon to predict levels and changes in pollutant concentrations.

Concentration-Response (C-R) Function. A C-R function 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.

Configuration. A Configuration stores the health impact functions and model options used to estimate adverse health effects. Configurations are stored in files with a *.cfgx* file extension. CFGX files are by default stored in the *CFG* folder. The results derived from a Configuration have a *.cfgrx* file extension. CFGR files are by default stored in the *CFGR* folder.

Contingent Valuation. A survey-based economic technique for the valuation of non-market resources, such as environmental preservation or avoidance of air pollution health risk.

Control Scenario. In a modeling study, this is a sensitivity scenario in which emissions from one or more source sectors are changed (increased or decreased) from a given “baseline scenario”. The control scenario generally represents air quality levels after a new policy has been implemented.

Core BenMAP-CE. The fully-featured benefits analysis program that accepts user-defined air quality data, quantifies health impacts, aggregates, values and pools results (details available in Section 3.1). Non-Core BenMAP-CE features include Command Line and PopSim, described in the following Chapters and Appendices.

Cost of Illness (COI). The cost of illness includes the direct medical costs and lost earnings associated with illness. These estimates generally understate the true economic value of reductions in risk of a health effect, as they include just the direct expenditures related to treatment and lost earnings but not the value of avoided pain and suffering.

Currency Year. The value of the currency based on the year specified. Valuation estimates should use a consistent currency year to account for inflation. For example, you might want to report the valuation estimate in 2000 dollars to make it easier to compare with your cost analysis, which uses that same currency year.

Deltas. The difference between two data points. As used in BenMAP-CE, mapping the air quality deltas shows the change in air pollution between the baseline air quality grid and the control air quality grid.

Discount Rate. In a cost-benefit analysis, the discount rate is a quantitative method to account for the fact that people generally value future benefits and costs less than current costs and benefits. Typically, if a benefit occurs over multiple years, the economic benefit would be discounted.

Endpoint. An endpoint is a subset of an endpoint group, and represents a more specific class of adverse health effects. For example, within the endpoint group *Mortality*, there might be the endpoints *Mortality, Long Term, All Cause* and *Mortality, Long Term, Cardiopulmonary*.

Endpoint Group. An endpoint group represents a broad class of adverse health effects, such as premature mortality or hospital admissions. BenMAP-CE only allows pooling of adverse health effects to occur within a given endpoint group, as it generally does not make sense to sum the number of cases of disparate health effects, such as premature mortality and hospital admissions.

Epidemiology. The study of factors affecting the health and illness of populations. Epidemiological studies cannot prove that a specific risk factor actually causes the disease being studied but can only show that a risk factor is associated (correlated) with a higher incidence of disease in the population exposed to that risk factor.

FIPS Code. Federal Information Processing Standard codes. Each state in the United States is assigned a 2-digit code. For example, “01” refers to Alaska, “37” refers to North Carolina, and “56” refers to Wyoming.

Fixed Effect Pooling. Fixed effect pooling is one method to combine two or more distributions of health impact or economic value estimates into a single new distribution. Fixed effect pooling assumes that there is a single true underlying relationship between these component distributions, and that differences among estimated parameters are the result of sampling error. Weights for the pooling are generated via inverse variance weighting, thus giving more weight to the studies that exhibit lower variance and less weight to the input distributions with higher variance. See Random Effects Pooling below for additional information regarding pooling techniques.

Fixed Radius. An option to interpolate air quality data points that uses all monitors within a fixed radius (or distance) of a given point of interest. All monitors are used and weighted by their relative distance.

Geographic Area. Designation of a grid definition for linkage to a specific health impact function.

GIS. Geographic Information System. A GIS is a system of hardware and software used for storage, retrieval, mapping, and analysis of geographic data.

Global Burden of Disease. The World Health Organization global burden of disease (GBD) study measures burden of disease using the disability-adjusted-life-year (DALY). This time-based measure combines years of life lost due to premature mortality and years of life lost due to time lived in states of less than full health. The DALY metric was developed in the original GBD 1990 study to assess the burden of disease consistently across diseases, risk factors and regions.¹¹

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-CE Grid Definition provides a method of breaking a geographic region into areas of interest (Grid Cells) in conducting an analysis. This can be done in two ways - by loading a Shapefile (a particular type of GIS file) or by specifying a regularly shaped grid pattern. These are referred to as Shapefile Grid Definitions and Regular Grid Definitions, respectively. Typically a Shapefile Grid Definition is used when the areas of interest are political boundaries with irregularly shaped borders, while a Regular Grid Definition is used when the areas of interest are uniformly shaped grids (e.g., rectangles). All grid definitions must have a unique (i.e., non-repeating) column and row index.

¹¹ For more information on the GBD, see: http://www.who.int/topics/global_burden_of_disease.

Health Impact Function. A health impact function 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 has inputs specifying the air quality metric and pollutant; the age, race and ethnicity of the population affected; and the incidence rate of the adverse health effect.

Incidence. The total number of adverse health effects in a geographic region in a given unit time. In BenMAP-CE, this is the total number of adverse health effects avoided due to a change in air pollution levels.

Incidence Rate. The background rate of a health effect per person in a given geographic region. The average number of adverse health effects 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 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 willingness to pay (WTP).

Interpolation. The process of estimating the air quality level in an unmonitored area by using one or more nearby air quality monitors. BenMAP-CE uses two types of interpolation procedures: one is to simply choose the closest monitor, the other is to use a technique called Voronoi Neighbor Averaging. These interpolation methods are discussed in more detail in Appendix B.

Lat/Long. Latitude and longitude information to specify the geographic coordinates of a spatial location. The CMAQ model data are usually provided for each grid cell identified by the latitude and longitude of the grid cell's center point. Latitude identifies the north-to-south location of a point on the Earth. Longitude identifies the east to west location of a point on the Earth.

Layer. In GIS, a layer represents a logical separation of mapped data usually representing a theme, such as political boundaries, roads, ozone data, number of mortalities avoided, etc.

Layer Statistics. The summary statistics that correspond to the active layer in BenMAP-CE. For example, “mean”, “standard deviation” or “max” of PM_{2.5} air quality grid.

Metadata. Data that serves to provide context or additional information about other data. BenMAP-CE 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. For example, GIS metadata will include information about datum, geographic coordinate system, resolution, and units.

Micrograms per Cubic Meter ($\mu\text{g}/\text{m}^3$). The unit of measure for particulate matter in the NAAQS. This unit represents the mass of PM and other particle 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 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-CE we estimate monetized benefits by using either Willingness to Pay or Cost of Illness valuation functions (see above and below).

Monitor Data. Pollutant concentration data that are based upon measurements 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.

Monitoring. Actual measurements of air pollution concentrations. The U.S. EPA has 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-CE to quantify the confidence intervals around mean incidence and economic value estimate by randomly sampling an uncertainty distribution around the effect coefficients 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}, PM₁₀), and sulfur dioxide.

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-CE

Ordinality. In relation to air quality monitors, ordinality refers to the number of monitor values in the season that can exceed your standard. For example, if we had set the ordinality to four, then a monitor can have as many as three daily averages (assuming that we are using the daily average metric to define our standard) greater than your standard without violating the standard. In terms of rollback, if it has more than three daily averages in exceedance of the standard, then the rollback technique will be applied to that monitor.

Ozone (O₃). BenMAP-CE 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 (NO_x) 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 NO_x 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 a number of 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), PM₁₀ (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 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.

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

POC (Parameter Occurrence Code). An identifier used by U.S. EPA to distinguish between multiple monitors at the same site that are measuring the same parameter. For criteria pollutants, multiple monitors may be collocated to check precision. For combining data at the site level, the POC identifies the primary monitor (most frequent sampling). (POC appears in BenMAP-CE’s advanced filtering options for monitor data.)

Pooling. The combining of different sets of data. BenMAP-CE has several pooling methods, including fixed effects, fixed/random effects, and subjective weighting. Appendix K discusses the pooling approaches available in BenMAP-CE.

Point Mode. When defining the configuration, you may choose to either estimate adverse health effects in point mode or using percentiles. The point mode simply means that BenMAP-CE will use the mean value of the coefficient in the health impact function.

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-CE only represents population exposure.

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

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.

Random Effects Pooling. Random effect pooling is one method to combine two or more distributions of health impact or economic value estimates into a single new distribution. This approach allows the possibility that the estimated parameter from different studies may in fact be estimates of different parameters, rather than just different estimates of a single underlying parameter.

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

Relative Risk. Relative risk typically is used as 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, it 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.

Rollback. The process by which monitor data are reduced to a different level. BenMAP-CE rolls back monitor data in three ways. Percentage rollback reduces all monitor observations by the same percentage. Incremental rollback reduces all observations by the same increment. Rollback to a standard reduces monitor observations so that they just meet a specified standard.

Setup. A BenMAP-CE setup encapsulates all of the data needed to run analyses for a particular geographic area—a city, an entire country, etc. These data consist of grid definitions, pollutants, monitor data, incidence and prevalence rates, population data, health impact functions, variables, inflation rates, and valuation functions.

Shapefile. A shapefile is a particular type of GIS file, and 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.

Sum Dependent Pooling. Summing two or more incidence or valuation results, assuming the underlying functions are correlated. For example, summing the incidence

of respiratory hospital admissions for two different age groups quantified using C-R functions from the same study.

Sum Independent Pooling. Summing two or more incidence or valuation results, assuming the underlying functions are independent (uncorrelated). For example, summing the incidence of respiratory hospital admissions quantified using C-R functions from different studies using different methods.

Threshold. BenMAP-CE's advanced settings for health impact functions allows you to specify an air quality threshold; this is an air quality level below which benefits are not calculated. For example, if the threshold is 5 $\mu\text{g}/\text{m}^3$, then only areas with $\text{PM}_{2.5}$ concentrations equal to or greater than 5 $\mu\text{g}/\text{m}^3$ will be included in estimating health incidence results. Specifying a threshold does not affect the shape of the C-R function used to quantify impacts.

Unit Value. A unit value is the estimated mean economic value of avoiding a single case of a particular health effect.

User-defined Weights Pooling. User-defined weights let you specify the weights that you want to use when combining two or more distributions of results. The weights should sum to one. If not, BenMAP-CE normalizes the weights so that they do.

Valuation Function. Valuation functions are used by BenMAP-CE to estimate the economic values of changes in the incidence of health effects. These are selected within an Aggregation, Pooling, and Valuation Configuration (APV Configuration).

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

VNA (Voronoi Neighbor Averaging). An algorithm used by BenMAP-CE to interpolate air quality monitoring data to an unmonitored location. BenMAP-CE first identifies the set of monitors that best “surround” the center of the population grid cell, and then takes an inverse-distance weighted average of the monitoring values. This is discussed in detail in Appendix B.

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.

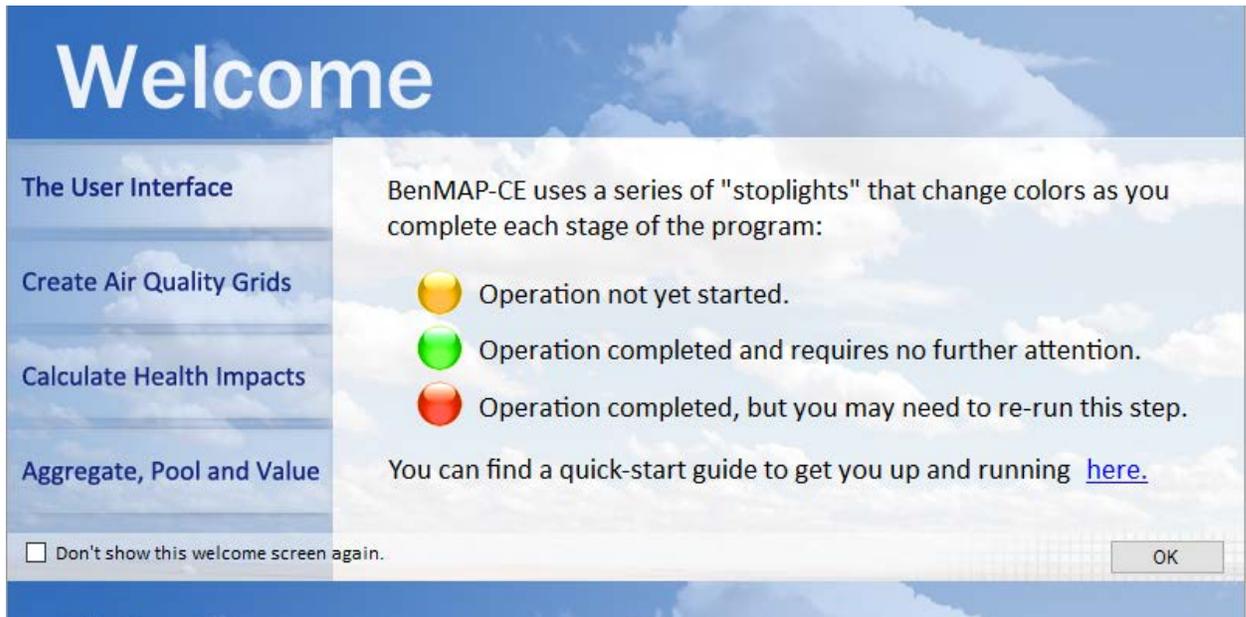
Chapter 3

Overview of BenMAP-CE Features

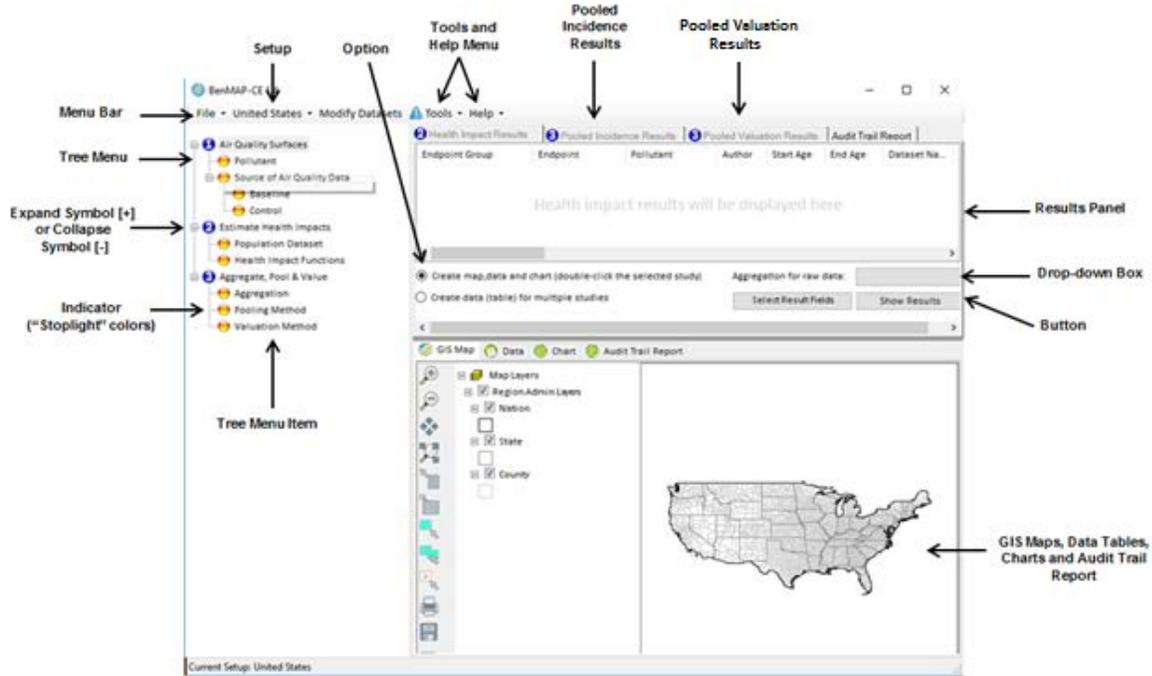
In this chapter...

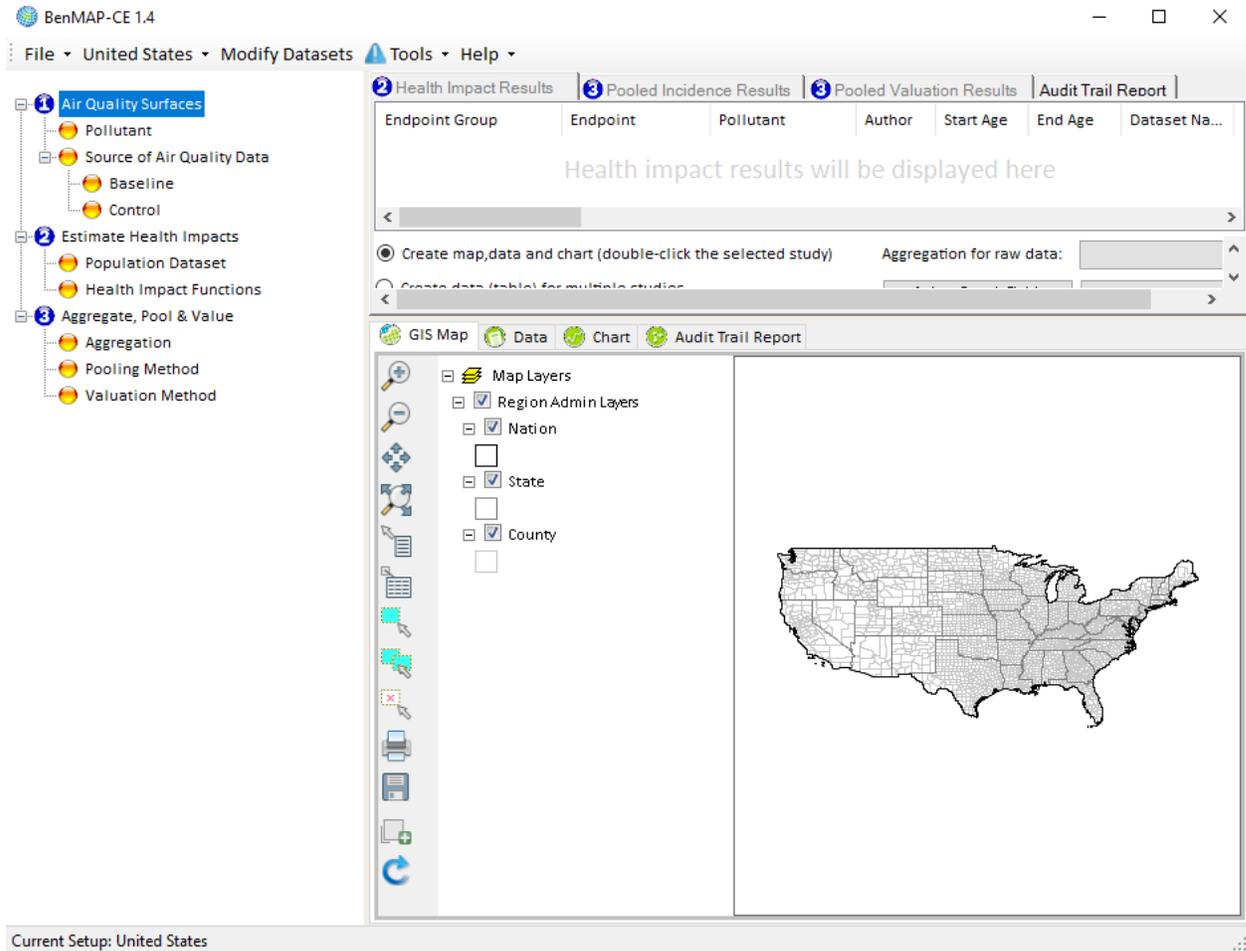
- **Get an overview of the features available with the Core Program.**
- **Learn about additional BenMAP-CE modules.**
- **Learn about the Tools and Help menu options.**
- **Find descriptions of the various outputs including types of files, results, maps and reports available from BenMAP-CE.**

Upon starting BenMAP-CE for the first time, you will see the following **Welcome** screen.



The **Welcome** screen gives a brief description of the user interface and highlights the “stoplight” metaphor used in BenMAP-CE to indicate the status of analytical steps performed using the tree menu on the left side of the main window. Clicking the links on the left side of the **Welcome** screen (e.g., **Create Air Quality Grids**) will provide information about each feature. You will also find a link to the BenMAP-CE website for downloading the most current BenMAP-CE software and other reference information. If you do not wish to see the **Welcome** screen at program start-up, check the option ‘*Don't show this window screen again*’ in the lower left-hand corner of the screen. To re-enable the **Welcome** screen, go to **Tools** menu on the main BenMAP-CE window, select **Options**, and check the option for *Show Start Window*. Press **OK** on the **Welcome** screen to close this window and display the BenMAP-CE main window.



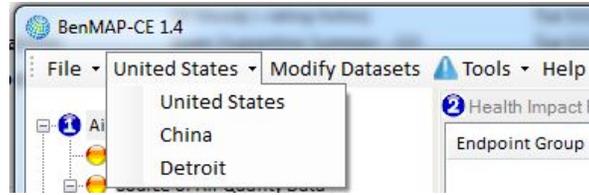


The tree menu on the left-hand window pane lists the analytical steps used in the Core Program. The tree menu items allow you to perform a highly customized health impact analysis. The **Tools** menu at the top of the screen is for less frequently used functions, such as importing and exporting data and special modules described in later sections.

The first section in this Chapter describes the Core Program features. The second section describes the additional functions found in the **Tools** and **Help** menus. The next section covers BenMAP-CE output options. Lastly, we answer some frequently asked questions. Note that this chapter provides an overview of functionality, not keystroke by keystroke instructions. Those detailed instructions may be found in Chapters 4 through 9.

3.1 Core Program

Beginning at the main BenMAP-CE window, you can choose the **Setup** you want to use for an analysis by selecting it from the **Setup** drop-down list. You are then ready to begin using the features available through the tree menu.



The tree menu takes you through the steps of an analysis. The first step, **Air Quality Surfaces**, allows you to select the pollutant of interest, and then specify the baseline and control air quality surfaces. The second step, **Estimate Health Impacts**, lets you choose the population dataset for a particular analysis, and then specify the health impact functions to estimate the incidence of adverse health effects. The last step, **Aggregate, Pool & Value**, gives you options for combining the health effects estimates and choosing economic valuation functions.

3.1.1 Create Air Quality Surfaces

BenMAP-CE is not an air quality model. Instead, it relies on externally-created air quality modeling data inputs. To estimate population exposure to air pollution, BenMAP-CE combines population data with air quality surfaces, which it generates using some combination of air quality modeling and/or monitoring data. In BenMAP-CE, air quality surfaces can be described as air quality grids (the structure) that have been populated with air pollution values (the data). The following is a brief description of each step. For detailed instructions, see Chapter 4: Loading Data and Chapter 5: Creating Air Quality Surfaces.

Pollutant

The **Pollutants** section of a setup specifies the pollutants that BenMAP-CE will analyze and defines the air quality metrics to be used by BenMAP-CE. You are not importing air pollution data, but rather naming your pollutants and defining the measures or metrics BenMAP-CE will use when assessing the health impacts associated with each pollutant.

Grid Definition

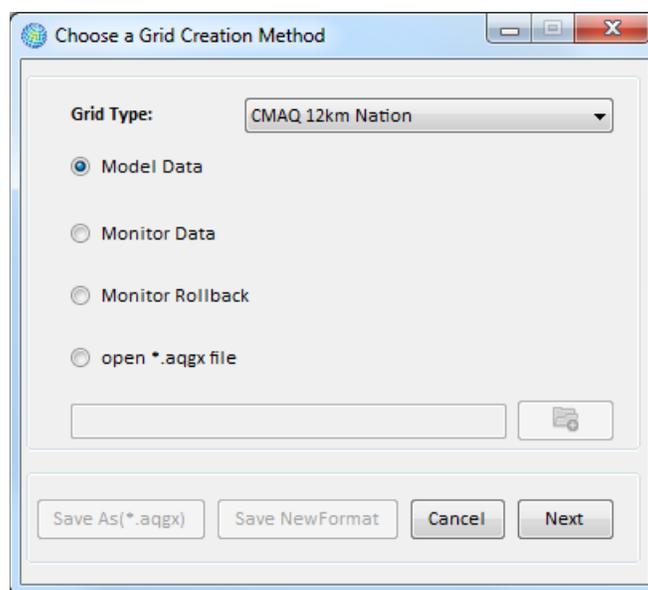
Air quality surfaces contain air pollution exposure estimates for a particular **Grid Definition**, as defined in the **Modify Datasets** window. **Grid Definitions** are typically comprised of either regularly shaped rectangles covering the region of analysis, or irregularly shaped polygons corresponding to political boundaries.

Modeling and Monitoring Data

To generate air quality grids, you can use air quality modeling data and air quality monitoring data in three different ways, as discussed below. However, once generated, all air quality grids have the same structure, and have the same *.aqgx extension that BenMAP-CE uses to designate these file types.

- **Model Data.** This procedure simply takes raw model data and converts it into a file that BenMAP-CE recognizes as an air quality grid. Defining the grid this way allows you to directly specify the air pollution values for each grid cell in a Grid Definition.
- **Monitor Data.** This procedure uses air pollution monitoring data to estimate air pollution levels for each grid cell in the selected Grid Definition. This may be done using one of three interpolation procedures - *Closest Monitor*, *Voronoi Neighborhood Averaging (VNA)*, or *Fixed Radius*. With closest monitor, BenMAP-CE simply uses the data of the monitor closest to each grid cell's centroid. With VNA, BenMAP-CE first identifies the set of monitors that most closely "surround" each grid cell, and then calculates an inverse-distance weighted average of the data from these neighboring monitors. With fixed radius, BenMAP-CE constrains the VNA interpolation to a user-specified distance around each monitor.
- **Monitor Rollback.** This procedure allows you to reduce, or roll back, monitor data using three methods: *Percentage Rollback*, *Incremental Rollback*, or *Rollback to a Standard*. Percentage rollback reduces all monitor observations by the same percentage. Incremental rollback reduces all observations by the same increment. Rollback to a standard reduces monitor observations so that they just meet a specified standard. After the monitor data is rolled back, it may be directly interpolated (as in *Monitor Data* grid creation) or combined with modeling data. This approach is described in more detail in Chapter 5: Creating Air Quality Grids, as well as in the Appendix A: Monitor Rollback Algorithms.

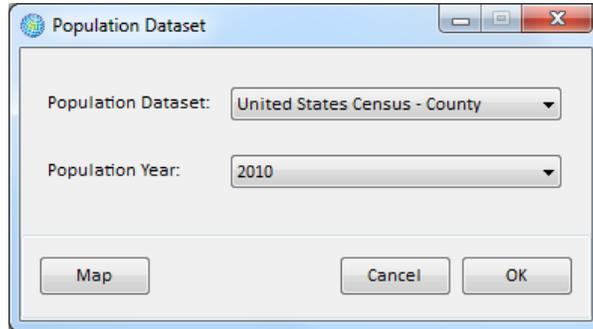
Once an air quality grid is created, it can be saved as a .aqgx file and reopened without any additional inputs needed.



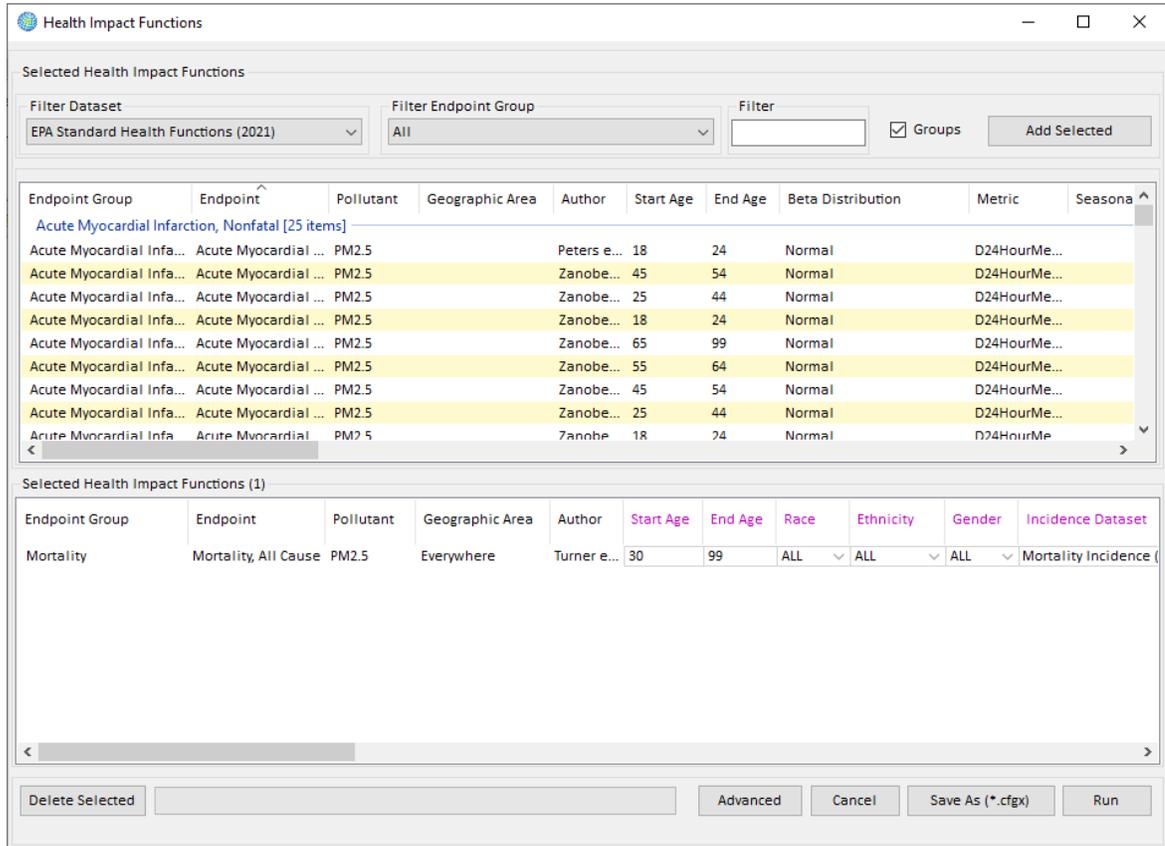
3.1.2 Estimate Health Impacts

The **Estimate Health Impacts** section allows you to calculate the change in the incidence of adverse health effects associated with changes in air quality. There are three steps in the process. The following is a brief description of each step. For detailed instructions, see Chapter 6: Estimating Incidence.

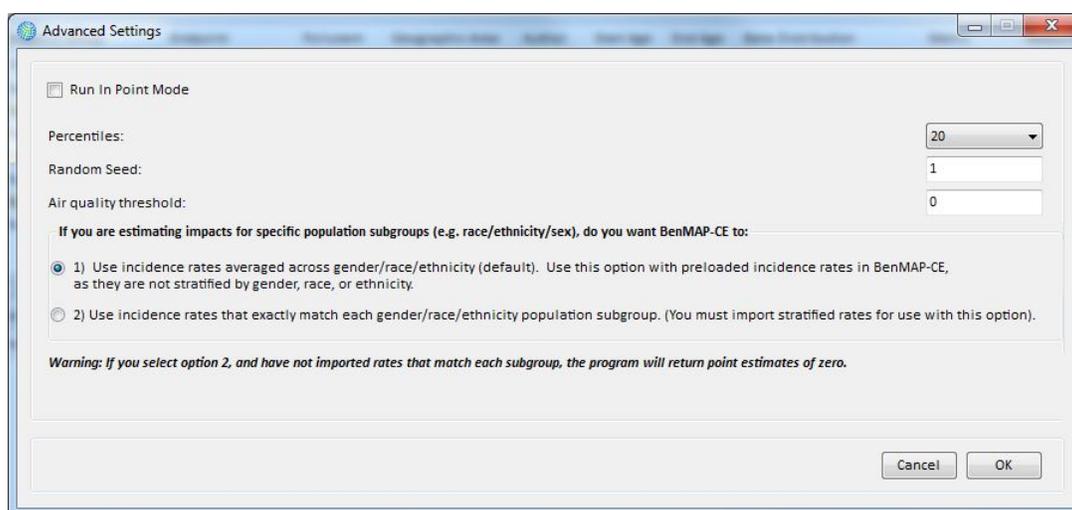
- **Step 1.** Specify the *Population Dataset* and *Population Year*.



- **Step 2.** Choose the *Health Impact Functions* that will be used to estimate the incidence of adverse health effects.



- Step 3.** BenMAP-CE performs a full Monte-Carlo analysis to quantify the confidence intervals around mean incidence and economic value estimates by randomly sampling an uncertainty distribution around the effect coefficients or willingness to pay estimates. This step typically occurs automatically using default parameters for the Monte-Carlo analysis. However, you may change these defaults in Advanced Settings, as shown in the screenshot below. In general, the computation time increases as you specify additional percentiles to report from the Monte-Carlo generated distribution. If you want to replicate the Monte-Carlo distribution from another analysis, then you may also specify the *Random Seed*.) Specify the *Air Quality Threshold*, or a lowest value for air quality data. Any observations which fall below this threshold will be replaced with the threshold value in all calculations.



BenMAP-CE can store configuration choices in a user-named file with a *.cfgx* extension, and can store incidence change estimates in a user-named file with a *.cfgrx* extension.

BenMAP-CE Decision Point

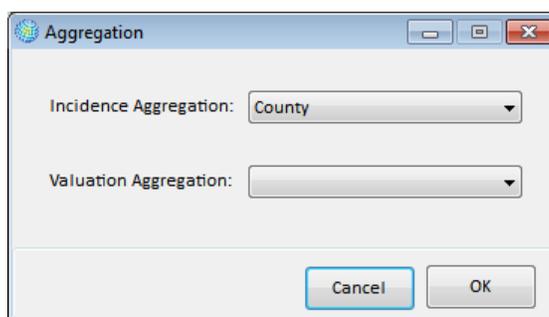
Once you generate a CFGRX file, BenMAP-CE will have quantified endpoint-specific incidence results, expressed as numbers of pollution-attributable cases, for each of the individual health impact functions you specified. At this point, you may:

- Export your results to complete your analysis. (Proceed to Section 3.3.1)
- Continue to the Aggregate, Pool, and Value part of the analysis (Section 3.1.3). Here you may:
 - Pool (i.e., combine) your incidence results for one or more health endpoints. Pooling is useful when you have multiple valid health impact functions for an endpoint that generate different results or have multiple subcategories of effects within a single endpoint that can be combined. (See Section 7.2.2)
 - Estimate the economic value of your incidence changes by applying health endpoint-specific valuation functions to those results. (See Section 7.2.3)

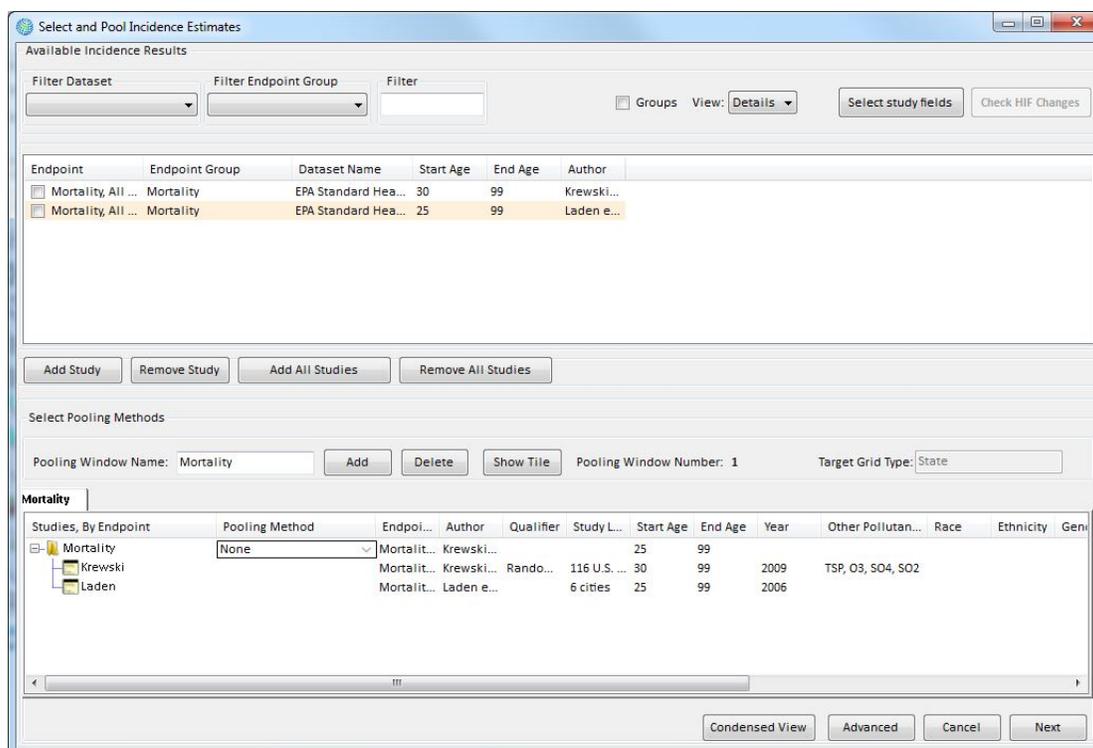
3.1.3 Aggregate, Pool, and Value

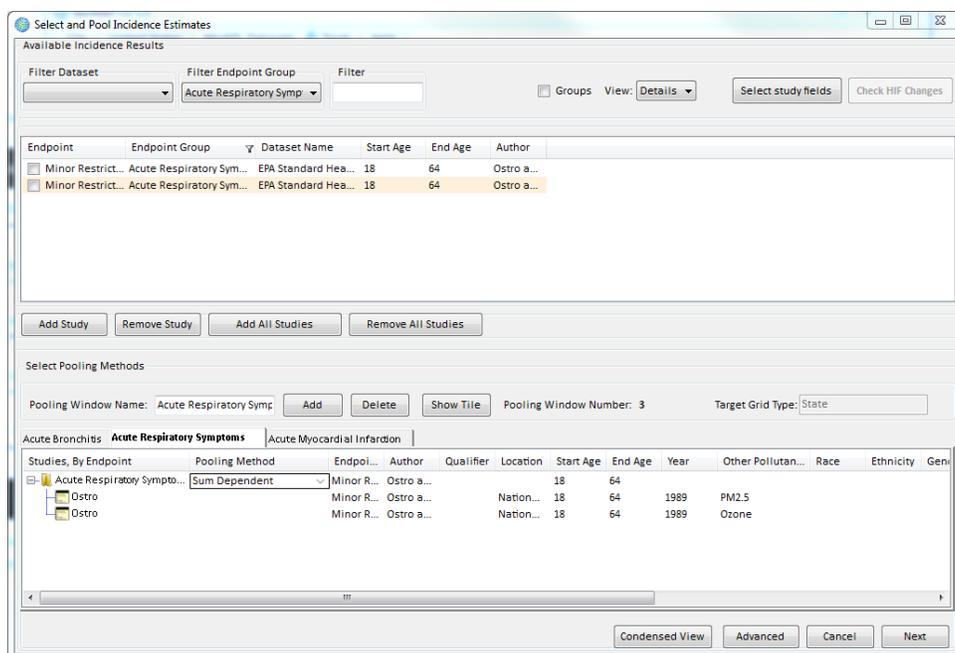
The **Aggregate, Pool, & Value (APV)** feature on the BenMAP-CE tree menu allows you to aggregate and pool previously calculated incidence estimates and place an economic value on these pooled and aggregated incidence estimates. You can also aggregate the economic values, and finally pool the aggregated economic values. There are several steps in this process. The following is a brief description of each step. For detailed instructions, see Chapter 7: Aggregating, Pooling, and Valuing.

- **Step 1.** Decide how to aggregate your results.

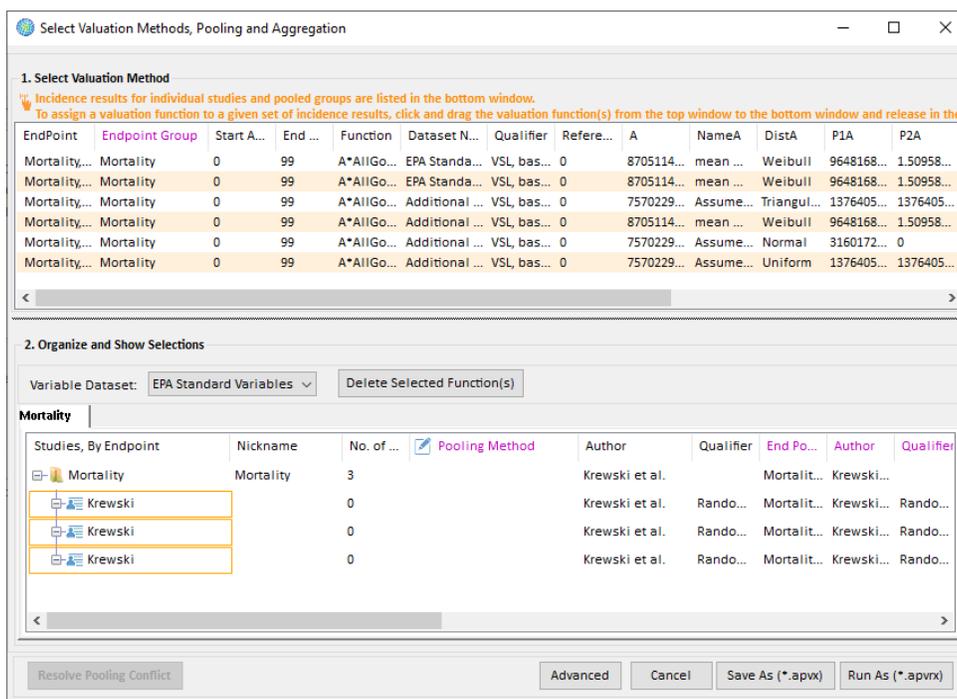


- **Step 2.** Decide how to pool and aggregate your incidence results.





- **Step 3.** Decide how you will estimate the economic value of your pooled and aggregated incidence results.



BenMAP-CE can store APV configuration choices in a user-named file with an *.apvx* extension, and can store APV configuration results in a user-named file with an *.apvrx* extension. You can access both files to use later.

3.2 Menus

There are five menu options found at the top of the main window: **File**, **Setup**, **Modify Datasets**, **Tools** and **Help**.

- **File.** This menu provides options for selecting saved project files (*.projx*) to open, starting a new project file, saving your work in a project file, and exiting the program.
- **Setup.** BenMAP-CE comes pre-loaded with datasets for the *United States*, *China*, and *Detroit* setups. The selected setup will be displayed in the menu bar, next to the File menu. You may click on the selected setup to see a menu of available setups. Each setup includes the information needed to run analyses for a particular geographic area. To learn more about modifying setups, see Chapter 4: Loading Data.
- **Modify Datasets.** BenMAP-CE stores the information needed to run analyses for a particular geographic area, such as a city, region, or nation in a single dataset called a setup. Many users will never need to modify the preloaded setups. However, the **Modify Datasets** menu provides tools to add, modify (load additional datasets), or delete these setups if needed. This is discussed in detail in Chapter 4: Loading Data.
- **Tools.** This menu provides access to data import and export functionality in addition to a number of other features. An overview is provided below in Section 3.2.1.
- **Help.** This menu provides access to a *Quick Start Guide* (available on EPA's website), information *About* BenMAP-CE, and a form to *Provide Feedback* about software errors or requested features. An overview is provided below.

3.2.1 Tools Menu

The **Tools** menu has several options: *Aggregate Air Quality Surface*, *Database Export*, *Database Import*, *Online Database Export*, *Online Database Import*, *Export Air Quality Surface*, *GBD Rollback*, *Monitor Data Conversion*, *Neighbor File Creator*, *PopSim Options*, and *Compute Grid Crosswalks*. A brief description is given below, but further information can be found in Chapter 9: Tools Menu.

- *Aggregate Air Quality Surface.* Create a new air quality surface for a specified grid definition (e.g., County) from an existing air quality surface created with a different (and generally finer) grid definition (e.g., 12km CMAQ).
- *Database Export.* Export all or part of BenMAP-CE's internal database to a database (.bdbx) file or multiple csv or shapefiles which can later be used on another computer or by another user. Manually loading data into BenMAP-CE can be time and labor intensive, so this tool can be quite useful in sharing data with other users

or computers. This tool can be used to share all or part of an existing setup, or to backup Setups prior to upgrading BenMAP.

- *Database Import.* Import data created using the *Database Export* tool into a specified setup.
- *Online Database Export* (Feature currently disabled). Allows users to post/share data for BenMAP-CE via a cloud-based system.
- *Online Database Import* (Feature currently disabled). Allows users to download data shared by others for BenMAP-CE via a cloud-based system.
- *Export Air Quality Surface.* Generate a text file (.csv) with all of the data in air quality surface, including summary statistics such as mean, median, minimum, and maximum.
- *GBD Rollback Tool.* The *GBD Rollback* tool allows you to select a country, region, or group of countries and see the impact of lowering PM_{2.5} emissions based on the data from the 2010 GBD study¹². The outputs include the baseline and policy case PM_{2.5} concentrations as well as the population-weighted air quality change.
- *Monitor Data Conversion.* Create a monitor file in BenMAP-CE format using an external data file in a different format.
- *Neighbor File Creator.* This tool creates a text file (.txt) identifying "neighbor" monitors and associated interpolation weights for each grid cell in an air quality grid. Identifying the major contributors to air quality in a given cell can be helpful for understanding and troubleshooting BenMAP results, and for understanding how different geographic patterns of air quality changes can affect health benefits.
- *PopSim.* A dynamic population simulation that incorporates the cumulative effects of air pollution on different age groups over time.
- *Options.* Select options for start-up and exit screens, validation logs and default setups.
- *Compute Grid Crosswalks.* Remove all crosswalks for selected setups and re-create them. This tool is for creating or repairing crosswalks which were broken due to database error or force quitting of the application.

¹² Exposure Assessment for Estimation of the Global Burden of Disease Attributable to Outdoor Air Pollution. Michael Brauer, Markus Amann, Rick T. Burnett, Aaron Cohen, Frank Dentener, Majid Ezzati, Sarah B. Henderson, Michal Krzyzanowski, Randall V. Martin, Rita Van Dingenen, Aaron van Donkelaar, and George D. Thurston. *Environmental Science & Technology* **2012** 46 (2), 652-660

3.2.2 Help Menu

The **Help** menu has a few options to choose from: *Quick Start Guide link*, *User Documentation*, *About*, and a form to *Provide Feedback*.

- *Quick Start Guide.* A link will open a webpage to the U.S. EPA site for BenMAP-CE training materials. On the webpage, users can download a series of self-paced exercises for seven different regions of the world, including two exercises in Spanish and one in French. Each self-paced exercise will take you through the basic operations of BenMAP-CE data imports and decision-making.
- *User Documentation.* Opens a link to the U.S. EPA site with the BenMAP-CE user manual and appendices.
- *About.* Opens a window that displays information about the program (e.g., software version, contact information, and a suggested citation). You can click on **Release Notes** to read about software modifications and any known issues.
- *Provide Feedback.* This feature allows you to submit any problems that you may encounter while running BenMAP-CE or requested features to the BenMAP-CE development team. There are fields to provide your contact information (optional) and information about the error or requested feature. Your feedback will be logged into an issue tracking system for U.S. EPA to evaluate.

BenMAP-CE is open-source software. If you encounter an error or would like to request a feature, please consider submitting a report by filling out this form. We apologize for any issues you may have encountered and value your feedback as it will help us continue to improve BenMAP-CE.

Operating System: Microsoft Windows NT 6.1.7601 Service Pack 1
 BenMAP-CE Version: 1.3.5.12 (April 18, 2017)

Your name (optional):
 Your email address (optional):
 Country where you are located (optional):

What type of report would you like to submit?
 Software Error
 Requested Feature

How severe is the error you experienced?
 Minor - this issue has little or no impact on my ability to use BenMAP-CE
 Major - this issue significantly hinders my ability to use BenMAP-CE
 Blocking - this issue prevents me from using BenMAP-CE

What component of BenMAP-CE does the error affect?
 Graphic User Interface: suggested changes

The audit trail report may help us to debug or understand your report better.
 Include BenMAP-CE generated audit trail report.

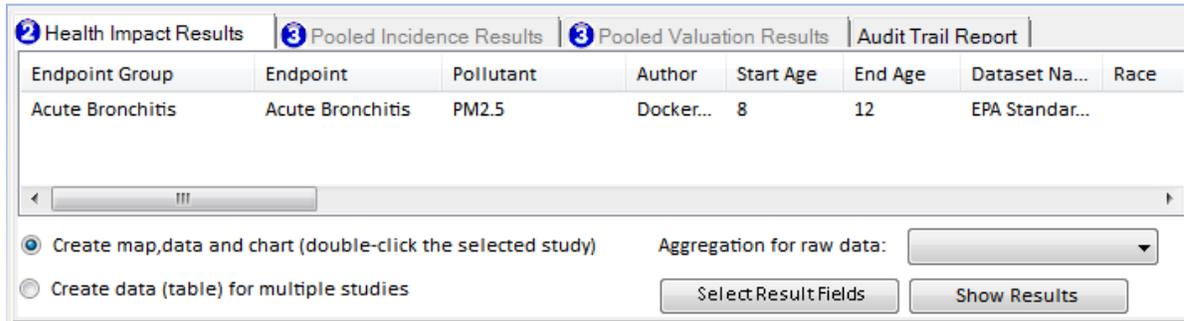
Please describe what you were doing when you encountered the error. Can you tell us how to reproduce the error? (5000 character limit)

To submit this report to us, click the Submit button. Thank you for your time.

3.3 Outputs

3.3.1 Results

If you are interested in viewing or exporting the results of an analysis, these reports can be accessed by clicking on the appropriate **Results** tabs from the upper portion of the main BenMAP-CE window. The number next to the tab description indicates the associated “step” of the BenMAP-CE analysis (from the tree menu).



Select the appropriate tab for the type of results you wish to create:

- **Health Impact Results** uses a *Configuration Results* file (with the *.cfgrx* extension) to create a map, data table and bar chart for incidence results based on the selected health impact studies. “Raw” incidence estimates are those that have not been aggregated, pooled or valued. See Chapter 6: Estimating Incidence, for detailed instructions for creating health impact results.
- **Pooled Incidence Results** uses an *Aggregation, Pooling, and Valuation Results* file (with the *.apvrx* extension) to create maps, data tables and bar charts for incidence, aggregated incidence and pooled incidence results. See Chapter 7: Aggregation, Pooling, and Valuation, for detailed instructions for creating pooled incidence results.
- **Pooled Valuation Results** also uses an *Aggregation, Pooling, and Valuation Results* file (with the *.apvrx* extension) to create maps, data tables and bar charts for valuation, aggregated valuation and pooled valuation. See Chapter 7: Aggregate, Pool, and Value, for detailed instructions for creating pooled incidence results.

The results data are viewable on the **Data** tab in the lower right frame of the BenMAP-CE main window (see below). You can also view the results in a **GIS Map** (described in the following section), or simple **Chart** format. All results can be exported as comma-separated value files (*.csv*), which can be read into spreadsheet and database programs.

Column	Row	D24HourMean	QuarterlyMean
1	29	11.95	11.95
1	31	10.69	10.69
1	37	11.66	11.66
1	39	10.22	10.22
1	41	10.91	10.91
1	45	10.90	10.90
1	49	12.01	12.01
1	53	10.12	10.12
1	57	11.12	11.12

3.3.2 Maps

GIS Maps can be viewed in the lower right frame of the BenMAP-CE main window. Once an air quality surface is displayed, you can choose which layers to view by selecting or deselecting items in the GIS table of contents. Spatial layers (except regional administrative layers) are semi-transparent so that overlapping layers are viewable. You can export a formatted graphics file (e.g., .png format) with the map legend and title. For detailed instructions on GIS maps, see Chapter 8: GIS/Mapping.

3.3.3 Audit Trail Report

The **Audit Trail Report** provides a summary of the options selected in the various parts of the analysis. You may generate an audit trail with any of the file types used in BenMAP-CE: Air Quality Grids (with the *.aqgx* extension), Configurations (with the *.cfgx* extension), Configuration Results (with the *.cfgrx* extension), Aggregation, Pooling, and Valuation Configurations (with the *.apvx* extension), and Aggregation, Pooling, and Valuation Configuration Results (with the *.apvrx* extension). The report itself has a tree structure. Below is an example of an Audit Trail Report in the default output format.

Name
BenMAP-CE 1.5.7
Current Audit Trail (Requested: 2020-12-14 13:43:07)
⊕ Runtime
⊕ Setup Name: United States
⊕ Air Quality Surfaces
⊕ Estimate Health Impacts
⊕ Aggregate, Pool & Value

Note that each successive step in an analysis contains a summary of its inputs and attributes, and those of each previous step in the analysis, laid out to follow the sequence of the inputs provided by the user. Below is an example of an **Audit Trail Report** when nodes have been expanded.



For example, in the above report the attributes of the *Health Impact Function* file used to generate the APV Results are present in the *Estimate Health Impacts* then *Selected Health Impact Functions* node. Similarly, the metadata for both the baseline and control air quality grids are present under the *Air Quality Surfaces* node.

The **Audit Trail Report** is now searchable. You may type a string into the Search text box at the bottom right of the **Audit Trail Report** and the program will highlight the location of that string within the report. Below is an example of the Search function.



For more information on audit trails, see Chapter 8: GIS/Mapping.

3.3.4 Dataset Validation Reports

You may load data to BenMAP-CE to tailor the analysis to your specific needs (click **Modify Datasets** from the main menu). Loading data requires specific formatting. BenMAP-CE offers a validation option to confirm that the proper headings and data types are present in the selected file. The validation routines also check that values are within reasonable ranges for certain types of data. If the file does not meet the validation requirements, error and/or warning messages will be reported. For more information about loading and validating data, see Chapter 4: Loading Data.

3.3.5 File Types

BenMAP-CE has a number of file types that you can use to store the settings used in a BenMAP-CE analysis, the results of an analysis, as well as maps and reports. Table 3-1 presents the names of the different file types, their functions, and their default folder locations.

Table 3-1. File Types Generated by BenMAP-CE

File Extension	Description	Default Folder Location¹
*.aqgx	Air quality grid.	Result\AQG
*.apvx	Aggregation, Pooling, and Valuation configuration specifying the aggregation levels, pooling options, and valuation methods used to generate aggregated incidence estimates, pooled incidence estimates, valuation estimates, aggregated valuation estimates, and pooled valuation estimates.	Result\APV
*.apvrx	Aggregation, Pooling, and Valuation configuration results, containing incidence results at the grid cell level, aggregated incidence results, valuation results, aggregated valuation results, and pooled valuation results.	Result\APVRX
*.bdbx	BenMAP-CE Database Export tool creates files which can contain individual datasets or entire setups. These are saved in a specific format for importing to BenMAP-CE.	User-specified
*.cfgx	Configuration specifying the health impact functions and other options used to generate incidence estimates.	Result\CFG
*.cfgrx	Configuration results, containing incidence results at the grid cell level.	Result\CFGR
*.csv	Reports (Results Tables) and PopSim tool results are exported as *.csv files, which may be viewed in a text editor, or in programs such as Excel. Users may also export portions of BenMAP setups (e.g., baseline incidence rates) as .csv files.	Result\CFGRX or Result\APVRX, PopSim
*.projx	Project files save a BenMAP configuration (not results) so that users can reload analysis parameters without having to repeat the analysis steps. The configurations for the Health Incidence and Aggregation, Pooling, and Valuation steps may be saved simultaneously in a single project file.	Result\Project
*.rtf	Validation results from data imports	ValidationResults
*.shp	Shape files generated by BenMAP-CE's geographic information maps system. These files can be viewed within BenMAP-CE or within shape file viewers, such as ArcView.	AppData\...\Shapefiles ²
*.xlsx	GBD Rollback Tool results are exported as .xlsx files, which may be viewed in a spreadsheet tool such as Excel (there is also a .csv option).	GBD

¹ Most files generated by BenMAP-CE are stored within the User's directory under C:\Users\\Documents\My BenMAP-CE Files\.

² Shape files (*.shp) are stored at

C:\Users\\AppData\Local\BenMAP-CE\Data\Shapefiles\\.

3.4 Frequently Asked Questions

When creating reports from *.apvrx files, why do some of the variables that I have checked appear as “Pooled” or as blanks?

When results are pooled, some of the identifying information for individual health impact functions is not carried forward. For example, when pooling incidence results from multiple health impact functions with different study locations, there is no longer a unique study location for the pooled result. So, BenMAP-CE would indicate the study location as “Pooled.”

How do I export my results?

Identify the type of report that you want to create, then refer to the Section 3.3.1 in this chapter on exporting reports.

How do I determine what the Column and Row refer to?

The Column and Row are variables designed to uniquely identify each grid cell in the grid definition. In the case of the County grid definition, the Column refers to the state FIPS code and the row refers to the county FIPS code. One way to get a good sense of the Column and Row variables is to create a map and then view where particular Column and Row variables occur in the map.

Chapter 4

Loading Data

In this chapter...

- **Learn how to create a new setup for your project.**
- **Learn more about the file structure for data inputs.**
- **Learn how to export and import a setup.**

BenMAP-CE can store the information needed to run analyses for a particular geographic area, such as a city, region, or nation, in a single dataset. This dataset is called a "Setup" and consists of 10 categories of data:

- Grid definitions
- Pollutants
- Monitor data
- Incidence and prevalence rates
- Population data
- Health impact functions
- Variable data (socioeconomic variables)
- Inflation rates
- Valuation functions
- Income growth data.

Grouping the data in this way has a number of advantages. It makes it easy to organize and view the data, export the data (either a whole setup or a portion of a setup), and import setups generated by others.

In this chapter we discuss how to add, modify, and delete a setup. It is important to keep in mind that if you delete one part of a setup, you may be affecting other parts of the setup. We discuss this further below.

Fundamental Concept - Setup

A BenMAP-CE **setup** refers to the set of data files that encapsulates all of the data that can be used in BenMAP analyses for a particular geographic area—a city, an entire country, etc. A setup **must** contain at minimum one or more grid definitions for geographic area to which data are assigned spatially (e.g., county polygons or 12 km grid cells); a pollutant definition specifying the exposure season and relevant exposure metrics (e.g., 8-hour max concentration); health incidence and population size data; and at least one health impact function for the defined pollutant in order to run a successful analysis. Setups may also include tables of air quality monitor data; population size and sociodemographic data; economic data; and valuation functions.

Once input into a given setup, these data are available for application in all subsequent BenMAP analyses that use that setup, facilitating future runs.

Modeled air quality data files are not considered part of the setup; they are input into the tool at the time of analysis.

Many users will never need to modify the setup. If you are performing an analysis

Decision Point

When do you need to add or modify a BenMAP-CE setup?

Setups are generally based on a specific geographic area. Hence, you will want to **add** a new setup to analyze a geographic area that is not pre-loaded with BenMAP-CE. Creating a new setup for each geographic area you are analyzing will help you organize the databases you import.

You will want to **modify** an existing setup if a setup already exists for the geographic area to be analyzed but you want to add or change one or more of the datasets (e.g., adding updated population data and new health impact functions to the pre-loaded *United States* setup).

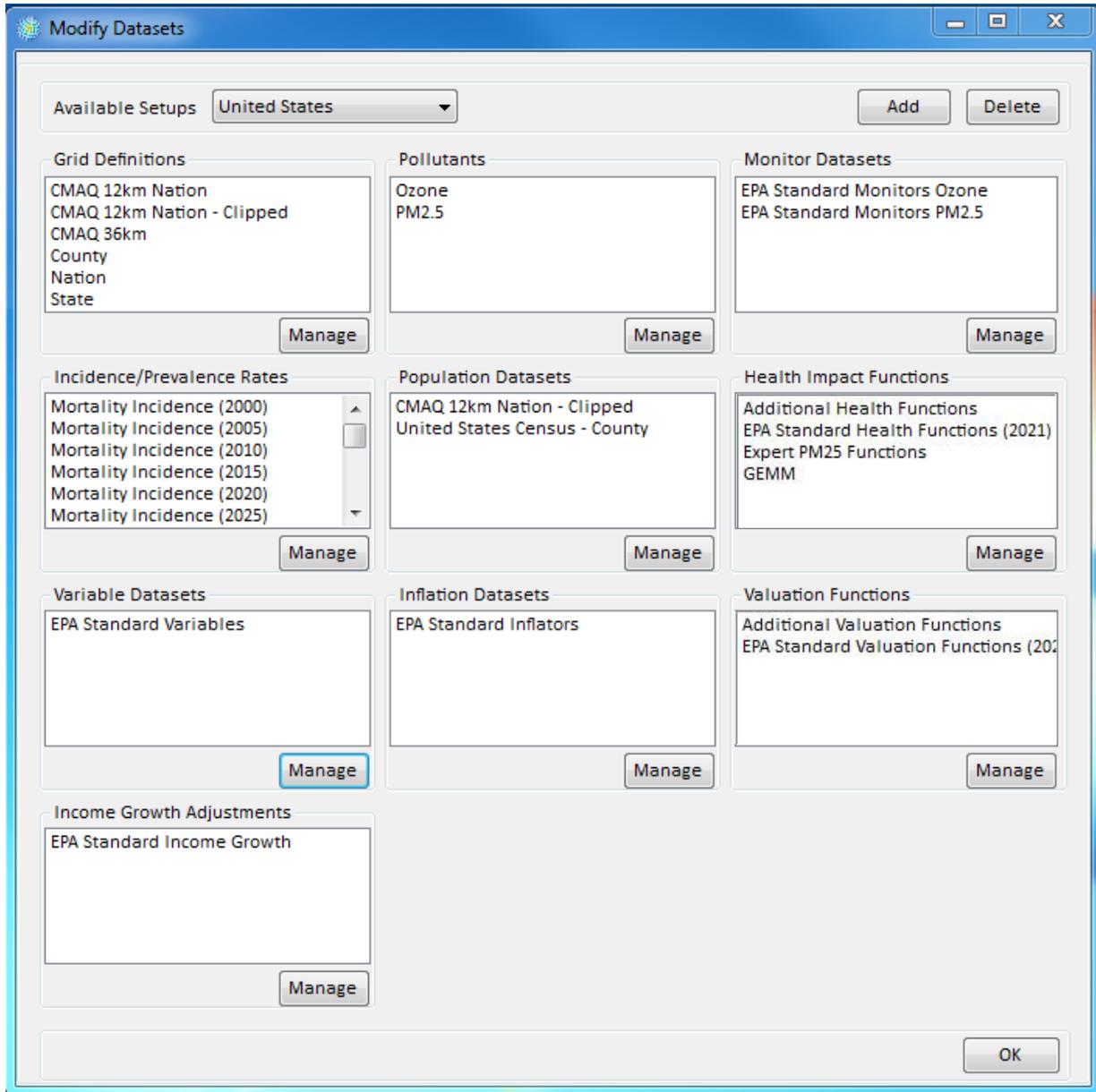
with the pre-loaded *United States, China, or Detroit* setups, you may find that BenMAP-CE contains all of the data you need to perform your analysis, and no additional modifications are necessary.

We will also discuss how to load (import) data into an existing setup. There are a number of steps involved in formatting and loading these data, so it is important to carefully review the steps in this chapter. For most dataset types, BenMAP-CE provides a validation tool to help you check your data file format (column names, required columns, and data types) before import. Validation reports are provided which describe

any errors or warnings with the associated row number and column name.¹³

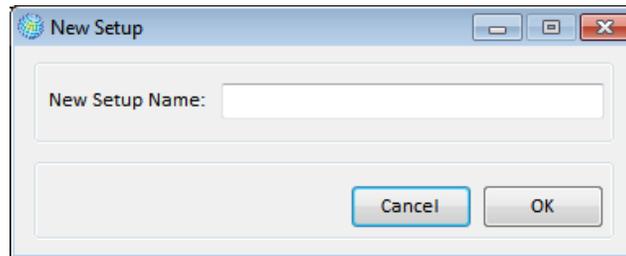
4.1 Add, Modify, and Delete a Setup

To add a new setup, modify an existing setup, or delete a setup, choose **Modify Datasets** from the menu bar. This will bring up the **Modify Datasets** window. The United States setup, which comes preinstalled with BenMAP-CE, includes a variety of datasets and looks like this:



¹³ Validation reports are saved to C:\Users\\Documents\My BenMAP-CE Files\ValidationResults.

Add a Setup. To add a setup (e.g., to add a new country that was not pre-loaded with BenMAP-CE), click the **Add** button. The **New Setup** window will appear where you can type a name for the new setup.



After naming the new setup, you can define the elements that comprise a setup. Table 4-1 lists the 10 dataset types within BenMAP-CE and indicates which types of data are needed to perform certain analyses.

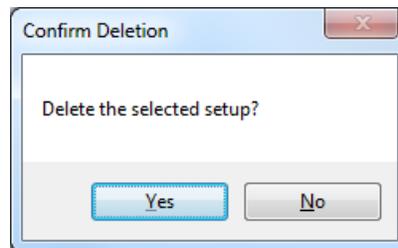
Table 4-1. BenMAP-CE Data Elements

Dataset Type	Required to Estimate Health Impacts	Required to Quantify Economic Values
Grid Definitions	✓	✓
Pollutants	✓	✓
Monitor Datasets (or Modeled Data)	✓	✓
Incidence/Prevalence Rates	✓	✓
Population Datasets	✓	✓
Health Impact Functions	✓	✓
Variable Datasets		
Inflation Datasets		✓
Valuation Functions		✓
Income Growth Adjustments		✓

Some of the elements of a setup are fundamental and should be entered before the others, namely, **Grid Definitions** and **Pollutants**. The **Incidence/Prevalence Rates**, **Population**, and **Variable Datasets** depend on the **Grid Definitions**, and the **Monitor** and **Health Impact Functions** datasets depend on the **Pollutants** that you have defined. Therefore, it is best to start by defining your **Grid Definitions** and **Pollutants**, and then define the other elements of the setup.

Modify a Setup. To modify a setup, click **Modify Datasets** on the menu bar. Choose the setup for modification from the drop-down list of **Available Setups** (the default value is *United States*). Then, click on the **Manage** button under one of the ten components comprising a setup. The sections below provide more information for each of these components.

Delete a Setup. To delete a setup, click **Modify Datasets** on the menu bar. Choose the setup for deletion from the drop-down list of **Available Setups**. Click the **Delete** button. You will then be asked to confirm your decision.



4.1.1 Grid Definitions

Fundamental Concept – Grid Definition

BenMAP-CE **Grid Definitions** help to spatially allocate data used in the benefits assessment. They enable users to conduct analyses with data sets at a variety of spatial scales and to aggregate and map results at different scales.

There are two types of grid definitions: **Shapefile Grid Definitions** and **Regular Grid Definitions**. Typically, a Shapefile Grid Definition is used when the areas of interest are political boundaries with irregularly shaped borders, while a Regular Grid Definition is used when the areas of interest are uniformly shaped grids (e.g., rectangles). All grid definitions must have a unique (i.e., non-repeating) column and row index.

A BenMAP-CE **Grid Definition** specifies geographic units (i.e., grid cells) that serve two purposes: (1) the program assigns air quality data, population data, baseline incidence rates, and health impact functions to these grids in order to calculate impacts; and (2) you can use these grids to report results in the GIS tool. You can define a grid in one of two ways: by loading a Shapefile (a particular type of GIS file) or by specifying a regularly shaped grid pattern. These are referred to as **Shapefile Grid Definitions** and **Regular Grid Definitions**, respectively. A **Regular Grid Definition** is used when you want to specify a grid that is regularly shaped (e.g., 12 x 12 km squares). A **Shapefile Grid Definition** can be used to create either grids that are regularly shaped or grids that match an irregular shape, like a political boundary. All

Shapefile Grid Definitions must contain an attribute table with a unique (i.e., non-repeating) column and row index. Column and row values should not exceed 1,000,000.

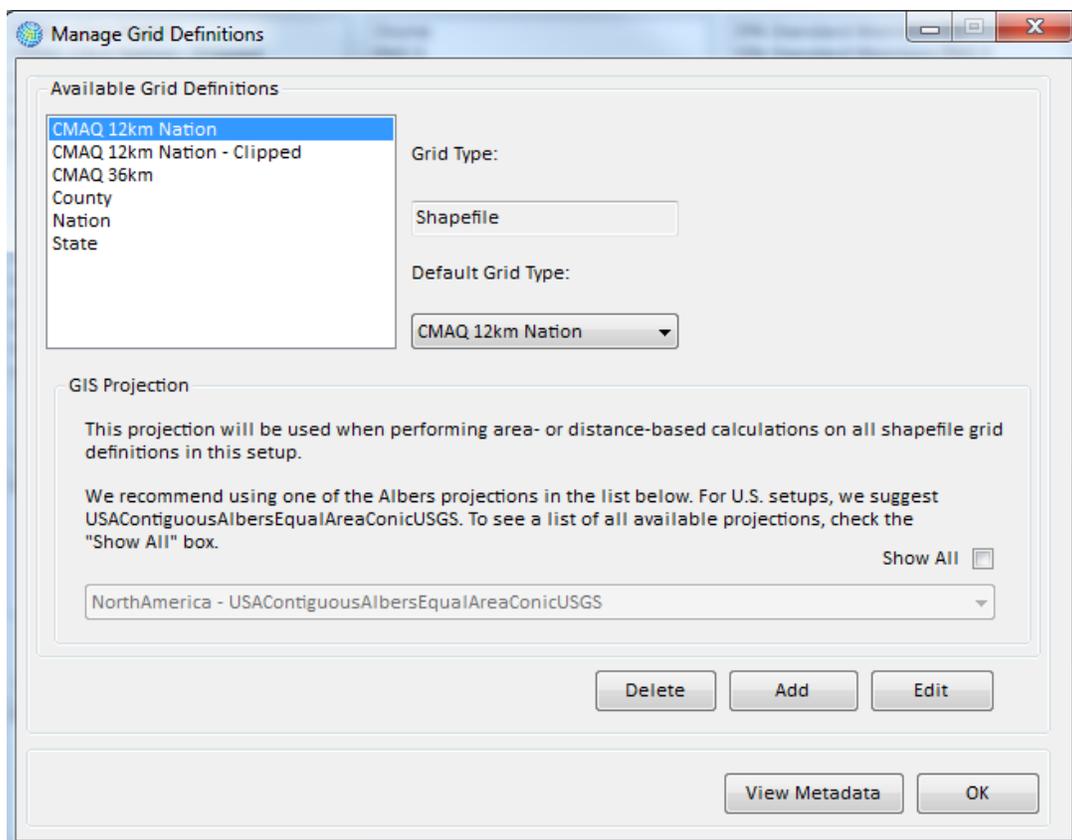
At least one **Grid Definition** should be created to outline the area of interest for the BenMAP-CE analysis (a city boundary, for example). Additional grid definitions can also be created for subdivisions of that area for which (a) data are available (see the Air Monitoring, Population, Incidence and Prevalence, and Variables sections below), or (b) reports or maps are desired.

For example, an analysis for the United States might use one or more of the following grid definitions:

- **Nation** – this Shapefile Grid Definition contains an outline of the United States (just the lower forty-eight states), defining an overall area of interest.

- **State** – this Shapefile Grid Definition contains state borders, for use in generating reports and maps with results aggregated to the state level.
- **County** – this Shapefile Grid Definition contains county borders, for use with county-based population and incidence rate data.
- **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.

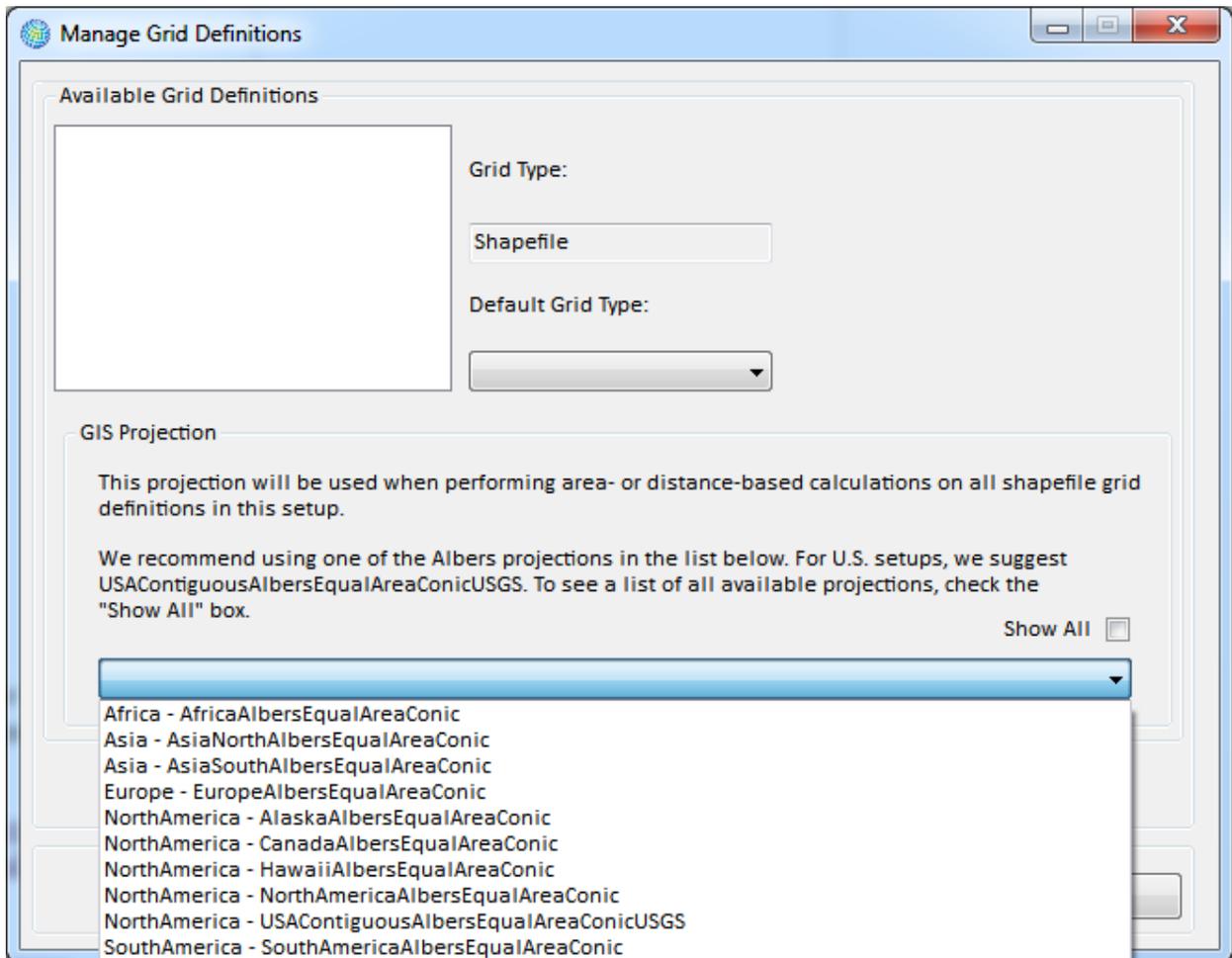
To start adding or modifying grid definitions, click **Modify Datasets** on the menu bar. Click on the **Manage** button below the **Grid Definitions** box. The **Manage Grid Definitions** window will appear.



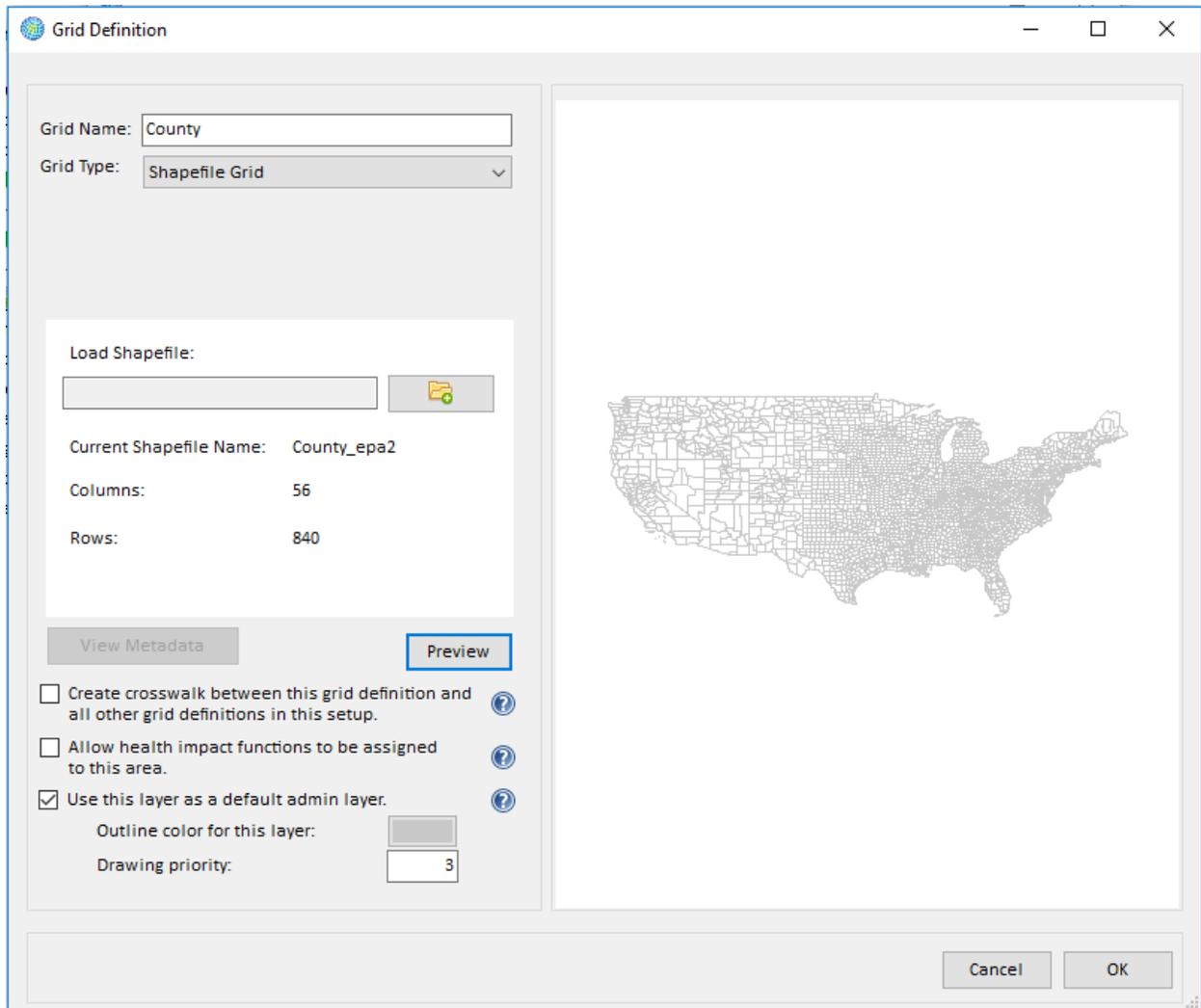
Click on the **Add** button to display the **Grid Definition** window. Provide a name for the Grid Definition in the **Grid ID** field and specify the **Grid Type**: *Shapefile Grid* or *Regular Grid*.

When you add a new Setup, you will be asked to select a GIS projection. The projection you specify will be used when performing area- or distance-based calculations on all shapefile grid definitions in this setup. We recommend selecting a regional setting that best represents the geographic area you are evaluating from one of the Albers

projections provided in the list box. (For U.S. setups, we suggest USAContiguousAlbersEqualAreaConicUSGS.)



If you have reason to choose another type of projection (e.g., in consultation with a geospatial analyst), you may check the “Show All” box and select from an expanded list of choices.



Starting with BenMAP-CE version 1.4, you can apply different health impact functions to different air quality grids within the same BenMAP run. This feature is designed to facilitate the application of region-specific air pollution effect coefficients (e.g., you can apply a Los Angeles-specific health impact function to that city and a national function to the remainder of the United States). If you plan to link specific health impact functions to a new grid definition, you must check the “Allow health impact functions to be assigned to this area” box in the lower left of the screen. Checking this box does not require you to assign a health impact function to this area, but enables this feature as an option.

Also added in version 1.4, you can now specify whether you want to use a grid definition as a default administrative (admin) layer; these layers are useful for showing political boundaries in GIS maps of BenMAP results. By checking the box “Use this layer as a default admin layer” you are telling BenMAP to draw this layer on the GIS results map by default. You may select multiple admin layers, such as a country boundary and state or province boundaries, and you can turn individual administrative layers on or off manually at any point using the GIS tool.

4.1.1.1 Regular Grid

Regular Grid Definitions are defined by a lower left corner (specified as decimal degree latitude and longitude, with West and South having negative values and East and North having positive values), a total number of columns and rows, a number of columns per degree longitude, and a number of rows per degree latitude. Individual cells within the resultant grid are numbered in sequential order (columns from left to right, rows from bottom to top) starting at (1, 1). These field values will be used to link the **Regular Grid Definition** with other sources of data, as discussed in more detail below.

To define a **Regular Grid**, start by selecting *Regular Grid* from the **Grid Type** drop-down menu. Type the name of the grid definition in the **Grid ID** box, and then define the number of **Columns** and **Rows** in the grid. To locate this grid geographically, provide the decimal degree coordinates for the lower left-hand corner of the grid in the **Minimum Longitude** and **Minimum Latitude** boxes.

To give the overall geographic size of the grid, provide the number of **Columns Per Longitude** and **Rows Per Latitude**. For example, if you specify 16 columns and 2 columns per degree longitude, then the grid will span 8 degrees of longitude. And if you specify 25 rows and 4 rows per degree latitude, then the grid will span 6.25 degrees of latitude.

Combining the numbers in this example, if the minimum longitude and latitude are -81 and 38 and the grid spans 8 degrees longitude and 6 degrees latitude, then the grid will run between -81 and -73 degrees longitude and between 38 and 44.25 degrees latitude.

After defining the grid, click the **Preview** button to see what the grid looks like. You may change the parameters and click the **Preview** again to see how the grid changes. When you are satisfied with the grid definition, click the **OK** button.

Grid Definition

Grid Name: CMAQ 12km Nation

Grid Type: Regular Grid

Columns: 16

Rows: 25

Minimum Longitude: -61

Minimum Latitude: 30

Columns Per Longitude: 12

Rows Per Latitude: 4

View Metadata

Preview

Create crosswalk between this grid definition and all other grid definitions in this setup.

Allow health impact functions to be assigned to this area.

Use this layer as a default admin layer.

Outline color for this layer:

Drawing priority: 0

Cancel OK

Fundamental Concept - Crosswalks

BenMAP-CE calculates **crosswalks** between different grid definitions to relate data at one spatial scale to another. For the default grid definitions in the U.S. BenMAP Setup, EPA developed population-weighted crosswalk factors between geographic elements using census tract-level population data. In all other cases, BenMAP-CE calculates a crosswalk allocation factor based on the percent overlap in area between a geographic element of one grid definition and every element of another grid. This allows BenMAP-CE, for example, to use population at the county level with air quality at the 12km CMAQ grid level in a health impact analysis. Crosswalks also allow BenMAP-CE to aggregate or disaggregate results to various geographic resolutions.

To calculate health impacts and economic benefits, BenMAP-CE uses air quality, population, and demographic data at different spatial scales. To do this, the program calculates a crosswalk (percentage file) that relates data at one spatial scale to another (e.g., 12km CMAQ grid to county). This step is performed only once per crosswalk and the results are saved to the database for subsequent calculations.

If you wish to pre-calculate the crosswalk (percentage overlap) between this grid definition and all other grid definitions in this setup, check the option box. This will take longer to load the data now, but can save you time later when you are ready to calculate health impacts. If you do not create the crosswalks at the grid definition stage, BenMAP-CE

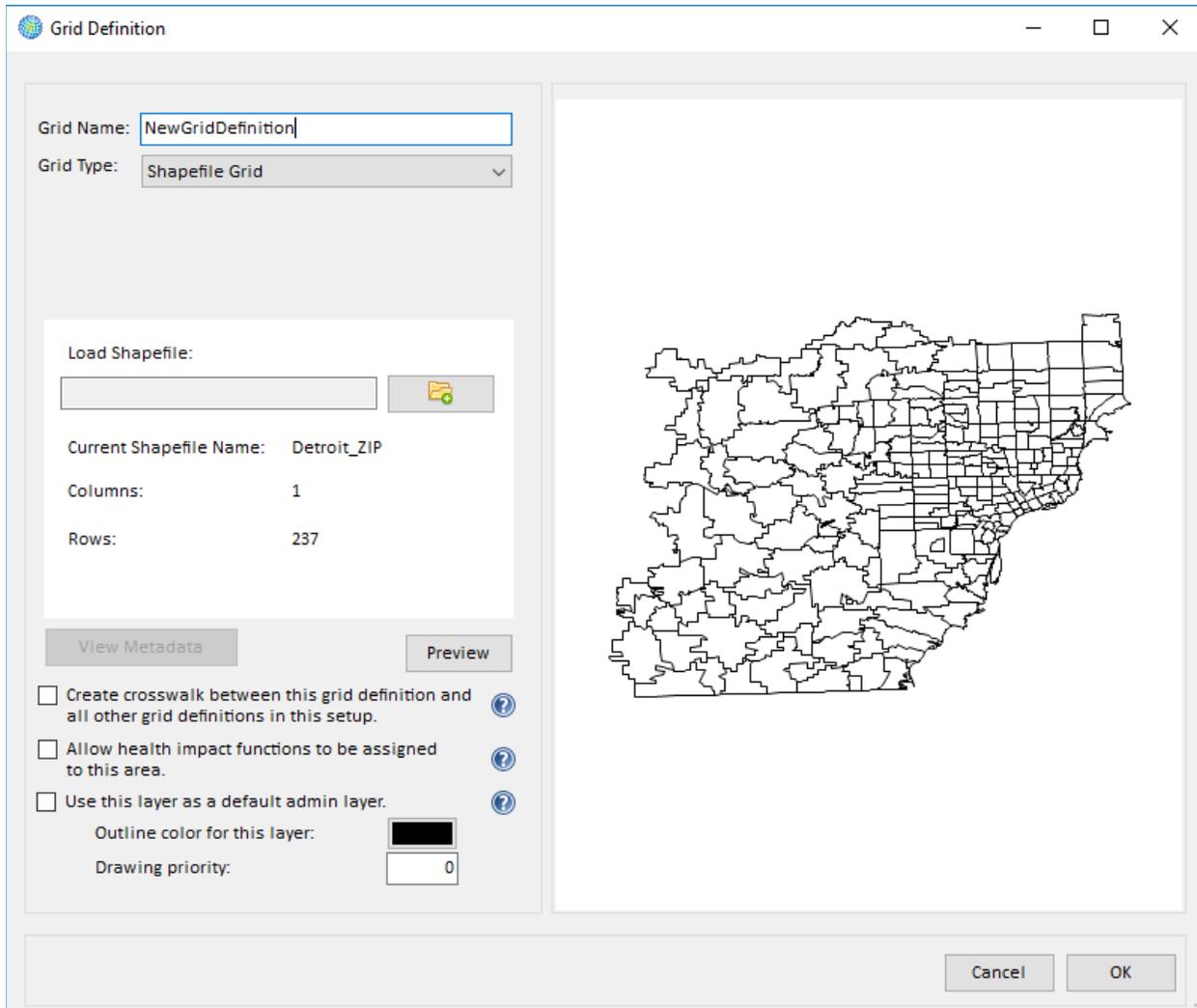
will create crosswalks as needed during the configuration or aggregation, pooling, and valuing stages.

The name of your newly defined grid will then appear in the **Manage Grid Definitions** window. You may click **Edit** to change the grid definition, **Delete** to permanently remove the grid that you just defined, or **Add** to define a new grid definition. **View Metadata** is not applicable for **Regular Grid Definitions**. Click **OK** to return to the **Modify Datasets** window.

4.1.1.2 Shapefile Grid

Shapefiles used to create **Shapefile Grid Definitions** should be of the ESRI Shapefile format. Details on this format can be found at <https://www.esri.com/library/whitepapers/pdfs/shapefile.pdf>. When a shapefile is being used to create a new grid, BenMAP-CE will: (1) check to see if the file is projected to GCS NAD 83; and (2) if necessary, re-project the file (with notification) from the native projection to GCS NAD 83. Any shapefiles used must contain integer fields named Column (or Col) and Row, and each shape within the shapefile must contain a unique combination of values for these two fields. These column and row values are used, just as the Column and Row field values in Regular Grid Definitions, to link the Shapefile Grid Definition with other sources of data, as discussed in more detail below.

To add a **Shapefile Grid**, click on the **Add** button in the **Manage Grid Definitions** window, choose *Shapefile Grid* from the **Grid Type** dropdown menu, name the grid in the **Grid ID**, and browse for the correct shapefile by clicking on the small open-file icon just to the right of the **Load Shapefile** input box. After locating the file, click **Open**. This will choose the file, and bring you back to the **Grid Definition** window. To view the shapefile, click **Preview**. You can add metadata using the **View Metadata** button. Adding metadata allows you to supply a file **Reference** (e.g., person, organization, publication, or model that produced or supplied the values in the electronic file) and **Description** (or any relevant notes about the use or limitations of the data). Other minimal file attributes are pre-populated automatically during import.



When you are satisfied that the shapefile looks correct and have checked the appropriate boxes on the lower left, click **OK**. This will save the shapefile and bring you back to **Manage Grid Definitions** window.¹⁴ Note that the **Grid Type** box displays the type of grid for each of your grid definitions. The **View Metadata** button will allow you to see and edit any comments that were previously entered.

Click **OK** when you are finished loading grid definitions. The **Modify Datasets** screen will now list the **Grid Definitions** that you have just created. At any time, you may click the **Manage** button to add, modify, or delete grid definitions.

Warning

If you delete a **Grid Definition**, you will also permanently delete any gridded data (e.g., **Population** or **Incidence**) that is dependent on it.

¹⁴ Shapefiles are saved to C:\Users\\AppData\Local\BenMAP-CE\Data\Shapefiles\\.

WARNING! If you delete a **Grid Definition**, you will permanently delete any gridded data that is dependent on it, such as any **Incidence/Prevalence, Population, and Variable Datasets** that use this particular **Grid Definition**. As we discuss each of these other setup elements below, we will describe how this might happen.

4.1.2 Pollutants

The **Pollutants** section of a setup specifies the pollutants that BenMAP-CE will analyze and defines the air quality metrics to be used by BenMAP-CE. You are not importing air pollution data, but rather naming your pollutants and defining the measures or metrics BenMAP-CE will use when performing an analysis for each pollutant. You may include any pollutant, though typically air pollutants such as particulate matter, ozone, sulfur dioxide, and carbon monoxide are used in a BenMAP-CE analysis.

A key concept for pollutants is the **Metric**. Air quality metric describes the period of the day over which the pollutant observations are averaged. For example, a metric of *D24HourMean* is a daily average of hourly measurements. A metric of *D8HourMax* is the average of the 8-hour period during the day when pollutant levels are the highest (see Table 4-2 below). The air quality change must be expressed in a metric that matches the metric used by the health impact function; this concept is discussed further below.

Fundamental Concept – Pollutant Metric

The **metric** expresses the time period over which air quality values are modeled or observed and the type of statistic being used (e.g., average, maximum or minimum). For example, the metric **D24HourMean** represents the average concentration for the sampling day. **D8HourMax** represents the highest of the 8-hour moving averages during the day. One pollutant may have multiple metrics defined. In order to use a Health Impact Function, the BenMAP pollutant definition must include the metric associated with that function.

In general, air pollution data in BenMAP-CE is hierarchical – a pollutant can have multiple **Metrics**, each of which has multiple **Statistics** (these are automatically calculated by BenMAP-CE) and which can have multiple **Seasonal Metrics**. Similarly, **Seasonal Metrics** have multiple **Statistics**. Furthermore, air pollution data can be provided to BenMAP-CE at any of these levels, in addition to the daily and hourly observation level, as described in more detail in Section 4.3.

Table 4-2. Example Calculation of D8HourMax

Hourly Period ¹	Hourly average O ₃ concentration ² (ppm)	Moving 8-hour average ³	D8HourMax ⁴
07:00	0.000	---	← 0.049
08:00	0.005	---	
09:00	0.010	---	
10:00	0.015	---	
11:00	0.020	---	
12:00	0.025	---	
13:00	0.030	---	
14:00	0.035	---	
15:00	0.040	0.020	
16:00	0.045	0.025	
17:00	0.050	0.030	
18:00	0.055	0.035	
19:00	0.060	0.040	
20:00	0.055	0.044	
21:00	0.050	0.047	
22:00	0.045	0.048	
23:00	0.040	<u>0.049</u>	
00:00	0.035	0.048	
01:00	0.030	0.047	
02:00	0.025	0.044	
03:00	0.020	0.040	
04:00	0.015	0.035	
05:00	0.010	0.030	
06:00	0.000	0.024	

¹ Days for measuring ozone start and end at 7:00 AM local standard time.

² Hourly average is the average of individual measurements taken during the hour.

³ Moving 8-hour average is the average of the hour and the preceding 7 hours.

⁴ *D8HourMax* is the maximum of the moving 8-hour averages.

Reference: Federal Register 79 FR 75233, Dec. 17, 2004.

<https://www.federalregister.gov/articles/2014/12/17/2014-28674/national-ambient-air-quality-standards-for-ozone#h-193>

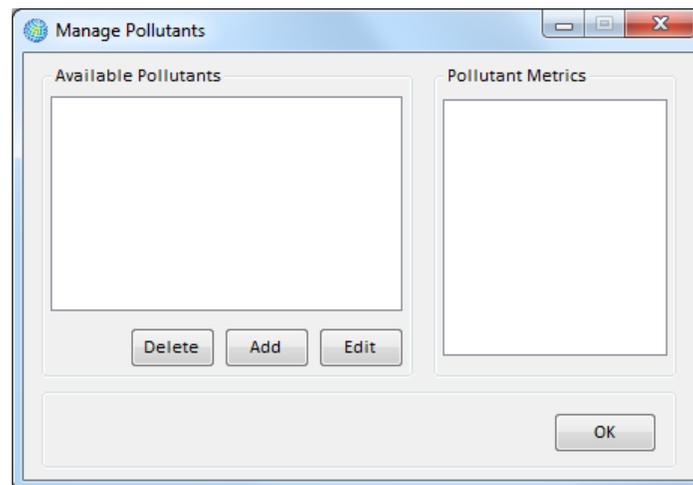
Add a Pollutant

Air pollution data in BenMAP-CE is of two types: (1) point source monitoring data and (2) **Grid Definition**-based modeling data. For both types, the data must be associated with a particular pollutant. Table 4-3 describes these variables used to define a pollutant in BenMAP-CE.

Table 4-3. BenMAP-CE Pollutant Definitions

Pollutant Field Name	Notes
Pollutant ID	Unique name for the pollutant which will be referenced in health impact functions, associated with monitoring and modeling data, etc.
Observation Type	Pollutants may have hourly observations or daily observations. In the United States, Ozone has hourly observations, while PM ₁₀ and PM _{2.5} have daily observations.
Metrics	Daily values calculated directly from daily observations, or through various mathematical manipulations of hourly observations. Typical ozone metrics include the highest hourly observations during the course of each day, the mean of all twenty four hourly observations, etc.
Seasonal Metrics	Seasonal values calculated from metric values. In the United States, for example, quarterly means are calculated for PM _{2.5} from daily means.

To add pollutant definitions to BenMAP-CE, click **Modify Datasets** on the menu bar. Then click on the **Manage** button below the **Pollutants** group. The **Manage Pollutants** window will appear. Here you may click **Add** to add a new **Pollutant**, **Delete** to remove a previously defined pollutant, or **Edit** to modify an existing pollutant.



To start defining a pollutant, click the **Add** button and the **Pollutant Definition** window will appear. In the **Pollutant ID** box, you give a unique name for the pollutant (e.g., PM_{2.5}), and then define the characteristics of this pollutant – the **Observation Type** and **Metrics**.

The screenshot shows the 'Pollutant Definition' dialog box. The 'Main' section on the left contains a 'Pollutant ID' text box with 'PM2.5', an 'Observation Type' dropdown menu set to 'Daily', and a 'Metrics' list box containing 'D24HourMean'. Below the list are 'Delete', 'Add', and 'Define Seasons for all Pollutant Metrics' buttons. The 'Detail' section on the right has a 'Metric Name' text box with 'D24HourMean', an 'Hourly Metric Generation' dropdown menu set to 'Fixed Window', and a 'Fixed Window' sub-section with 'Start Hour' (0), 'End Hour' (23), and 'Statistic' (Mean) dropdowns. At the bottom of the 'Detail' section is a 'Manage Seasons for Individual Pollutant Metrics' list box containing 'QuarterlyMean' and an 'Edit' button. The dialog box has 'Cancel' and 'OK' buttons at the bottom right.

The **Observation Type** identifies whether a pollutant is measured *Hourly* or *Daily*. In the United States, ozone, sulfur dioxide, carbon monoxide, and others have hourly observations, while particulate matter has daily observations.

Next you need to define a pollutant's **Metrics**. A pollutant has to have one or more metrics, which are daily values calculated directly from daily observations, or through various mathematical manipulations of hourly observations.

To add a **Metric**, click on the **Add** button below the **Metrics** box in the **Pollutant Definition** window. A default name 'Metric 0' will appear in the box. Since the default name is not very descriptive of a metric, it is best to change the name. Typical names used for metrics given in Table 4-4. These are provided just as an example, you may use any names that you like. However, keep in mind that the names that you use for your

metrics need to be consistent with the metric names that you include in your air pollution monitoring and modeling data, as well as your health impact functions. (We will discuss this further below.) Additionally, metric names are used to display pollutant concentrations in BenMAP-CE's mapping window. As such, they must be consistent with GIS naming conventions, meaning they must begin with a letter, and may only contain letters, numbers, and underscores.

Table 4-4. Examples of Metric Names

Name	Description
D1HourMax	Highest hourly value from 12:00 A.M. through 11:59 P.M.
D8HourMax	Highest eight-hour average calculated between 12:00 A.M. and 11:59 P.M.
D24HourMean	Average of hours from 12:00 A.M. through 11:59 P.M.

4.1.2.1 Hourly Metrics

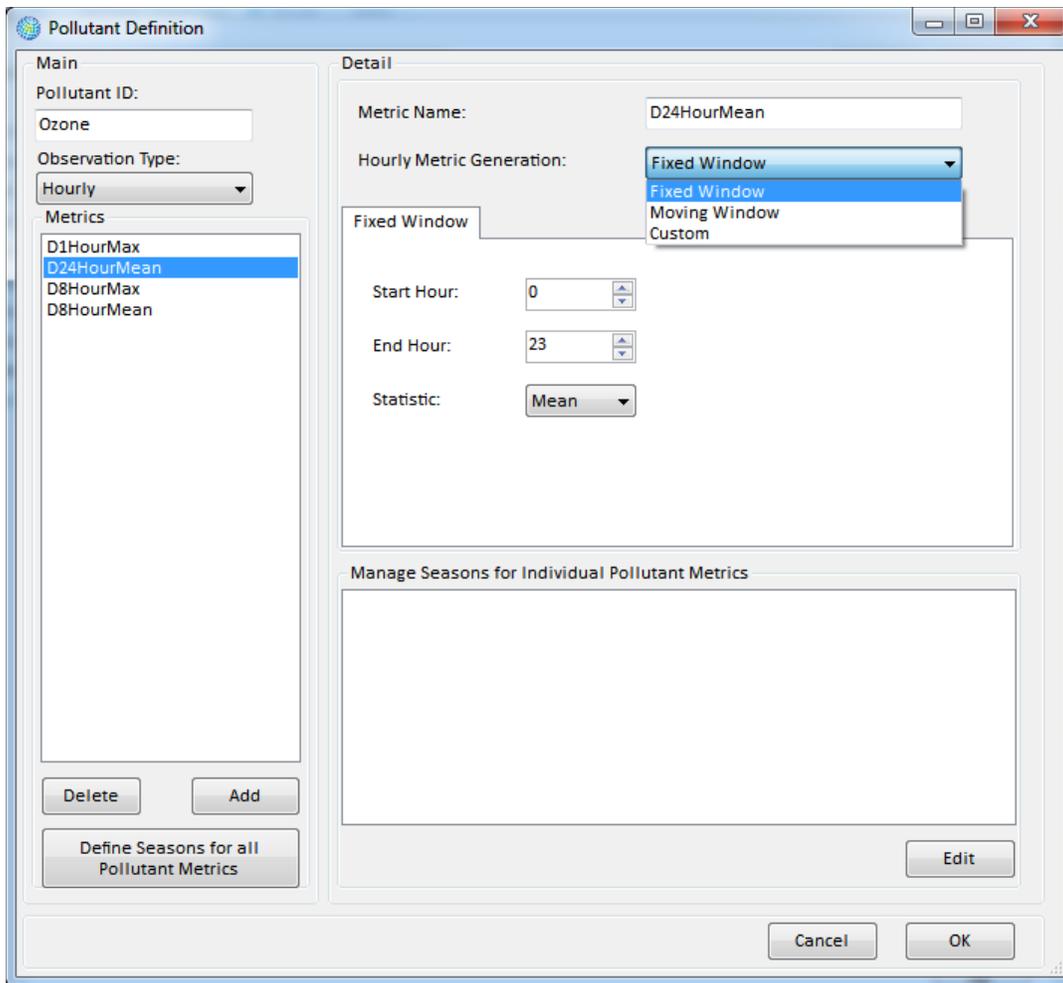
Pollutants that are measured hourly (**Observation Type** = *Hourly*), such as ozone, sulfur dioxide, carbon monoxide, and others, must be characterized by a daily metric, which mathematically summarizes the hourly observations.

Table 4-4 lists some of the ways that metrics can be generated from hourly values. Note that these metrics are not arbitrarily chosen, and instead match the metrics used in epidemiological studies.

The **Detail** section of the **Pollutant Definition** window lets you define the metrics that you want to use. There are three options that you may choose using the **Hourly Metric Generation** drop-down list: *Fixed Window*, *Moving Window*, and *Custom*.

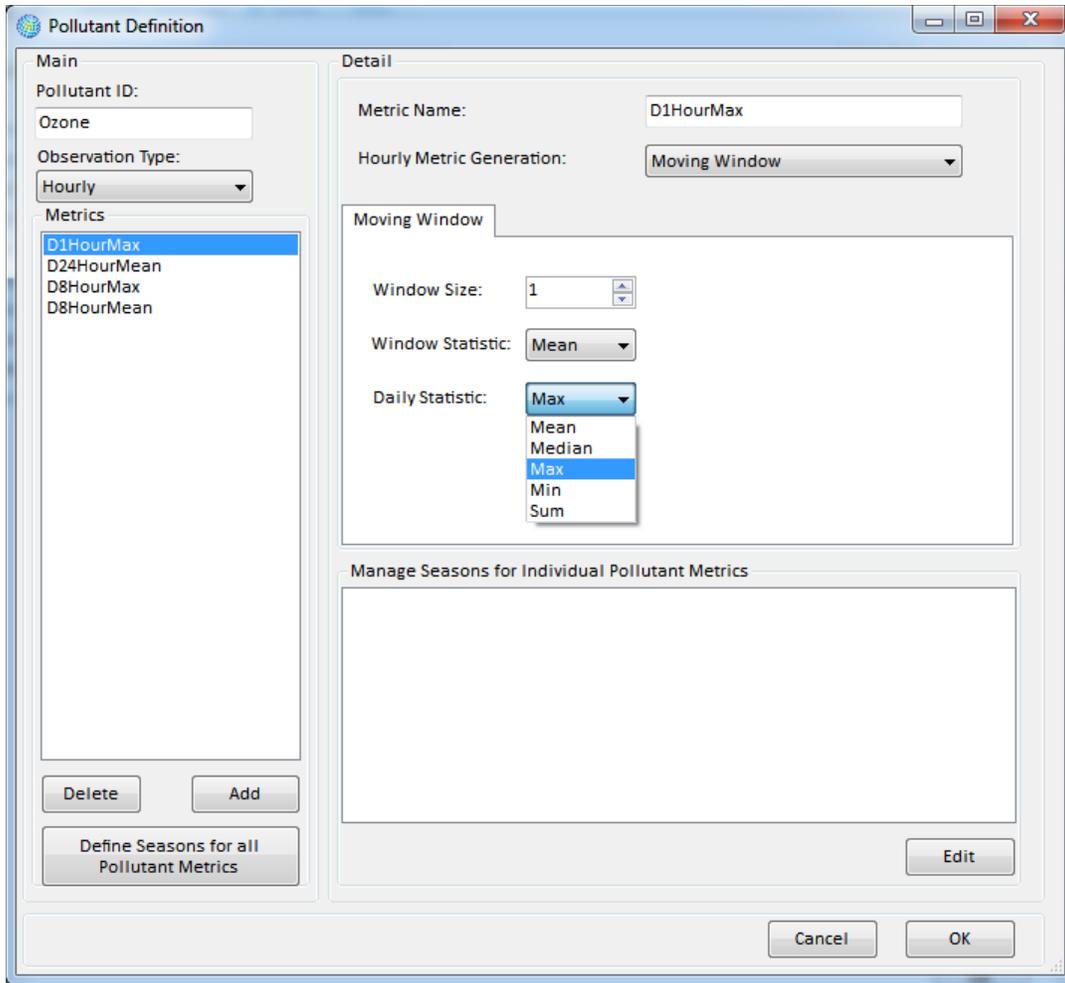
The **Fixed Window** option lets you define simple metrics which are calculated as statistics over a fixed window of hours (**Start Hour** and **End Hour**) within each day. The **Start Hour** should be less than or equal to the **End Hour**, and both can range from 0 to 23, where 0 stands for the period 12:00 am to 12:59 am, and 23 stands for 11:00 pm to 11:59 pm. The **Statistic** includes the *Mean*, *Median*, *Max*, *Min*, and *Sum*. Some examples follow:

- **D24HourMean**: The mean of the observations from 12:00 am through 11:59 pm. **Start Hour** = 0. **End Hour** = 23. **Statistic** = *Mean*.
- **D1HourMax**: The highest hourly value of the observations from 12:00 am through 11:59 pm. **Start Hour** = 0. **End Hour** = 23. **Statistic** = *Max*.
- **D12HourMean**: The mean of the daylight observations, defined as the period from 8:00 am through 7:59 pm. **Start Hour** = 8. **End Hour** = 19. **Statistic** = *Mean*.



The **Moving Window** option lets you consider metrics that are not based on the same set of hours each day. The **Window Size** defines the number of hours that will be considered together. The **Window Statistic** defines how the hours in the **Window Size** will be characterized. And the **Daily Statistic** defines how BenMAP-CE will use the statistics generated for each window.

For example, consider the highest eight-hour mean (*D8HourMax*) over the course of a day. You would have the following settings: **Window Size** = 8. **Window Statistic** = *Mean*. **Daily Statistic** = *Max*. BenMAP-CE would calculate every possible eight-hour mean, starting with the eight-hour mean from 12:00 am through 7:59 am, and ending with the eight-hour mean from 4:00 pm through 11:59 pm. This would generate 17 possible eight-hour means. BenMAP-CE would then choose the eight-hour mean that has the highest value (see Table 4-2 above for an example).



The **Custom** tab lets you define **Metrics** using a mathematical function that you specify. These functions can include measures such as the sum of the number of hours of ozone exposure above 60 ppb. The possibilities are quite diverse, as evidenced by the range of functions and variables available for use as shown in Table 4-5. However, the syntax for using these functions is somewhat involved, so we have reserved discussion of this for the Appendix M: Function Editor.

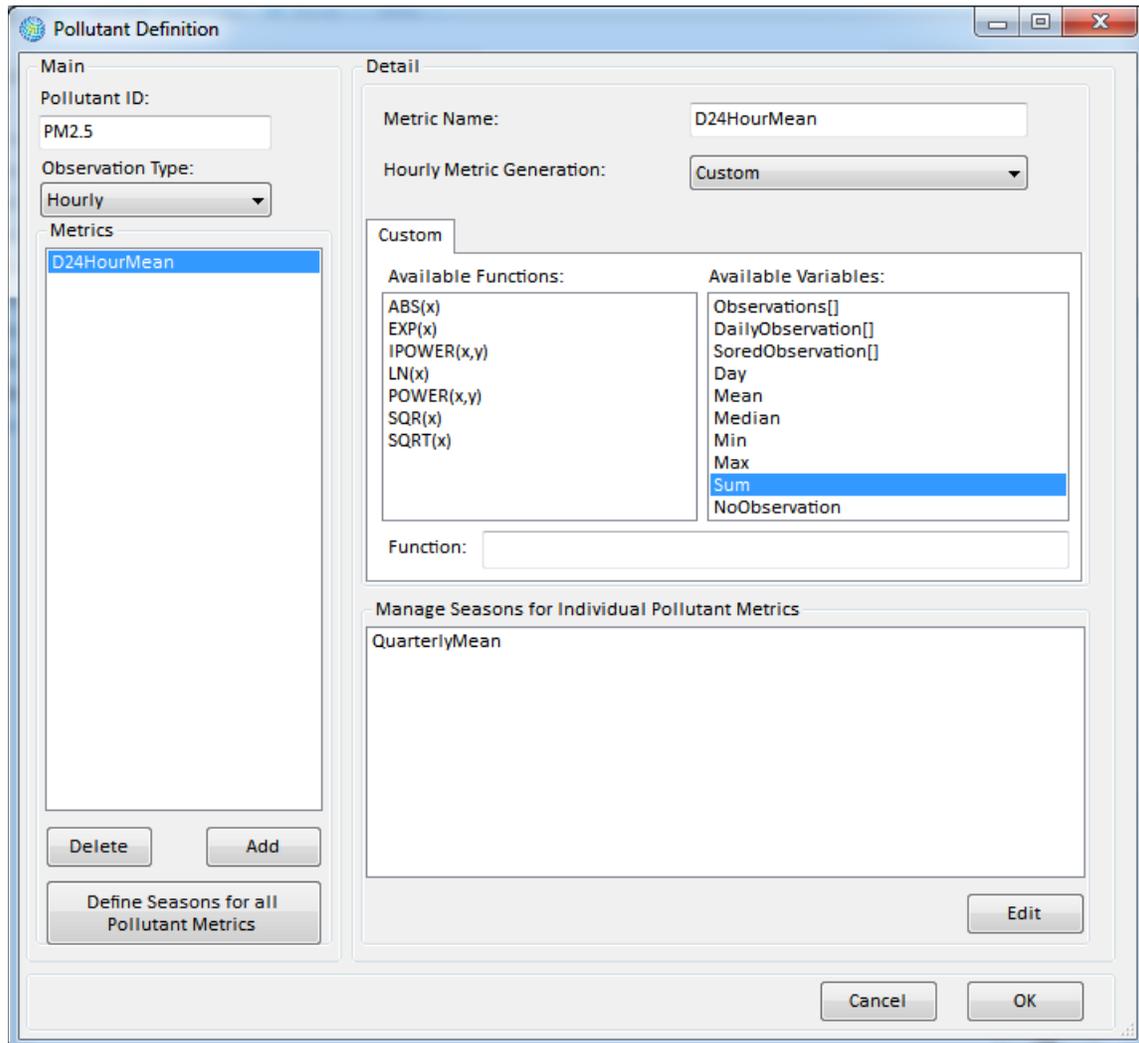


Table 4-5. Available Functions and Variables for Custom Metrics

Name	Description
Functions	
ABS(x)	Returns the absolute value of x.
EXP(x)	Returns e the power x, where e is the base of the natural logarithm.
IPOWER(x,y)	Returns x to the power y (y an integer value).
LN(x)	Returns the natural logarithm of x.
POWER(x,y)	Returns x to the power y (y a floating point value).
SQR(x)	Returns the square of x.
SQRT(x)	Returns the positive square root of x.
Variables	
Observations[i]	All hourly observations for the year (index begins at zero, typically ranging to 8,760).
DailyObservations[i]	All hourly observations for the day (indexed zero to twenty-three).
SortedObservations[i]	All hourly observations for the day, sorted from low to high (indexed zero to twenty-three).
Day	Index of the day whose metric value is being generated (index begins at zero).
Mean	Mean of the daily observations.
Median	Median of the daily observations.
Min	Minimum of the daily observations.
Max	Maximum of the daily observations.
Sum	Sum of the daily observations.
NoObservation	Flag value indicating a missing observation (-345)

4.1.2.2 Manage Seasons for Individual Pollutant Metrics

Manage Seasons for Individual Pollutant Metrics allow you to aggregate daily **Metric** values over a portion of the year that you define. This has a number of uses. For example, if pollutant values vary greatly by season of the year, you can calculate separate pollutant measures for each season of interest. You might be interested in *Dry Season* versus *Wet Season*, or differences between *Winter*, *Spring*, *Summer*, and *Fall*.

Fundamental Concept - Seasonal Metric

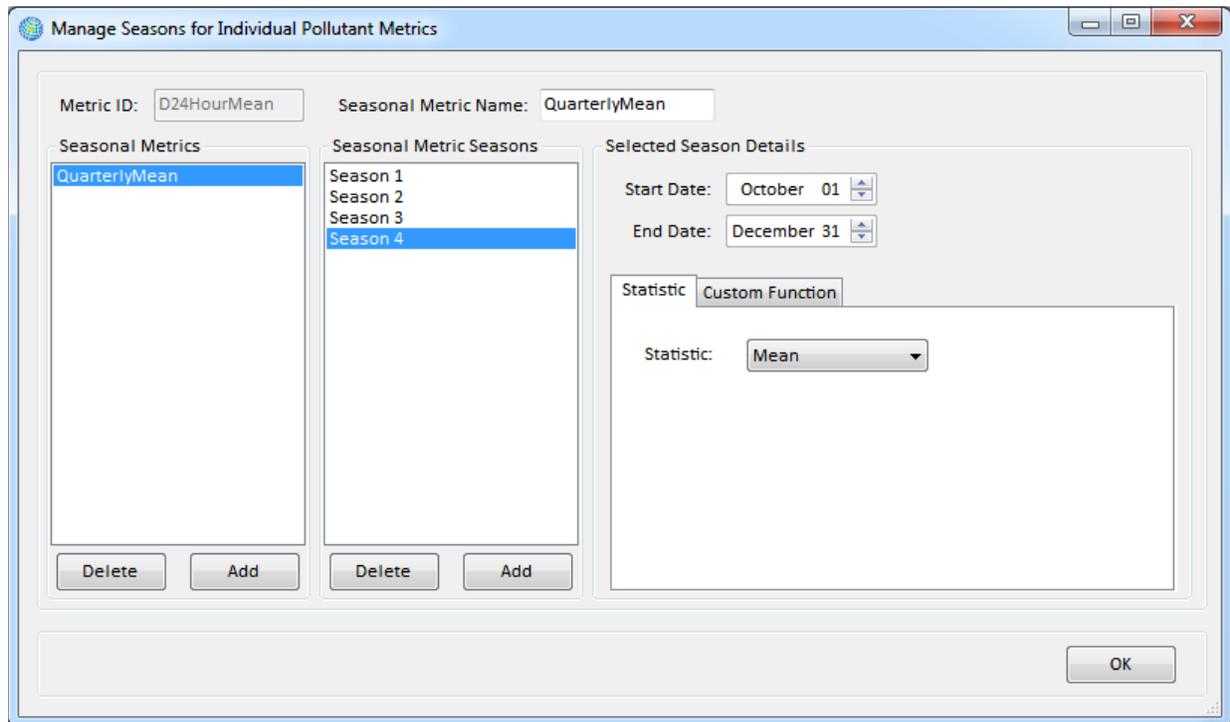
A **Seasonal Metric** allows you to define portions of the year over which to aggregate daily air quality metrics. For example, you could create a warm season and cold season for D8HourMax ozone and calculate an average WarmSeasonD8HourMax ozone value and an average ColdSeasonD8HourMax to use in an analysis of seasonal health impacts.

To add seasonal metrics for individual pollutant metrics, first select the metric of interest (e.g., D24HourMean), then click on the **Edit** button below the **Manage Seasons**

for **Individual Pollutant Metrics** box. The **Manage Seasons for Individual Pollutant Metrics** window will appear.

To add a **Seasonal Metric**, click on the **Add** button below the **Seasonal Metrics** box. A default name *Seasonal Metric 0* will appear in the box. Since the default name is not very descriptive, it is best to change the name to something more informative such as the *QuarterlyMean*. As with the **Metric** names, keep in mind that the **Seasonal Metric** names need to be consistent with the metric names that you include in your air pollution monitoring and modeling data, as well as your health impact functions.

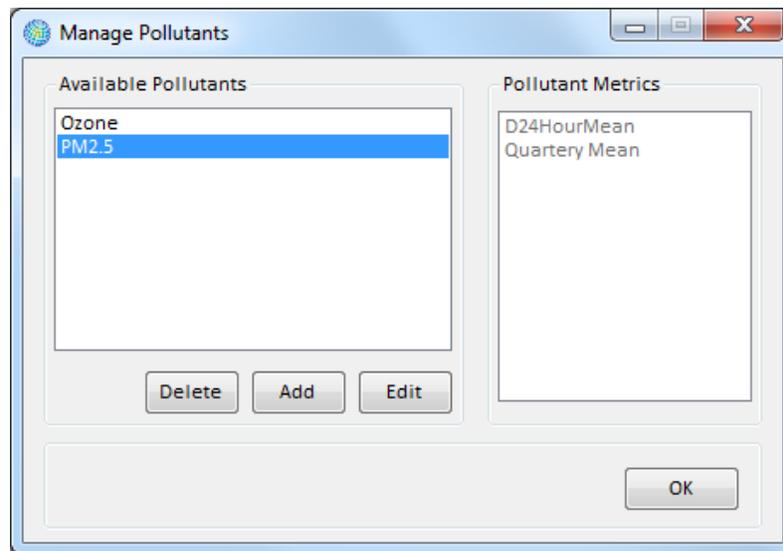
The next step is to define the seasons that you want associated with your **Seasonal Metric** name. For example, in the case of a *Quarterly Mean*, you would want to define four seasons. To start this process, click on the **Add** button below the **Seasonal Metric Seasons** box. Then, in the far right side of the window under **Selected Season Details**, give the **Start Date** and **End Date** for each season. To change the date, click on the month or day, and use the arrows to change the month or day accordingly.



Next, you need to choose the **Statistic** tab or the **Custom Function** tab to determine how the daily **Metric**s will be combined in each season. For example, you might choose the *Mean* from the drop-down list on the **Statistics** tab. This would calculate the mean of the daily metrics in each season. The **Custom** tab allows seasonal metric values to be calculated using customized functions, similar to those used to calculate daily metric values from hourly observations. See Appendix M: Function Editor for more detail on this topic.

Once you have finished defining the **Seasonal Metrics**, click **OK** to return to the **Pollutant Definition** window.

Click **OK** after defining each **Pollutant**. This will return you to the **Manage Pollutants** window.



4.1.2.3 Define Seasons for all Pollutant Metrics

The **Define Seasons for all Pollutant Metrics** button on the **Pollutant Definition** window allows you to associate **Seasons** with a **Pollutant** and all of its associated metrics. These seasons differ somewhat from the **Seasonal Metrics** defined for individual pollutant metrics (discussed above). They are used to define:

Fundamental Concept – Global Season

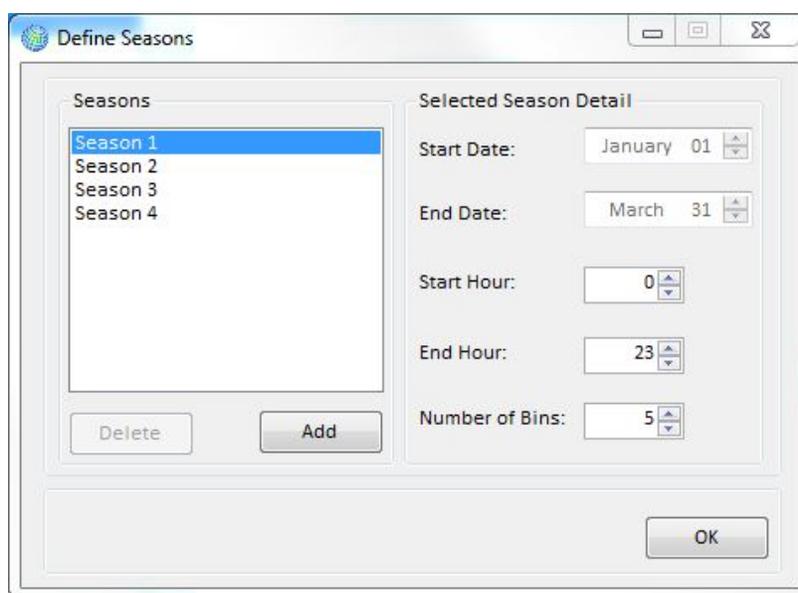
Defining **Seasons** (or **Global Seasons**) for all pollutant metrics constrains a pollutant’s benefits analyses to a user-specified portion of the year. For example, if you define the Global Season for ozone as April 1 – September 30, then BenMAP-CE will only calculate benefits during that window for each metric associated with ozone. Any Seasonal Metrics should be contained within and collectively span the Global Season. For example, for a Global Season of April 1 – Sept. 30, you might define a Seasonal Metric to analyze health impacts in early summer (April 1 – June 30) vs. late summer (July 1 – Sept 30).

A Global Season also defines the portion of the year for which missing pollutant concentrations are estimated in by BenMAP-CE and how the replacement value is calculated (e.g., days with missing air quality data may be replaced with the average value from the Global Season).

- The portion of the year for which benefits are calculated for a **Pollutant**. You can think of **Seasons** as defining this period of the year “globally” for the pollutant, as it affects the portion of the year over which both **Metrics** and **Seasonal Metrics** are calculated. For example, in the United States ozone benefits are often only calculated for the ozone season, from April 1 through September 30.
- The portion(s) of the year for which missing pollutant concentrations are filled in by BenMAP-CE. That is, in order to

calculate benefits, BenMAP-CE in certain cases needs to generate complete sets of metric values by estimating concentrations for those days that have missing observations. This can be important if certain seasons tend to have more missing values than others.

To define **Seasons** for a **Pollutant**, click the **Define Seasons for all Pollutant Metrics** button. This will bring up the **Define Seasons** window. For each season desired, click the **Add** button, select the appropriate **Start Date** and **End Date**, which define the days included in the season; and the appropriate **Start Hour** and **End Hour**, which define the hours included in monitoring period. The advanced options for PM_{2.5} look like the following:



Once you have finished defining the **Seasons**, click **OK** to return to the **Pollutant Definition** window.

If you later wish to View or Edit a particular **Pollutant** definition, simply select the appropriate **Pollutant** within the **Available Pollutants** box and click the **Edit** button. When you are done, click **OK** to return to the **Modify Datasets** window.

After defining all of the pollutants that you want, click **OK**. This will return you to the **Modify Datasets** window.

4.1.3 Monitor Datasets

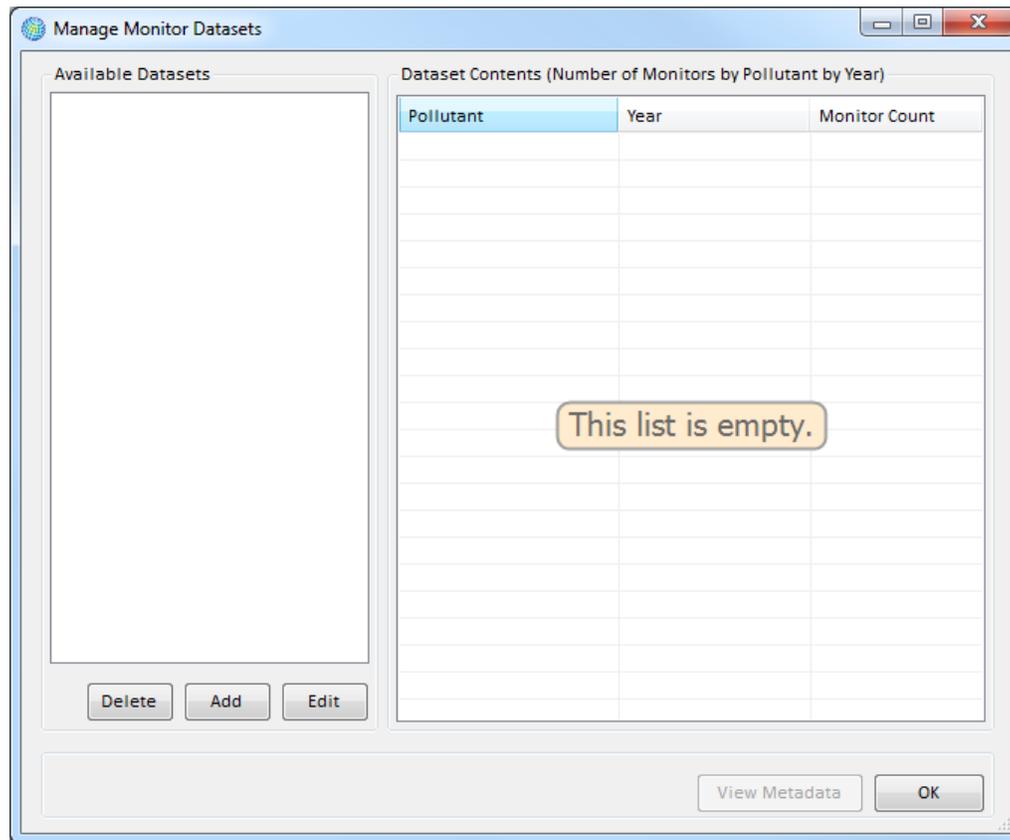
The **Monitor Datasets** section of the **Modify Datasets** window allows you to add air pollution monitoring data to your setup. Air pollution monitoring data may be used to estimate ambient pollution levels in each grid cell defined by a **Grid Definition**.

BenMAP-CE uses a variety of procedures (such as *Voronoi Neighbor Averaging*, discussed later) to interpolate the monitor data points across the area of interest.

NOTE: Air pollution data in BenMAP-CE is of two types: (1) point source monitoring data and (2) **Grid Definition**-based modeling data. Both types of data must be associated with a particular pollutant that you have already defined. Only the point source monitoring data is stored in the setup database. The modeling data are loaded into BenMAP-CE as you need them for a particular analysis.

4.1.3.1 Add Monitor Datasets

To start, click on the **Manage** button below the **Monitor Datasets** box. The **Manage Monitor Datasets** window will appear. From this window you may **Add** monitoring data, view and **Edit** existing datasets, as well as **Delete** them. The section on the left under **Available Datasets** lists the monitor datasets that are currently in the setup. The section on the right under the **Dataset Contents** identifies the number of monitors in each dataset by *Pollutant* and by *Year*. To view the metadata of a particular monitor dataset, select an **Available Dataset** and click on a row from the **Dataset Contents**, then click the **View Metadata** button. This allows you to view further information about references or descriptions of the file.



To start adding data, click the **Add** button. This will bring up the **Monitor Dataset Definition** window. Give the dataset a name in the **Dataset Name** box, choose the appropriate pollutant from the **Pollutant**¹⁵ drop-down menu, and then type the 4-digit *Year* of the data in the **Year** box.

NOTE: The **Dataset** that you define can have one or more pollutants and multiple years of data (e.g., representing a particular monitoring network). However the data must be imported one pollutant and one year at a time.

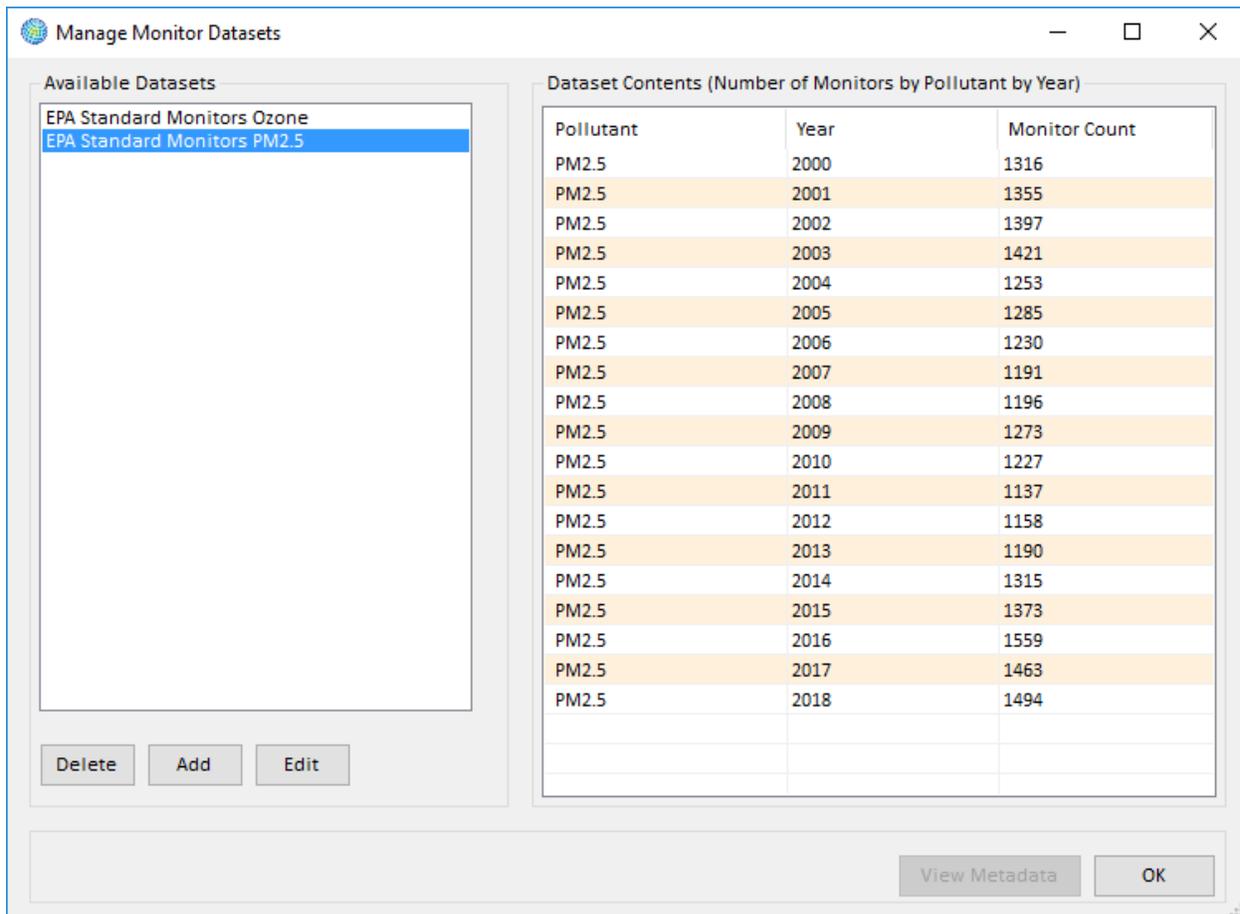
Monitor data must be formatted in a database file, with monitor definition information and monitor values in a single line.

After specifying the **Pollutant** and **Year**, click on the **Load Data From File** button to bring up a window from which you can **Browse** the BenMAP-CE Data directory to find the desired data file. Click **Open**, to choose the file.

¹⁵ The pollutants in the **Pollutant** drop-down menu have been defined under the **Pollutants** box on the **Modify Datasets** window.

Users are advised to click the **Validate** button before loading the monitor dataset. BenMAP-CE's validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings.¹⁶ The user also has the option of adding metadata to the file to save references and comments about that specific file. To add metadata, click the **View Metadata** button. After passing validation (and adding metadata if desired) click the **OK** button to bring you back to the **Monitor Dataset Definition** window.

Repeat this procedure to load all of your monitoring data. If you try to load the data for the same pollutant/year combination, BenMAP-CE will warn you of the duplication.



To see the years of data and the number of monitors each year, use the scrollbars on the bottom and on the right of the **Dataset Contents** box. To view the metadata for a particular entry in the dataset, choose an **Available Dataset** and row in the **Dataset Contents** and click the **View Metadata** button.

To delete existing datasets, select the dataset in the **Available Datasets** list and click the **Delete** button. To edit an existing dataset, select the dataset in the **Available**

¹⁶ Validation reports are saved to C:\Users\\Documents\My BenMAP-CE Files\ValidationResults.

Datasets list and click the **Edit** button. (Note: Certain pre-loaded datasets cannot be edited. Instead if you select one of these, you will have the option to **Copy** the locked dataset and then you can edit the copied dataset.)

When you have finished loading your monitor data, click **OK** in the **Manage Monitor Datasets** window. This will take you back to the **Modify Datasets** window, which will show the name of the **Dataset(s)** that you just entered.

4.1.3.2 Format for Monitor Data

Monitor data is required to be formatted in a single database file, with monitor definition information and monitor values in a single line. Tables 4-6a and 4-6b list the variables in the monitor dataset and provide a sample of what a data file might look like.

NOTE: The monitor data files do not specify the pollutant with which the data is associated—this is specified by the user when loading the monitor data into BenMAP-CE.

Table 4-6a. Required Format, Air Monitoring Data File Variables

Variable	Type	Required	Notes
Monitor Name	Text	Yes	Unique name for each monitor in a particular location.
Monitor Description	Text	No	Description of the Monitor.
Longitude	Numeric (double)	Yes	Values should be in decimal degree format. Values in the eastern hemisphere are positive, and those in the western hemisphere are negative.
Latitude	Numeric (double)	Yes	Values should be in decimal degree format. Values in the northern hemisphere are positive, and those in the southern hemisphere are negative.
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., 1-hour daily maximum) 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).
Statistic	Text	No	This is an annual metric, which is either blank (signifying that the values are not annual statistics) or must be one of: None, Mean, Median, Max, Min, Sum. (e.g., mean of the 1-hour maximum for the year)
Values	Text	Yes	If Metric is blank, values are supplied as a comma-delimited string of values for the year [e.g., 365 or 366 (leap year) values for daily data, 8760 or 8784 (leap year) values for hourly data]. If Metric is defined, but Seasonal Metric and Statistic are blank, 365 or 366 metric values. If Seasonal Metric is defined, but Statistic is blank, n seasonal metric values. If Statistic is defined, one annual statistic value for either the Metric (if Seasonal Metric is blank) or the Seasonal Metric. Missing values are signified with a period ('.').

Table 4-6b. Required Format, Sample Air Monitoring Data File

Monitor Name	Monitor Description	Latitude	Longitude	Metric	Seasonal Metric	Statistic	Values
260050003881011	'RESIDENTIAL, SUBURBAN'	42.7678	-86.1486	D24HourMean	QuarterlyMean	Mean	11.67
260170014881011	'RESIDENTIAL, URBAN AND CENTER CITY'	43.5714	-83.8907	D24HourMean	QuarterlyMean	Mean	11.62
260210014881011	'COMMERCIAL, RURAL'	42.1978	-86.3097	D24HourMean	QuarterlyMean	Mean	11.58
260490021881011	'RESIDENTIAL, URBAN AND CENTER CITY'	43.0472	-83.6702	D24HourMean	QuarterlyMean	Mean	11.56
260650012881011	'RESIDENTIAL, URBAN AND CENTER CITY'	42.7386	-84.5346	D24HourMean	QuarterlyMean	Mean	11.49
260770008881011	'COMMERCIAL, URBAN AND CENTER CITY'	42.2781	-85.5419	D24HourMean	QuarterlyMean	Mean	11.44
260810007881011	'INDUSTRIAL, URBAN AND CENTER CITY'	42.9561	-85.6791	D24HourMean	QuarterlyMean	Mean	11.4
260810020881011	'INDUSTRIAL, URBAN AND CENTER CITY'	42.9842	-85.6713	D24HourMean	QuarterlyMean	Mean	11.36
260990009881011	'COMMERCIAL, SUBURBAN'	42.7314	-82.7935	D24HourMean	QuarterlyMean	Mean	11.43
261010922881011	'RESIDENTIAL, URBAN AND CENTER CITY'	44.3070	-86.2426	D24HourMean	QuarterlyMean		11.43,11.07,10.98,10.90
261130001881011	'FOREST, RURAL'	44.3106	-84.8919	D24HourMean	QuarterlyMean		11.44,14.23,11.20,9.30
261150005881011	'AGRICULTURAL, RURAL'	41.7639	-83.4719	D24HourMean	QuarterlyMean		11.39,13.71,10.04,11.24
261210040881011	'COMMERCIAL, URBAN AND CENTER CITY'	43.2331	-86.2386	D24HourMean	QuarterlyMean		11.28,9.23,15.03,11.10
261250001881011	'RESIDENTIAL, SUBURBAN'	42.4631	-83.1832	D24HourMean	QuarterlyMean		11.27,11.12,13.15,12.03
261390005881011	'RESIDENTIAL, SUBURBAN'	42.8945	-85.8527	D24HourMean	QuarterlyMean		11.27,10.05,10.9,12.13
261470005881011	'RESIDENTIAL, SUBURBAN'	42.9533	-82.4562	D24HourMean	QuarterlyMean		11.26,12.13,15.00,10.01
261610008881011	'COMMERCIAL, URBAN AND CENTER CITY'	42.2406	-83.5996	D24HourMean	QuarterlyMean		11.24,14.05,11.03,10.10
261630001881011	'COMMERCIAL, SUBURBAN'	42.2286	-83.2082	D24HourMean	QuarterlyMean		11.23,9.23,13.04,10.09
261630015881011	'COMMERCIAL, URBAN AND CENTER CITY'	42.3028	-83.1065	D24HourMean	QuarterlyMean		11.23,11.24,10.45,10.04
261630016881011	'RESIDENTIAL, URBAN AND CENTER CITY'	42.3578	-83.0960	D24HourMean	QuarterlyMean		11.2,10.73,11.23,10.45
261630019881011	'RESIDENTIAL, SUBURBAN'	42.4308	-83.0001	D24HourMean			...,13.01,17.19,3.89,13.86,5.9
261630025881011	'COMMERCIAL, SUBURBAN'	42.4231	-83.4263	D24HourMean			...,13.48,8.73,6.079,14.534,3.
261630033881011	'INDUSTRIAL, SUBURBAN'	42.3067	-83.1488	D24HourMean			...,15.675,12.72,6.46,16.24,6.
261630036881011	'COMMERCIAL, SUBURBAN'	42.1873	-83.1539	D24HourMean			...,16.52,15.01,5.41,15.39,5.0
261630038881011	'RESIDENTIAL, URBAN AND CENTER CITY'	42.3350	-83.1096	D24HourMean			...,20.52,21.85,5.88,19.28,5.1
261630039881011	'RESIDENTIAL, URBAN AND CENTER CITY'	42.3233	-83.0685	D24HourMean			...,20.14,7.59,8.17,13.11,7.59

4.1.4 Incidence and Prevalence Rates Data

Most health impact functions, such as those developed from log-linear or logistic health impact functions, estimate the percent change in a health effect associated with a pollutant change. In order to estimate the absolute change in incidence using these functions, the baseline incidence rates (and in some cases the prevalence rate) of the adverse health effect are needed.

The incidence rate is the number of health effects per person in the population per unit of time, and the prevalence rate is the percentage of people that suffer from a particular chronic illness. For example, the incidence rate for asthma attacks may be 25 cases per asthmatic individual per year, and the prevalence rate (measuring the percentage of the population that is asthmatic) might be six percent of the total population.

Fundamental Concepts – Incidence and Prevalence

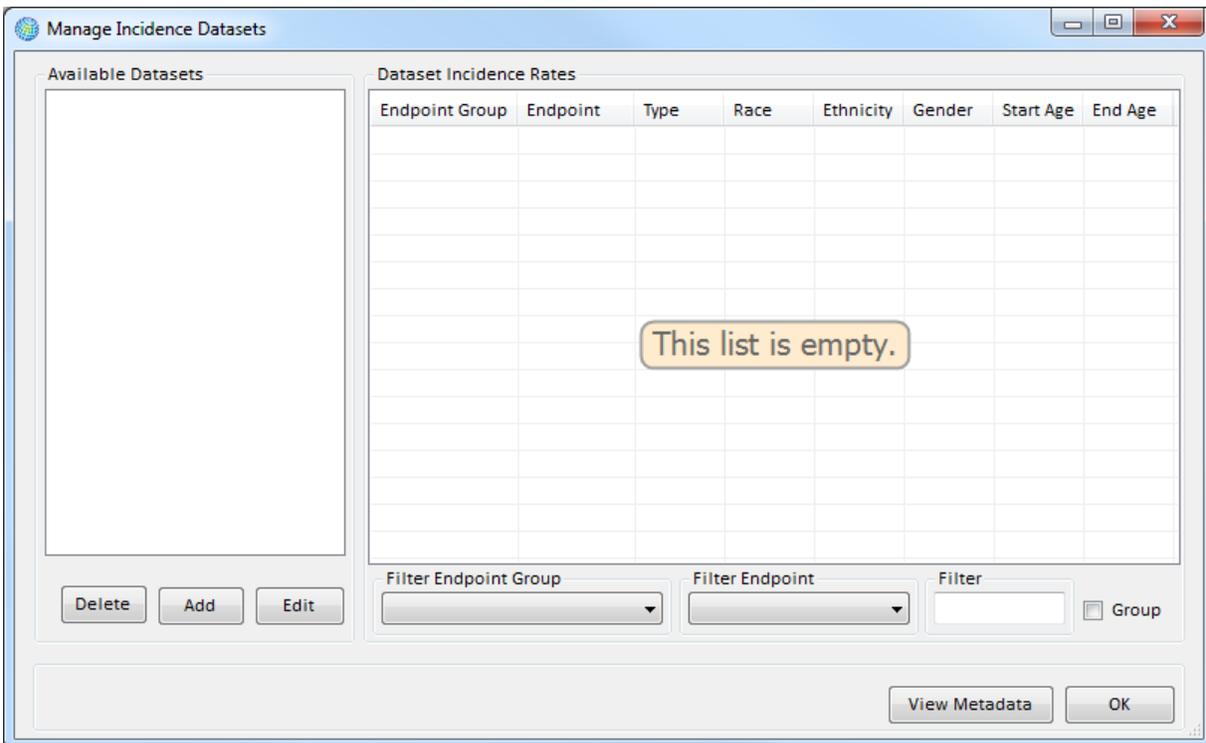
Incidence is a measure of the total number of new occurrences of an adverse health impact in a geographic area over time. The **incidence rate** is the average number of adverse health effects (e.g., respiratory hospital admissions) per person per unit of time, typically a day or a year. The **incidence 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).

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 adverse 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 exhibiting a co-morbidity.

NOTE: For both incidence and prevalence rates, BenMAP-CE allows the user to have rates that vary by race, ethnicity, gender, and age group. BenMAP-CE can support multiple sets of incidence and prevalence rates, if the rates differ by year or by grid definition.

4.1.4.1 Add Incidence/Prevalence Rates

To start adding baseline incidence and prevalence data files, click on the **Manage** button below the **Incidence/Prevalence Rates** box. The **Manage Incidence Datasets** window will appear.



In this window you may **Add**, **Edit**, and **Delete** datasets. The section on the left under **Available Datasets** lists the incidence/prevalence datasets that are currently in the setup. The section on the right under the **Dataset Incidence Rates** identifies the rates in the selected dataset.

To add a dataset, click the **Add** button. This will bring up the **Incidence Dataset Definition** window. Give a name to the dataset that you are creating by typing a name in **Dataset Name** box.

Incidence Dataset Definition

Dataset Name:

Grid Definition:

Dataset Incidence Rates

Filter Endpoint Group: Filter Endpoint: Filter: Group

Endpoint Group	Endpoint	Type	Race	Ethnicity	Gender	Start Age	End Age
This list is empty.							

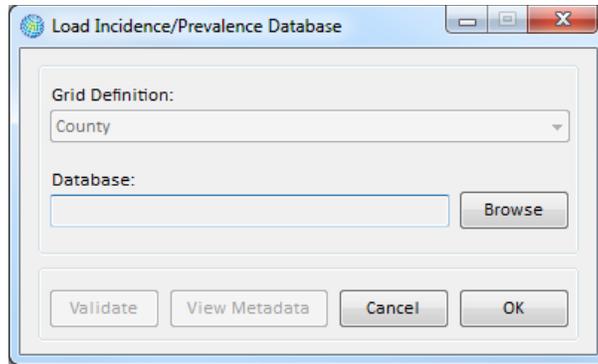
Column	Row	Value
This list is empty.		

Output Sample File

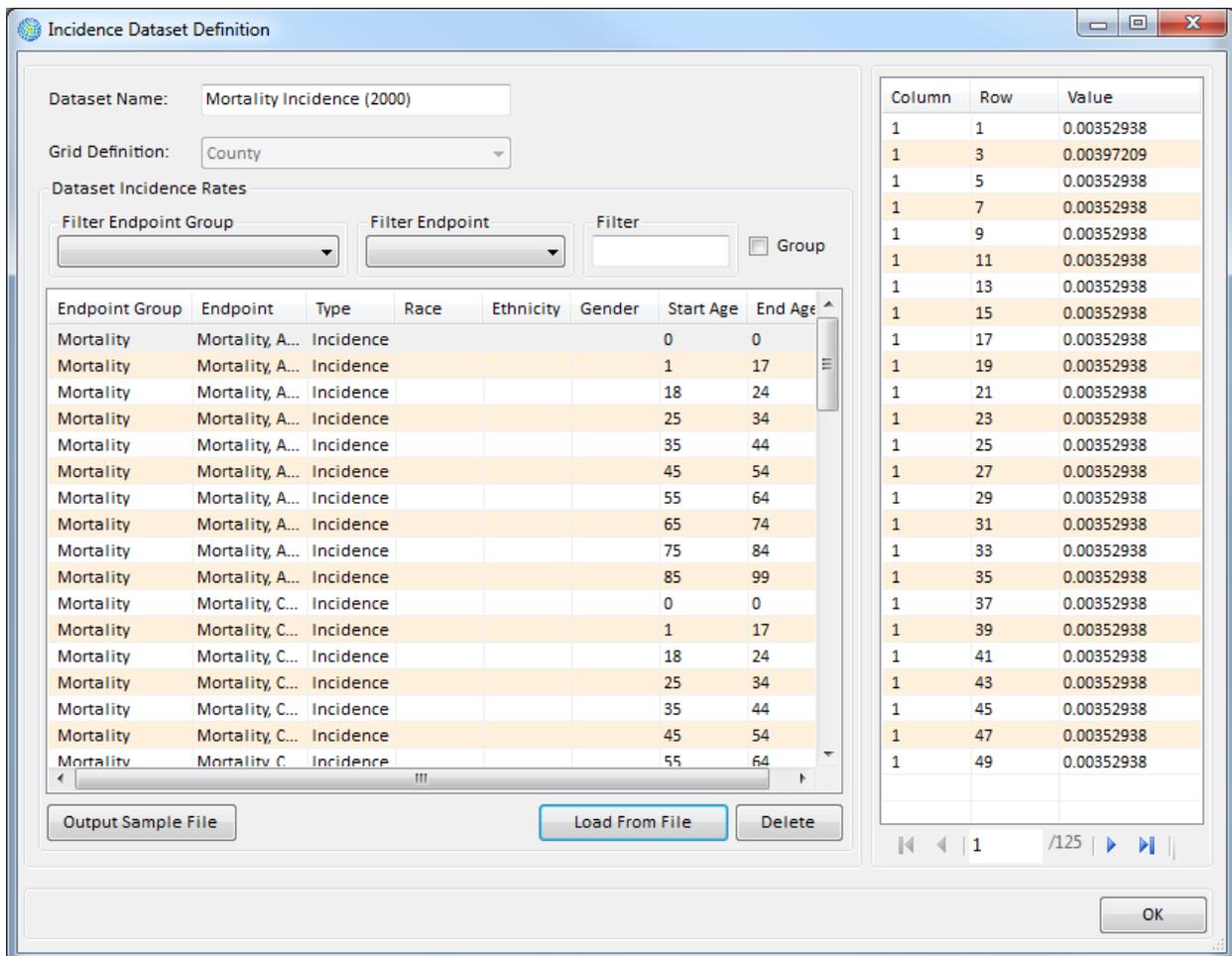
0 of 0

NOTE: If you have multiple incidence or prevalence datasets that vary, for example, by year and grid definition, then use the name to provide a reference to the year and grid definition (e.g., “Mortality Incidence (2000)”).

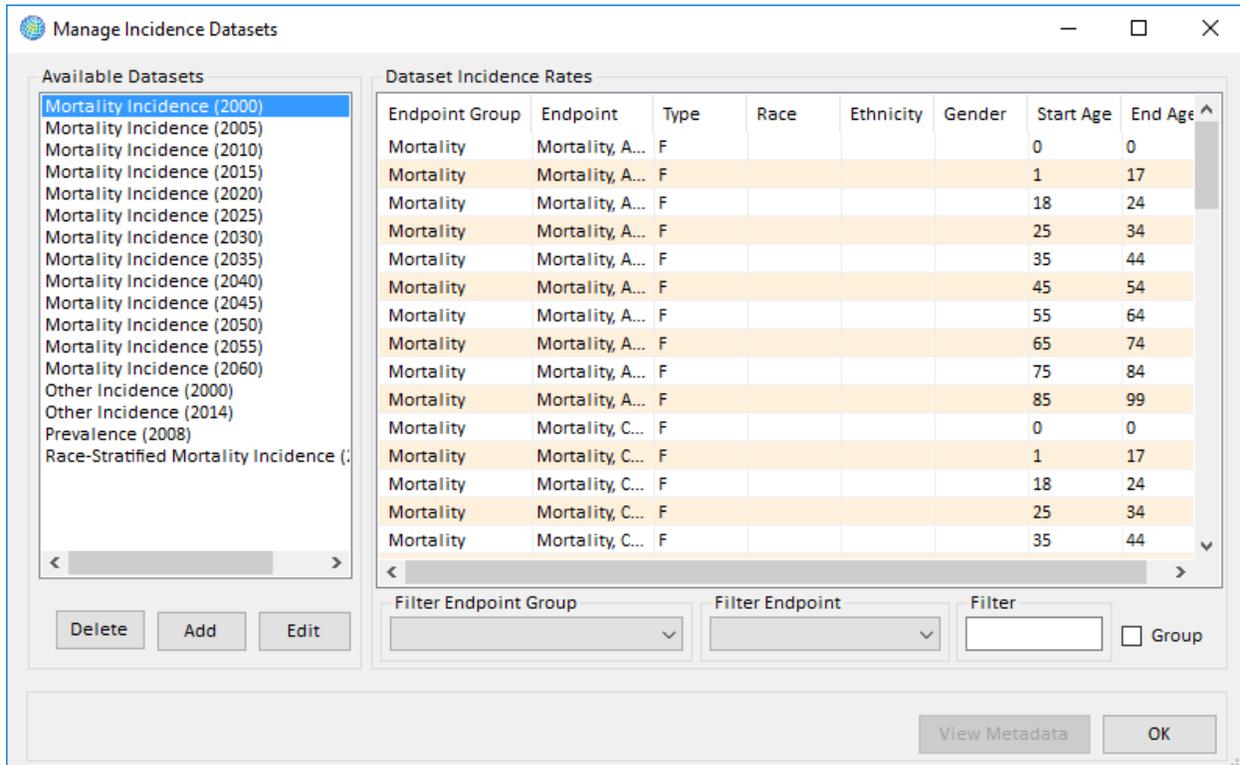
In the **Grid Definition** drop-down list choose the item that matches the grid definition used to develop the incidence/prevalence dataset. Note: The incidence and prevalence rate data must use the same column/row information as the matching grid definition. Click the **Load From File** button. Then click on the **Browse** button, to browse for the dataset file. (The format for the dataset is detailed in the next sub-section.)



After locating the file, click **Open**. Click the **Validate** button before loading the data. BenMAP-CE’s validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings. You also have the option of adding metadata to the dataset. This is done by clicking the **View Metadata** button and adding any references or descriptions that you see fit. Click **OK** on the **Load Incidence/Prevalence Database** window to load the selected file. The **Incidence Dataset Definition** window will appear, displaying the rates in the data file that you just loaded.



If the data look correct, click **OK**. This will return you to the **Manage Incidence Datasets** window. To view any metadata that was added, select an **Available Dataset** and a **Rate**, and click the **View Metadata** button. The user can view or edit the metadata of the imported files.



Follow the same procedure for any additional incidence/prevalence datasets that you want to add to the setup database. When you have finished adding data, click **OK** in the **Manage Incidence Datasets** window. The **Incidence/Prevalence Rates** box in the **Modify Datasets** window will show the datasets that you have entered.

Warning

In order to use a baseline incidence or prevalence rate data with a particular health impact function, the **Endpoint Group** and **Endpoint** of the baseline incidence or prevalence rate must match the **Endpoint Group** and **Endpoint** of the health impact function exactly.

4.1.4.2 Format for Incidence/Prevalence Data

Table 4-7a presents the variables that can be used in incidence and prevalence datasets, and Table 4-7b presents a sample dataset that follows this format.

Table 4-7a. Health Incidence and Prevalence Dataset Variables

Field Name	Type	Required	Notes
Endpoint Group	Text	Yes	If this does not reference an already defined Endpoint Group, one will be added.
Endpoint	Text	Yes	If this does not reference an already defined Endpoint for the Endpoint Group, one will be added.
Race	Text	No	Should either be blank (signifying All Races) or reference a defined Race, such as "Black" (from one or more Population Configurations).
Ethnicity	Text	No	Should either be blank (signifying All Ethnicities) or reference a defined Ethnicity, such as "Hispanic" (from one or more Population Configurations).
Gender	Text	No	Should either be blank (signifying All Genders) or reference a defined Gender (from one or more Population Configurations).
Start Age	Integer	Yes	Specifies the low and high ages, inclusive. For example, Start Age of "0" and End Age of "1" include infants through the first 12 months of life and all one-year old infants.
End Age	Integer	Yes	
Column	Integer	Yes	The Column and the Row link the incidence/prevalence data with cells from a Grid Definition.
Row	Integer	Yes	
Value	Numeric (double)	Yes	The incidence/prevalence rate for the specified demographic group for this location.
Type	Text	No	If value is a prevalence rate, then "Prevalence" should be specified. Otherwise BenMAP-CE assumes that the value is an incidence rate.

Table 4-7b. Sample Health Incidence Dataset

Endpoint Group	Endpoint	Year	Race	Gender	Ethnicity	Start Age	End Age	Column	Row	Type	Value
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	2	Incidence	2.62E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	3	Incidence	7.73E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	4	Incidence	1.68E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	5	Incidence	5.45E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	6	Incidence	1.02E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	7	Incidence	2.72E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	8	Incidence	5.05E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	9	Incidence	3.03E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	10	Incidence	5.89E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	11	Incidence	3.11E-06
Hospital Admissions, Respiratory	HA, Asthma	2002				0	99	1	12	Incidence	3.30E-06

4.1.5 Population Data

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

Population data loaded into BenMAP-CE 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. Age ranges are defined by integer values for starting age and ending age (inclusive), and a unique text value representing the name of the age range. For example, 'OT01' 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 provided to BenMAP-CE should then contain population values for all combinations of race, ethnicity, gender, and age range. The population values may be non-integer values.

Fundamental Concept – Population Configuration

BenMAP-CE 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 ethnicity. To add population data in BenMAP-CE, you must first specify a **population configuration**. 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 you are planning to input. Stratifying your data by these categories is not required, but detailed population data allows you to more accurately estimate health impacts by better aligning your data with the study populations of health impact research. 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.

In the U.S. setup, BenMAP-CE uses 2010 U.S. Census block data to forecast the population in future years at various spatial resolutions. Sources of population data will vary by country and are generally available from the country's governmental agency responsible for collecting demographic and population data.

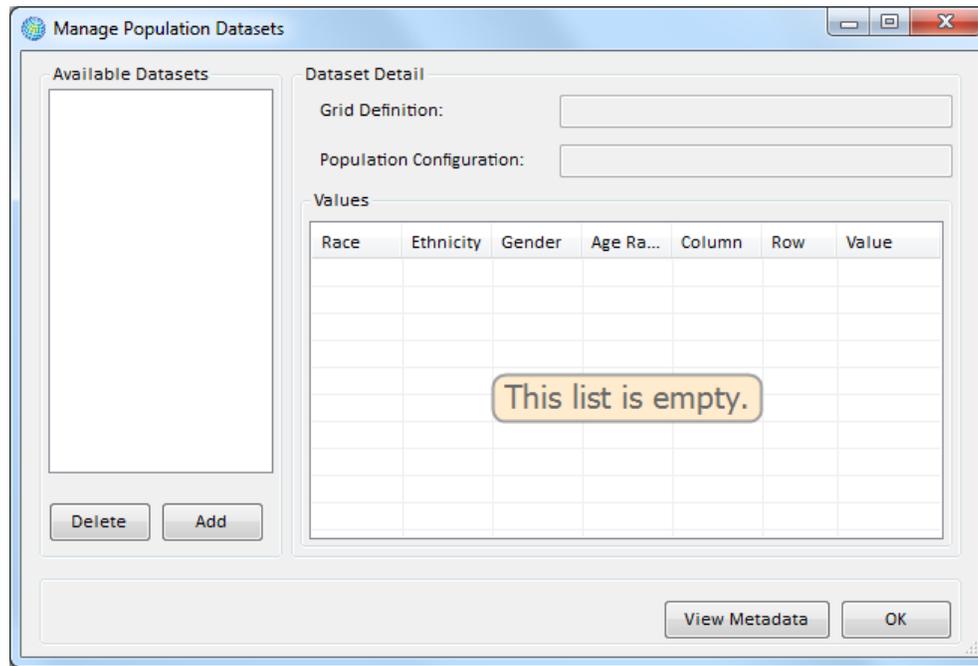
Population data must also be associated with a **Grid Definition** which specifies the geographic areas for which the data is available (see for more details the section on Grid Definitions). If population data is available for multiple grid definitions (cities and

neighborhoods, for example), you can have the option of using different sets of population data for different analyses.

BenMAP-CE can also estimate populations for Grid Definitions for which no population data is available by calculating spatial overlap percentages with Grid Definitions for which data is available.

4.1.5.1 Add Population Data

To add population data to BenMAP-CE, click on the **Manage** button below the **Population Datasets** box in the **Modify Datasets** window. The **Manage Population Datasets** window will appear.



Click on the **Add** button to display the **Load Population Dataset** window. Name the dataset using the **Population Dataset Name** box.

The **Grid Definition** drop-down list provides the list of existing grid definitions. Choose a grid definition that matches your population dataset.

The **Population Configuration** section allows you to define the variables that are in the population data file to be loaded into BenMAP-CE. Use the drop-down list to choose an existing population configuration and then view it by clicking the **View** button, or you may click the **Add** button and define a new population configuration. Clicking the **Add** button will open a **Population Configuration Definition** window where you can enter the fields that appear in the file that will be later uploaded (discussed in more detail below).

The **Browse** button to the right of the Database box allows you to find the data file that you want to load into BenMAP-CE. Once you click **Open** and load the file, the **Validate** and **View Metadata** buttons become active. You can click the **Validate** button before the file is loaded into the **Manage Population Datasets** form. BenMAP-CE's validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings. You can also add metadata, which includes references and descriptions, by clicking the **View Metadata** button.

If you wish to run an analysis based on an air quality grid not already defined in BenMAP-CE, you may need to import a new population file matched to that grid definition. The PopGrid program allocates the 2010 block-level U.S. Census population to a user-defined grid, creating a population file ready for importation to BenMAP-CE.¹⁷

The *Use Population Growth Weights* checkbox should be checked when using population data generated by the PopGrid software application. The population weights file assists in forecasting population levels. See Appendix J for a more detailed discussion of population growth weights in the *United States* setup.

¹⁷ The PopGrid program may be downloaded from EPA's website. See: <http://www2.epa.gov/benmap/benmap-community-edition>.

Defining a Population Configuration

If you are performing an analysis outside of the U.S. and are loading your own population data, you will need to first create a new **Population Configuration**. The Population Configuration defines the age range (**Start Age** and **End Age**), **Race**, **Ethnicity**, and **Gender** variables in your population database.

Check that your population variables align with the population configuration already defined for your setup (the spellings must match exactly). If your data fails to load correctly, you will need to go back and either develop a new population configuration to match your data, or you need to revise your population database so that it matches the population configuration.

Warning

It is critical that the age, race, ethnicity, and gender variables defined in the population configuration match your population input data **exactly**.

To add a population configuration, click the **Add** button in the **Load Population Dataset** window.

In the **Population Configuration Name** box, replace 'PopulationConfiguration0' with a name of your choosing. Under the **Races** box click on **New** and type in the name for any races present in your population data. The names appear in both the **Races** list box and

the **Available Races** list box. (If you later create alternative population configurations, you can simply drag the relevant names from the **Available Races** list box into the **Races** list box.) Similarly, under the **Available Genders** and **Available Ethnicity** list boxes, click on **New** and type in the name for any ethnicity and gender identifiers present in your population data.

If you want to remove a selected category from the **Races**, **Genders**, or **Ethnicity** list boxes, then highlight the category that you want to deselect and click the **Remove** button. (It is not possible to delete categories from the **Available Races**, **Available Genders**, or **Available Ethnicity** list boxes.)

The next step is to create the age ranges that match the age ranges in your population file. To start click on the **Add** button below the **Age Ranges** list box. The **AgeRange Definition** window will appear. You do not need to type anything in the **Age Range ID** box. You can enter the upper end of the age range into the **High Age** bound box, which will automatically populate the **Age Range ID**. The **Age Range ID** will be in the format **LowAgeTOHighAge** (e.g., 0TO0, 1TO17, 18TO25, etc.) For the next age range, BenMAP-CE will automatically fill in the value for the **Low Age** box based on the previous range you entered. For example, the age range names (with corresponding low and high ages) might include the following: *0to0, 1to4, 5to9, 10to14, 15to19, 20to24, 25to29, 30to34, 35to39, 40to44, 45to49, 50to54, 55to59, 60to64, 65to69, 70to74, 75to79, 80to84, and 85up*. You must be sure that the population configuration names exactly match those in your population input file.

The screenshot shows a dialog box titled "AgeRange Definiti...". It contains three text input fields: "Age Range ID" with the value "11TO20", "Low Age" with the value "11", and "High Age" with the value "20". An "OK" button is positioned at the bottom right of the dialog.

Click **OK** when you have defined the age range. If you make a mistake and want to delete an age definition after you have entered it, select the age range you would like to delete then click on the **Delete** button. The population configurations can be quite detailed, as in the case of the *United States Census* population configuration that comes loaded with BenMAP-CE.

Population Configuration Definition

Population Configuration Name:

Race

Available Races: ASIAN, BLACK, NATAMER, WHITE, ALL, Other, New

Races: ASIAN, BLACK, NATAMER, WHITE

Buttons: New, Remove

Gender

Available Genders: FEMALE, MALE, ALL

Genders: FEMALE, MALE

Buttons: New, Remove

Ethnicity

Available Ethnicity: NON-HISPANIC, HISPANIC, ALL

Ethnicities: NON-HISPANIC, HISPANIC

Buttons: New, Remove

Age Ranges

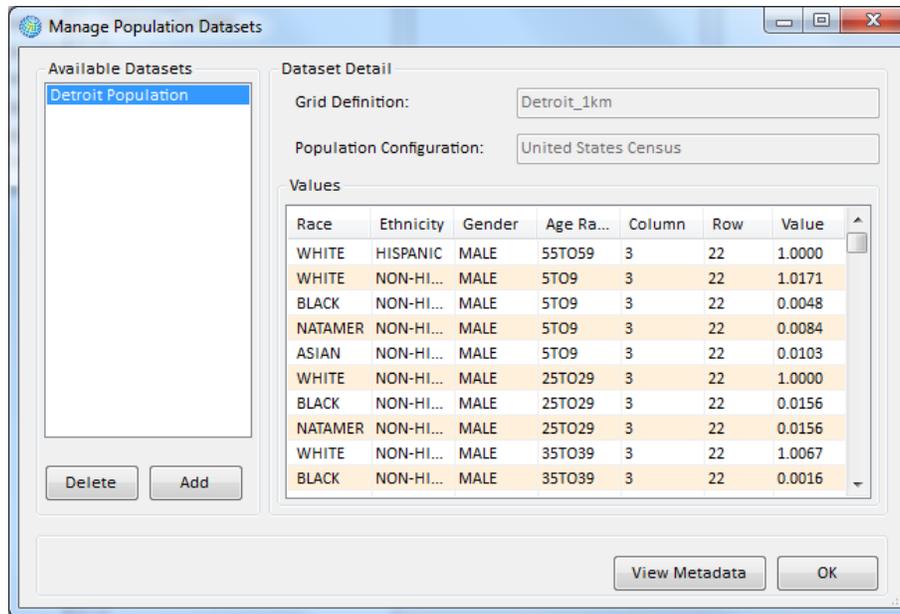
AgeRange	Start Age	End Age
0TO0	0	0
1TO4	1	4
5TO9	5	9
10TO14	10	14
15TO19	15	19
20TO24	20	24
25TO29	25	29
30TO34	30	34
35TO39	35	39
40TO44	40	44
45TO49	45	49
50TO54	50	54
55TO59	55	59
60TO64	60	64
65TO69	65	69
70TO74	70	74
75TO79	75	79
80TO84	80	84
85UP	85	99

Buttons: Delete, Add

Buttons: Cancel, OK

Click **OK** on the **Population Configuration Definition** window to return to the **Load Population Dataset** window.

Click **OK** on the **Load Population Dataset** window to return to the **Manage Population Datasets** window. To view or edit any metadata that was previously added, click the **View Metadata** button. Click **OK** on the **Manage Population Datasets** window. In the **Population Datasets** box of the **Modify Datasets** window you should see an entry for the population dataset that you just loaded.



4.1.5.2 Format for Population Data

Table 4-8 presents the variables that can be used in population datasets. Note that the names you define for age ranges do not need to follow the same pattern used in this manual; the age ranges should be based on what seems most appropriate for you. However, it is critical that the age, race, ethnicity, and gender variables in your population input data exactly match those defined for the population configuration, otherwise BenMAP-CE will fail to load the population data.

Table 4-8. Population Dataset Variables

Variable	Type	Required	Notes
AgeRange	Text	Yes	References a defined age range in the associated Population Configuration.
Column	Integer	Yes	The column and the row link the population data with cells in a Grid Definition.
Row	Integer	Yes	
Year	Integer	Yes	The year of the data. Note that this may include historical population estimates (such as from a census), as well as population forecasts (maximum value = 2499).
Population	Numeric (double)	Yes	Population estimate. Note that the estimate is not restricted to integers.
Race	Text	Yes	References a defined race in the associated Population Configuration.

Variable	Type	Required	Notes
Ethnicity	Text	Yes	References a defined ethnicity in the associated Population Configuration. If no ethnicity is specified in the data, “ALL” should be listed throughout the entire column.
Gender	Text	Yes	References a defined gender in the associated Population Configuration.

4.1.6 Health Impact Functions

Health impact functions calculate the change in the number of adverse health effects among a certain population associated with a change in exposure to air pollution. A typical health impact function has inputs specifying the pollutant; the metric (daily, seasonal, and/or annual); the age, race, ethnicity, and gender of the population affected; and the incidence rate of the adverse health effect.

Health impact functions are subdivided by user-specified types of adverse health effects. The broadest category is the **Endpoint Group**, which represents a broad class of adverse health effects, such as premature mortality, cardiovascular-related hospital admissions, and respiratory-related hospital admissions, among other categories. (BenMAP-CE only allows pooling of adverse health effects to occur within a given endpoint group, as it generally does not make sense to sum or average together the number of cases of disparate health effects, such as premature mortality and chronic bronchitis.) The **Endpoint Group** may then be subdivided by user-specified **Endpoints**.

Fundamental Concept – Endpoint Group

An **endpoint group** represents a broad class of similar or related adverse health effects, such as premature mortality or cardiovascular hospital admissions. An **endpoint** is an individual adverse health outcome or subclass of diagnoses within an endpoint group, identified by one or more codes in the International Classification of Disease system. For example, within the endpoint group *Mortality*, there might be the endpoints *Mortality, Long Term, Lung Cancer* and *Mortality, Long Term, Cardiopulmonary* to distinguish annual mortality impacts with different causes of death.

Fundamental Concept: Health Impact Function or Concentration-Response Function

A **health impact function** calculates 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 (using a specified metric such as annual D24HourMean); 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 (β) and is derived from epidemiological studies. The value of β typically represents the percent change in a given adverse health impact per unit of pollution.

Health impact functions are derived from **concentration-response (C-R) functions**, 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.

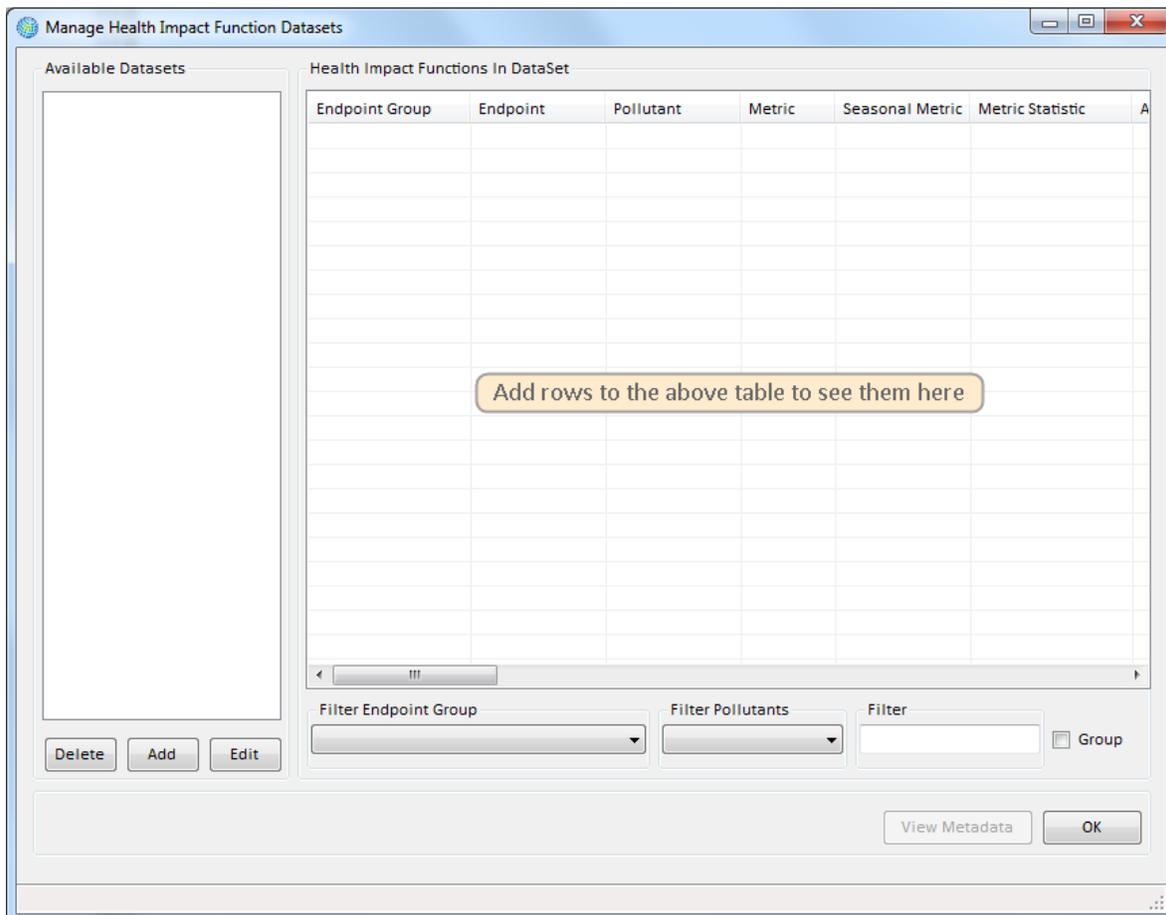
For example, the respiratory-related hospital admission **Endpoint Group**, may have separate **Endpoints** for asthma-related hospital admissions and chronic bronchitis-related hospital admissions.

There are a wide range of variables that can be included in a health impact function, to specify the parameters of the function and to identify its source, such as the **Author**, **Year**, and **Location** of the study, as well as

other **Pollutants** used in the study. The bibliographic **Reference** for the study may be included, as well as any additional information needed to identify a particular impact function. (The **Reference** variables are useful for this.) A number of health impact functions have been developed based on epidemiological studies in the United States and Europe. However, researchers have conducted an increasing number of epidemiological studies in Asia and Latin America that can be used to develop more location-specific impact functions. There are a number of issues that arise when deriving and choosing between health impact functions that go well beyond this user manual. *Hence, it is important to have a trained health researcher assist in developing the impact function data file.*¹⁸

4.1.6.1 Add Health Impact Functions

To add health impact functions to BenMAP-CE, click on the **Manage** button below the **Health Impact Functions** box in the **Modify Datasets** window. The **Manage Health Impact Functions Datasets** window will appear.



¹⁸ U.S. EPA-default configuration and pooling setup files for ozone and PM_{2.5} health impact assessments are available on the BenMAP-CE website. See: <http://www.epa.gov/benmap/benmap-community-edition>.

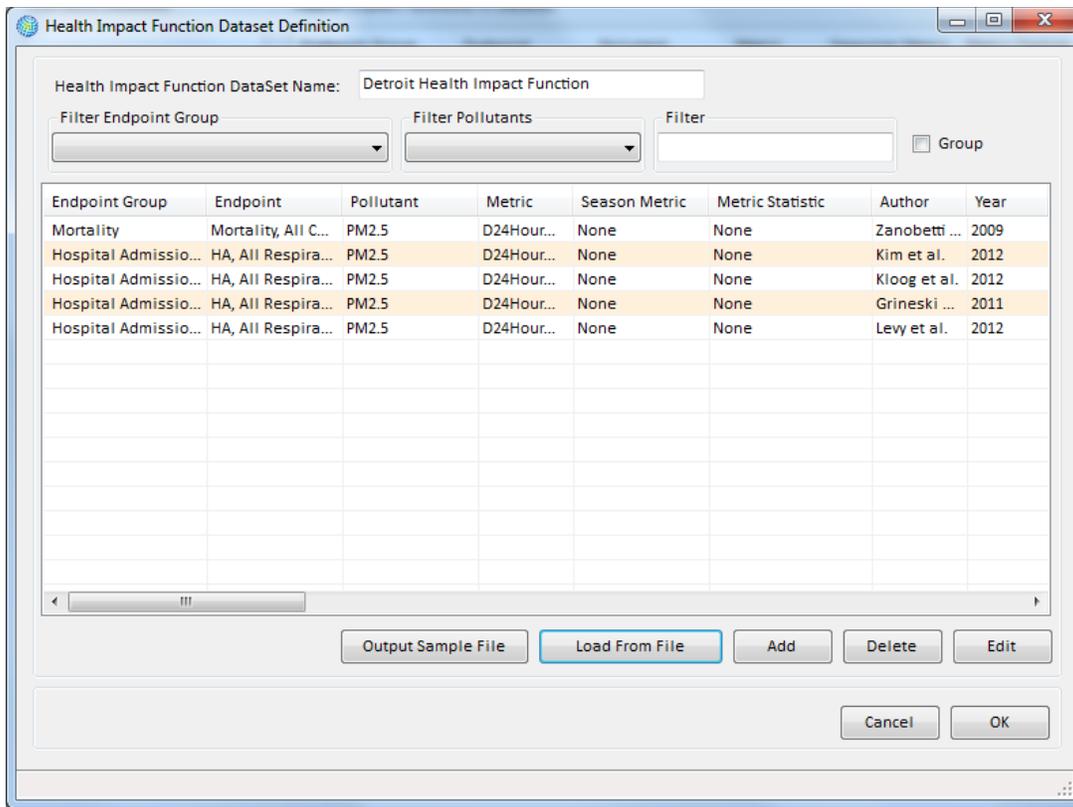
In this window you may **Add**, **Edit**, and **Delete** datasets. The section on the left under **Available Datasets** lists the health impact function datasets that are currently in the setup database. (See Appendices E, F, and G for more information about the pre-loaded health impact functions.) The section on the right under the **Health Impact Functions in Dataset** lets you view the functions in a selected dataset.

To add a new dataset, click the **Add** button. The **Health Impact Function Dataset Definition** window will appear. Type the name that you want to use for the dataset in the **Health Impact Function Dataset Name** box.

You may then enter functions into this dataset through an externally created database by clicking the **Load From File** button. Alternatively, you may **Add**, **Delete**, and **Edit** individual functions within BenMAP-CE.

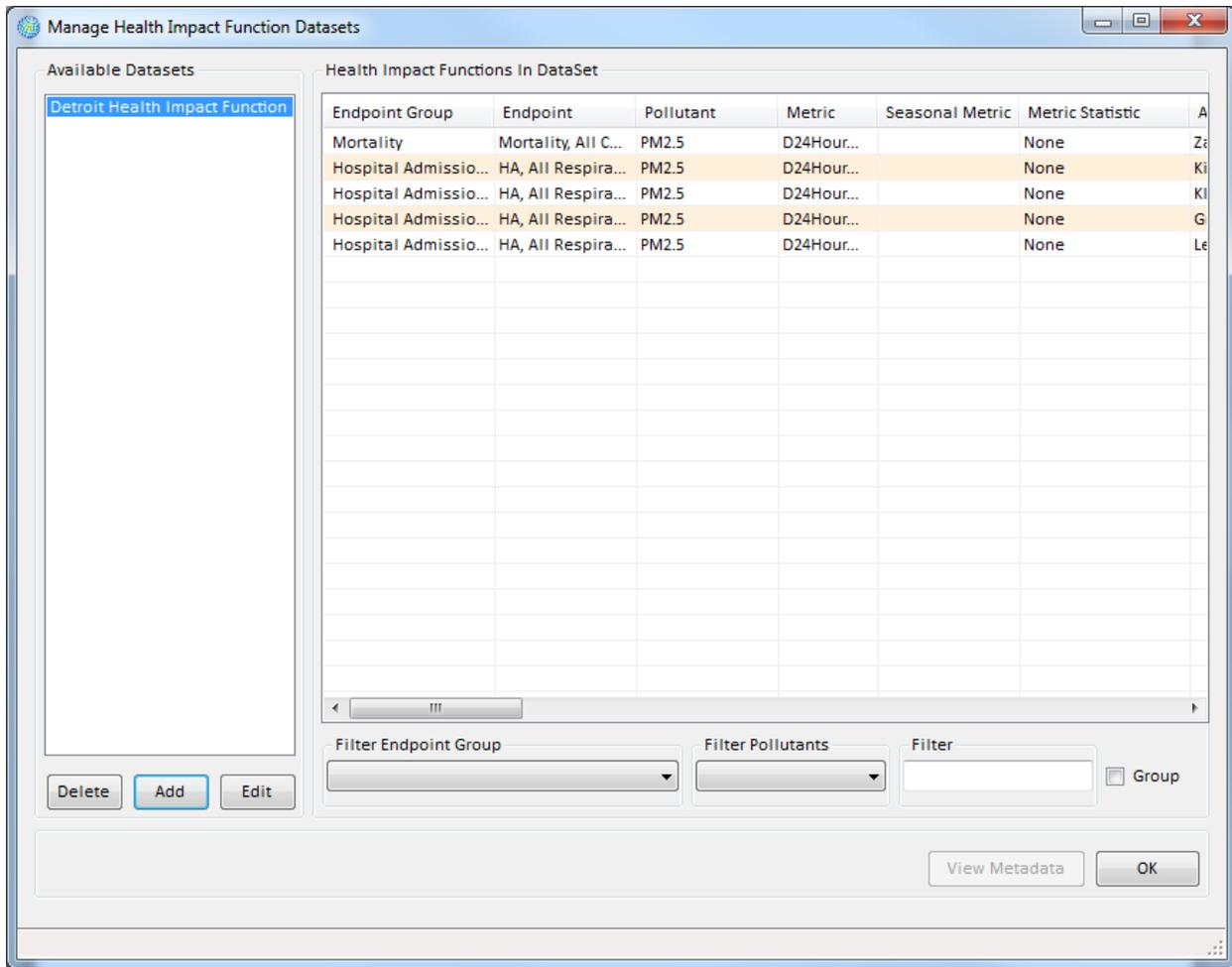
To add a database, click the **Load From File** button. In the **Load Health Impact Dataset** window, click the **Browse** button and then find and select the health impact function database that you want to load into your setup. Click **Open**. If validation is required, then you will have to click the **Validation** button before the file can be imported. BenMAP-CE's validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings. You can also add metadata (references and descriptions) to the file that is about to be imported by clicking the **View Metadata** button. Click the **OK** button on the **Load Health Impact Dataset** window to load the dataset.

The **Health Impact Function Dataset Definition** window will reappear, and you can then view the health impact functions that you have loaded into your dataset.



By clicking and holding the cursor on a column header, you may move it to provide the most useful display. For example, by clicking and holding on the **Pollutant** column header and then dragging it to the far left of the window, you can sort all of the health impact functions by **Pollutant**. (Note rearranging the columns is only for display and has no effect on the underlying health impact functions in the database.)

Clicking **OK** brings you back to the **Manage Health Impact Function Datasets** window. The new dataset you just loaded will be displayed in the list of **Available Datasets** and the associated functions will be displayed in the **Health Impact Functions In Dataset** grid to the right. If you have more than one dataset, you can select the dataset by clicking on it.



To edit an existing function, first click to select the dataset in the list of **Available Datasets**. Next, select a particular function in the data grid under **Health Impact Functions in Dataset**. Then, click the **Edit** button. The **Health Impact Function Dataset Definition** window appears, and you may change any of the values in the boxes and the drop-down lists. When you are finished, click **OK**.

From the **Health Impact Function Dataset Definition** window you can also add health impact functions to the ones that are already in your dataset. Click the **Add** button and a blank **Health Impact Function Definition** window will appear and you can then create new health impact functions. (See Appendix M: Function Editor for additional information about the syntax for developing functions with this editor.)

Starting in Version 1.4, you can link a health impact function to a specific geographic area for which you have uploaded a grid definition by selecting that area in the **Apply Function To** dropdown menu. This prevents you from applying the function to other grid definitions but may be appropriate for functions derived using sub-national data. By default this menu is set to “Entire Area”, which means the application of the health impact function is unrestricted. Note that if an air quality cell intersects multiple geographic areas, BenMAP-CE will calculate health impacts for the geographic area containing the majority of the air quality cell.

After defining the new health impact function, click **OK**. This will take you back to the **Health Impact Function Dataset Definition** window. When you are finished with any

editing or adding of health impact functions, click **OK**. From the **Manage Health Impact Function Datasets** window, you can also select an **Available Dataset** and **Data row** and view the Metadata. To view the Metadata associated with the data file, click the **View Metadata** button to view and edit existing references and descriptions. Click **OK** on the **Manage Health Impact Function Datasets** window when you are satisfied with all your inputs. The **Modify Datasets** window will appear. Here in the **Health Impact Functions** box you should see an entry for any health impact function datasets that you have loaded.

4.1.6.2 Format for Health Impact Functions

Table 4-9 presents the variables that can be used in health impact function datasets.

Table 4-9. Health Impact Function Dataset Variables

Variable	Type	Required	Notes
Endpoint Group	Text	Yes	If this does not reference an already defined Endpoint Group, one will be added.
Endpoint	Text	Yes	If this does not reference an already defined Endpoint for the Endpoint Group, one will be added.
Pollutant	Text	Yes	Should reference an already defined Pollutant.
Metric	Text	Yes	Should reference an already defined Metric for the Pollutant.
Annual Statistic	Text	No	Should either be blank (signifying no annual metric value) or be one of: None, Mean, Median, Min, Max, Sum.
Seasonal Metric	Text	No	Should either be blank (signifying no Seasonal Metric value) or reference an already defined Seasonal Metric for the Metric.
Race		No	Should either be blank (signifying All Races) or reference a defined Race.
Ethnicity		No	Should either be blank (signifying All Ethnicities) or reference a defined Ethnicity.
Gender		No	Should either be blank (signifying All Genders) or reference a defined Gender.
Start Age	Integer	Yes	Specifies the low and high ages, inclusive. For example, Start Age of '0' and End Age of '1' includes infants through the first 12 months of life and all one-year old infants.
End Age	Integer	Yes	
Author	Text	No	The author(s) of the study from which the function is derived.
Apply Function To		No	The specific geographic area to which you would like to apply the health impact function; unrestricted by default ("Entire Area")

Variable	Type	Required	Notes
Year of Publication	Integer	Yes	The year of publication of the study.
Qualifier	Text	No	Provides additional information to identify a particular health impact function, such as when a particular study has multiple functions.
Location Name		No	Type of study area. For the ‘United States’ setup, choose between ‘State’, ‘County’, and ‘MSA (metropolitan area)’.
Location	Text	No	The specific location of the study.
Co-Pollutants Specified in Regression Model	Text	No	Identifies other pollutants that were included simultaneously in the estimation equation for the pollutant of interest.
Reference	Text	No	Bibliographic reference, included to identify the source in the health literature.
Function	Text	Yes	The functional form, interpreted (executed) by BenMAP-CE when running an analysis to estimate air pollution-related health impacts. For example, the log-linear form is as follows: $(1 - (1 / \text{EXP}(\text{Beta} * \text{DELTAQ}))) * \text{Incidence} * \text{POP}$.
Baseline Incidence Function	Text	Yes	The functional form, interpreted (executed) by BenMAP-CE to estimate health impacts due to all causes. This typically has the form: $\text{Incidence} * \text{POP}$.
Beta Distribution	Text	No	If the Beta has no distribution, any value is acceptable. Otherwise, should be one of: <i>Normal, Triangular, Poisson, Binomial, LogNormal, Uniform, Exponential, Geometric, Weibull, Gamma, Logistic, Beta, Pareto, Cauchy, Custom</i> .
Beta	Numeric (double)	No	Mean value of the Beta distribution.
Beta Parameter 1	Numeric (double)	No	Parameter 1 of the Beta distribution (meaning depends on the distribution - for Normal distributions this represents the standard deviation).
Beta Parameter 2	Numeric (double)	No	Parameter 2 of the Beta distribution (meaning depends on the distribution - for Normal distributions this is not required).
Name A	Text	No	Description of variable A.
A	Numeric (double)	No	A constant value which can be referenced by the Function.
Name B	Text	No	Description of variable B.
B	Numeric (double)	No	A constant value which can be referenced by the Function.

Variable	Type	Required	Notes
Name C	Text	No	Description of variable C.
C	Numeric (double)	No	A constant value which can be referenced by the Function.
Incidence Dataset	Text	No	Specifies the dataset from which incidence data will be derived. The user may choose from multiple datasets. (Initially this field may be left blank.) See Section 4.1.4.
Prevalence Dataset	Text	No	Specifies the dataset from which prevalence data will be derived. The user may choose from multiple datasets. (Initially this field may be left blank.) See Section 4.1.4.
Variable Dataset	Text	No	Specifies the dataset from which "variable" data (e.g., income data) will be derived. The user may choose from multiple datasets. (Initially this field may be left blank.) See Section 4.1.7.

While the mean value of the Beta distribution is always available in BenMAP-CE, the values associated with Beta Parameter 1 and Beta Parameter 2 may change depending on the type of distribution. Table 4-10 provides a list of the variables associated with each type of beta distribution in BenMAP-CE.

Table 4-10. Beta Distribution Types and Variables

Distribution	Formula	Beta Parameter 1	Beta Parameter 2	Notes
Normal	$\frac{1}{\sigma\sqrt{2\pi}} \times e^{-\frac{(x-\mu)^2}{2\sigma^2}}$	Standard deviation (sigma)	N/A	The Normal distribution has two parameters - the mean, mu, and the standard deviation, sigma.
Triangular	$\frac{2(x-a)}{(b-a)(c-a)}$ for $a \leq x \leq c$ $\frac{2(b-x)}{(b-a)(b-c)}$ for $c \leq x \leq b$	Minimum value (a)	Maximum value (b)	The Triangular distribution has three parameters - the minimum value (a), the maximum value (b), and the most likely value (c). BenMAP-CE uses the mean value, the minimum, and the maximum to calculate the most likely value.
Poisson	$\lambda^x e^{-\lambda} / x!$	Lambda	N/A	The Poisson distribution has a single parameter, lambda.
Binomial	$\frac{n!}{x!(n-x)!} p^x (1-p)^{n-x}$	n	p	The Binomial distribution has two parameters, n and p.
LogNormal	$1/\sigma\sqrt{2\pi} \cdot e^{-(x-\mu)^2/2\sigma^2}$	Standard deviation (sigma) of the corresponding	N/A	The LogNormal distribution has two parameters - the mean of the corresponding Normal distribution, mu, and the

Distribution	Formula	Beta Parameter 1	Beta Parameter 2	Notes
		Normal distribution		standard deviation of the corresponding Normal distribution, sigma.
Uniform	$1/(B - A)$ for $A < x < B$	A	B	The Uniform distribution has two parameters, A and B, which define the interval on which the distribution is defined.
Exponential	$\frac{1}{\mu} e^{-x/\mu}$	Mu	N/A	The Exponential distribution has one parameter, mu.
Geometric	$p(1 - p)^x$ for $0 < p < 1$	p	N/A	The Geometric distribution has one parameter, p.
Weibull	$\left(\frac{\beta}{\alpha}\right)\left(\frac{x}{\alpha}\right)^{\beta-1} e^{-(x/\alpha)^\beta}$	alpha	beta	The Weibull distribution has two parameters, alpha and beta.
Gamma	$\frac{1}{b^a \Gamma(a)} x^{a-1} e^{-x/b}$	a	b	The Gamma distribution has two parameters, a and b.
Logistic	$\frac{e^{(x-m)/b}}{b[1 + e^{(x-m)/b}]^2}$	m	b	The Logistic distribution has two parameters, m and b.
Beta	$\frac{1}{\beta(a, b)} x^{a-1} (1-x)^{b-1}$	a	b	The Beta distribution has two parameters, a and b.
Pareto	ab^a/x^{a+1}	a	b	The Pareto distribution has two parameters, a and b.
Cauchy	$\frac{1}{\pi} \cdot \frac{b}{(x-m)^2 + b^2}$	b	m	The Cauchy distribution has two parameters, b and m.
Custom	N/A	Standard deviation	N/A	The custom distribution is used for specified expert distributions for pollutant effect (e.g., truncated parametric distributions or non-parametric distributions).

For the **Function** definition, commonly used mathematical operators (+, -, *, /) may be used. Other available “**Operators**” are listed to the right of the commonly used functional forms (e.g., ABS(x), EXP(x), LOG(x)). These operators are supported by the math and statistics library used within BenMAP-CE.

Under the heading “**Available Variables**”, are temporary runtime variables which can be used in the health impact function definition. The values for these variables (except for the A, B, C constants) are expected to change as the code loops over grid cells and stratified population groups. Table 4-11 presents the available runtime variables that can be used in health impact function definition.

Table 4-11. Health Impact Function Available Runtime Variables

Variable	Type	Notes
Beta	Numeric	Beta coefficient
DELTAQ	Numeric	Change in pollutant concentration (baseline – control)
POP	Numeric	Population
Incidence	Numeric	Number of people with new (or newly diagnosed) adverse health effects within a given period of time.
Prevalence	Numeric	Number of people who already have a given adverse health condition (i.e., chronic illness).
Q0	Numeric	Control pollutant concentration
Q1	Numeric	Baseline pollutant concentration
A	Numeric	User-defined constant
B	Numeric	User-defined constant
C	Numeric	User-defined constant

The “**Population Variables (optional)**” box provides a list of setup variables which have been defined under the **Variable Datasets** (see **Section 4.1.7**).

4.1.7 Variable Data

Health Impact Functions and **Valuation Functions** may sometimes refer to socioeconomic variables for which BenMAP-CE does not automatically calculate values.

Fundamental Concept – Variable Data

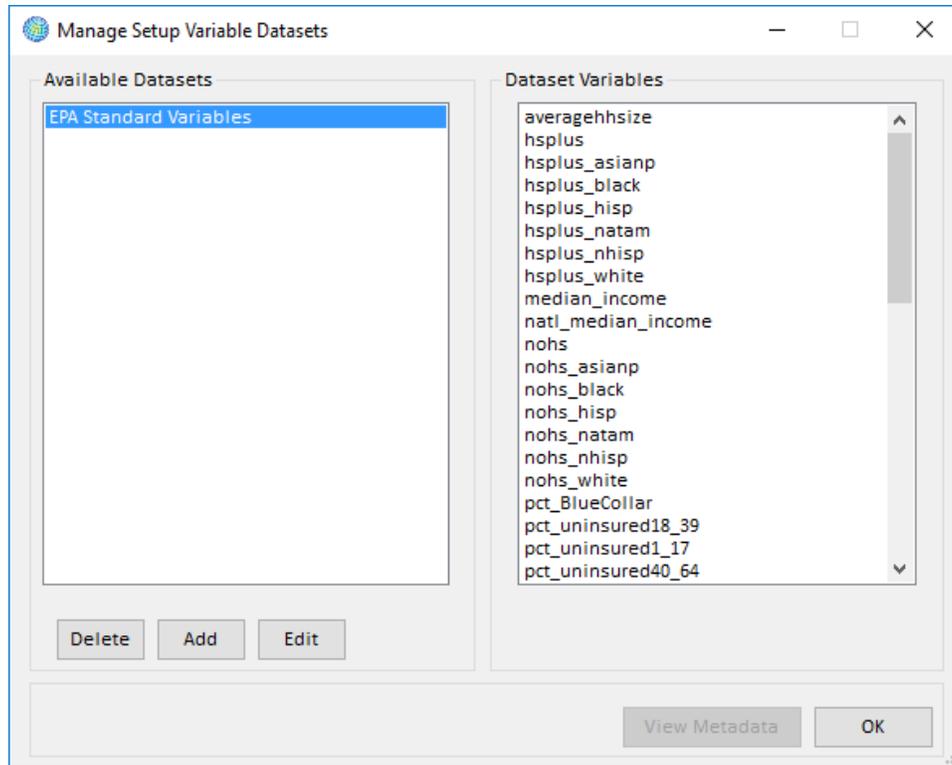
A **Variable Data** dataset contains sociodemographic and economic data not contained elsewhere that may be needed for certain health impact or valuation functions, or for certain types of analysis such as environmental justice studies. Examples of socioeconomic variables that might be included in **Variable Data** include median household income and percentage of population living below the poverty line.

Variables included in the dataset must be associated with a particular **Grid Definition**. Data at different spatial scales (e.g., county versus state) need to be input separately using different Variable Data files, each associated with the relevant grid definition.

For example, some valuation functions reference the median income within each area of analysis. Other functions apply to populations living below the poverty line in a given country. To facilitate this type of analysis, BenMAP-CE allows you to load datasets of socioeconomic **Variables**, which apply either globally or to a specific geographic area (i.e., they are associated with a **Grid Definition**).

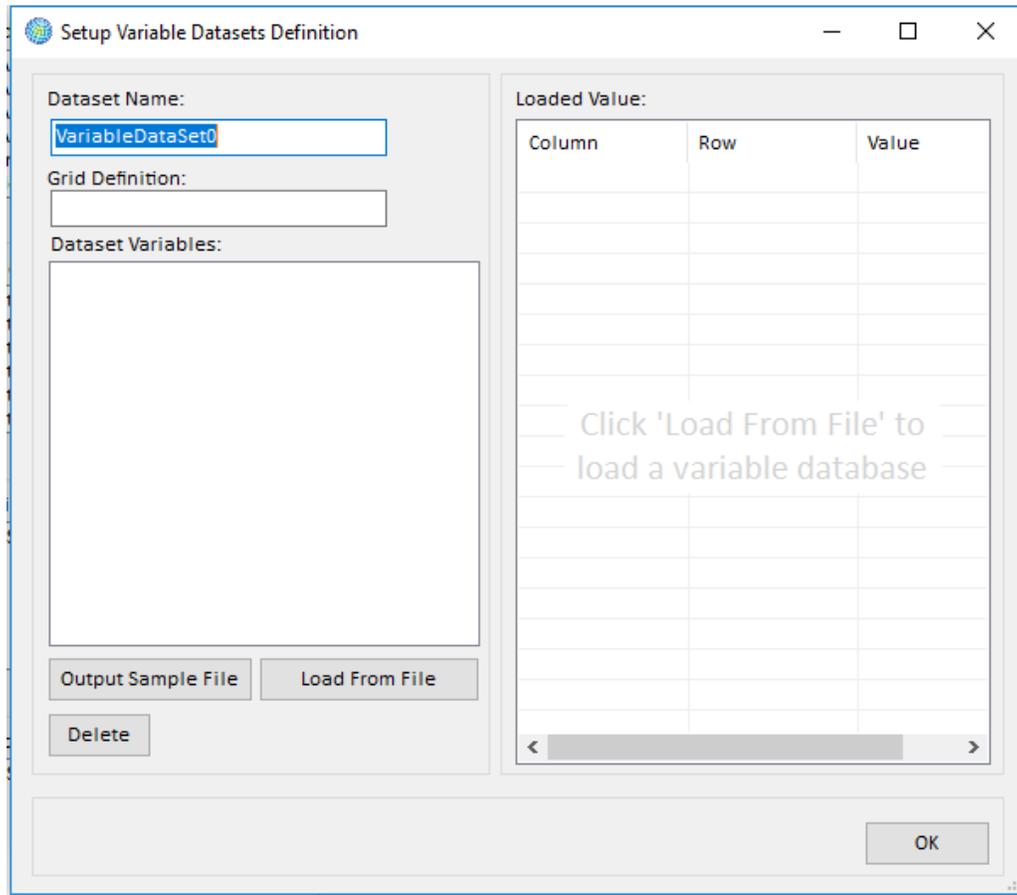
4.1.7.1 Add Variable Data

To add **Dataset Variables** to BenMAP-CE (such as income and other miscellaneous variables that might be needed in the analysis), click on the **Manage** button below the **Variables Datasets** box in the **Modify Datasets** window. The **Manage Setup Variable Datasets** window will appear.



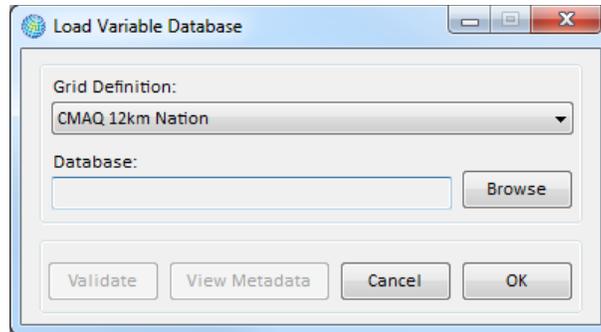
In this window you may **Add**, **Edit**, and **Delete** datasets. The section on the left under **Available Datasets** lists the variables datasets that are currently in the setup database. The section on the right under the **Dataset Variables** lets you view the variables in a selected dataset.

To add a **Variable** dataset click the **Add** button. This will take you to the **Setup Variable Dataset Definition** window. In this window you may add externally created variables through the **Load From File** button for any of your predefined **Grid Definitions**.

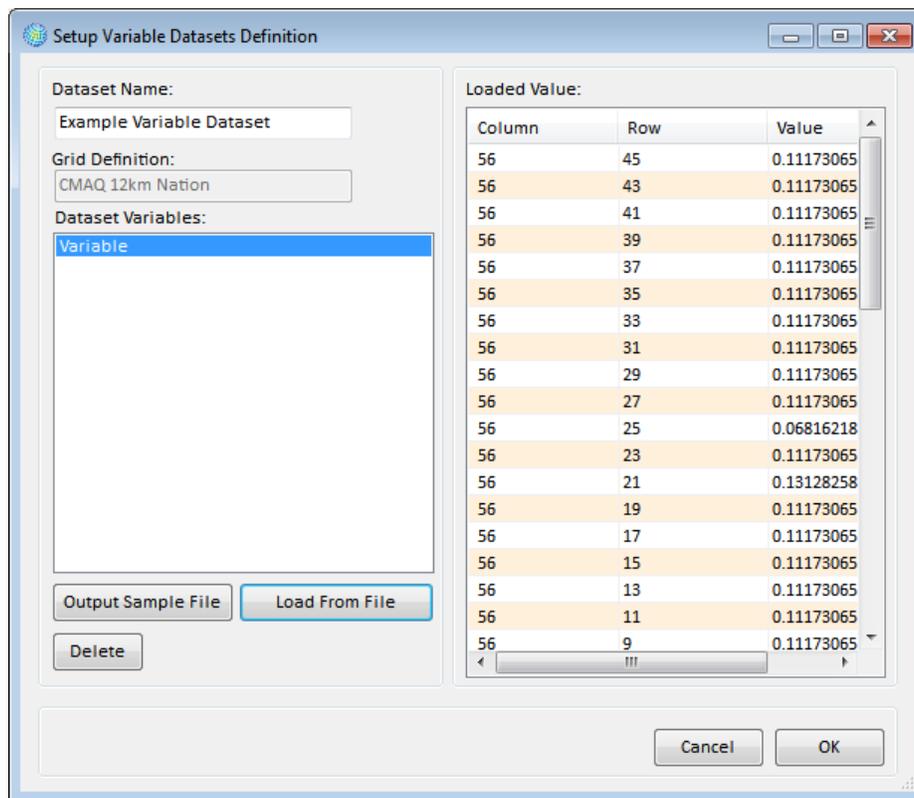


To start, type the name that you want to use for the **Variable Dataset** in the **Dataset Name** box. (This is a name that is internal to BenMAP-CE and used just for identification.)

To add an externally created **Variable Dataset**, click the **Load From File** button. This will bring up the **Load Variable Database** window. Here you need to choose the grid definition from the **Grid Definition** drop-down list that matches the level of aggregation in your variable data file. Remember that the **Variable Dataset** you import must use the same column/row index as the **Grid Definition**. Next, you may use the **Browse** button to find and select the desired **Database** (i.e., input file) and click **Open**. You can click the **Validate** button to ensure the file is properly formatted before importing. BenMAP-CE's validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings. You can also add metadata to the imported data (reference and description of the data file) by clicking the **View Metadata** button.

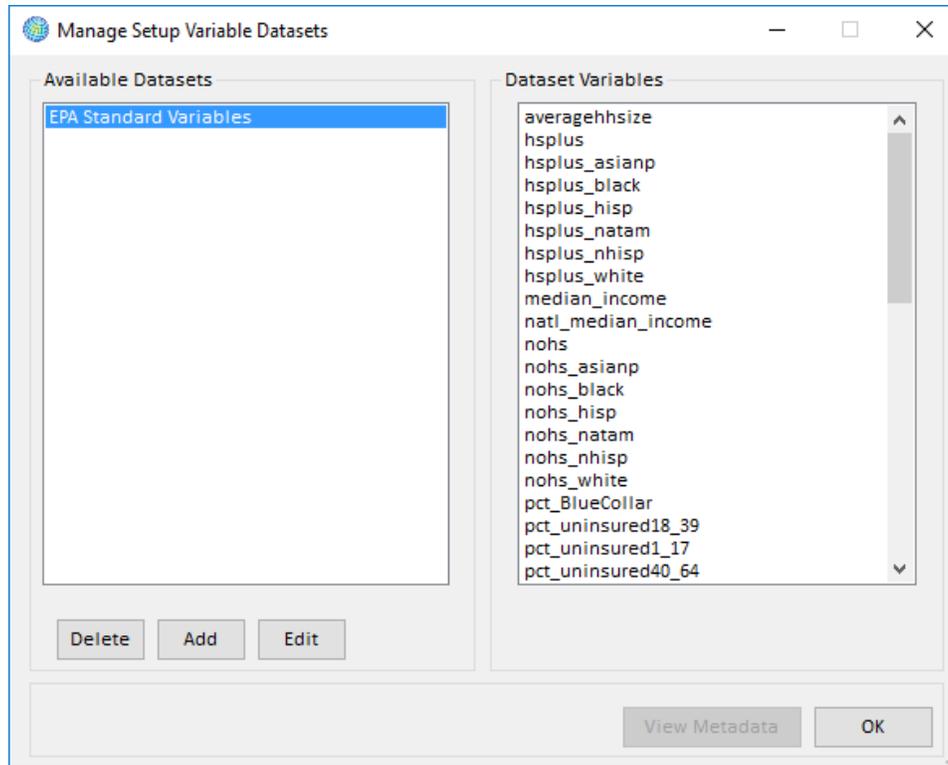


After choosing (and validating) the input file, click **OK**. This takes you back to the **Setup Variable Datasets Definition** window. This window displays the variables in the dataset and their values.



When finished adding variables, click **OK**. This will take you to the **Manage Setup Variable Datasets** window.

In the **Available Datasets** list box there is an entry for the dataset that you just added. And in the **Dataset Variables** list box are the variables in the highlighted dataset.



At this point you may click **Add** to load an additional dataset, click **Edit** to edit the selected dataset, click **Delete** to delete the selected dataset, or complete this variable management step by clicking **OK**.

Clicking **OK** returns you to the **Modify Datasets** window, where the entry for the variable dataset that you just entered should be visible under the **Variable Datasets** box.

4.1.7.2 Format for Variable Data

Table 4-12a presents the variables that can be used in variable datasets, and Table 4-12b presents a sample of what a dataset might look like. Note that if you are loading your own variable data, you can choose your own variable names (July 2018: this feature is currently disabled. Users should currently specify one variable per file).

Table 4-12a. Variable Dataset Variables

Variable	Type	Required	Notes
Column	Integer	Yes	The column and the row link the population data with cells in a Grid Definition.
Row	Integer	Yes	
Median_Income	Numeric (double)	Yes	Example: Median income value.
<Variable Name>	Numeric (double)	No	Additional variables may be specified.

Table 4-12b. Sample Variable Dataset

Column	Row	Median_Income
42	17	39871.19
42	29	43760.31
42	45	38186.77
42	91	42298.49
42	101	31261.07

4.1.8 Inflation Data

It may be desirable for the economic values generated by **Valuation Functions** to account for inflation and generate economic benefits using currency for years other than the year initially specified by your valuation data. To do this, you can load **Inflation Datasets** into BenMAP-CE, and then include a reference to your inflation data when developing valuation functions. (We give an example of this below.)

Fundamental Concept – Inflation Dataset

Inflation Datasets allow BenMAP-CE to account for changes in prices over time when presenting valuation estimates for health benefits. This dataset allows the user to specify valuation results in a currency year different from that reported in the valuation function applied. For example, you might use valuation data specified in 2015 dollars yet want to report the BenMAP-CE valuation estimate in 2020 dollars to compare to a cost analysis reporting costs in 2020 dollars. The inflation indices in this table are used to automatically convert currency to the dollar year the user selects when performing an analysis.

The **Valuation Functions** should have a consistent currency year, and this currency year has to be kept in mind when developing the inflation datasets. That is, whichever currency year is used for your valuation functions, then the inflation values for that year should be set to 1. For example, in the *United States* setup, the valuation functions have a dollar year of 2015, so the inflation datasets have a value of 1 for the year 2015. (Values for years earlier than 2015 are less than 1, and values for years after 2015 are greater than 1, because inflation has increased from one year to the next.) The *United States* setup in BenMAP-CE provides inflation factors for three different types of values:

- **All Goods Index** can be used to adjust the value of generic goods.
- **Medical Cost Index** can be used to adjust the value of medical expenses.
- **Wage Index** can be used to adjust the value of wages.

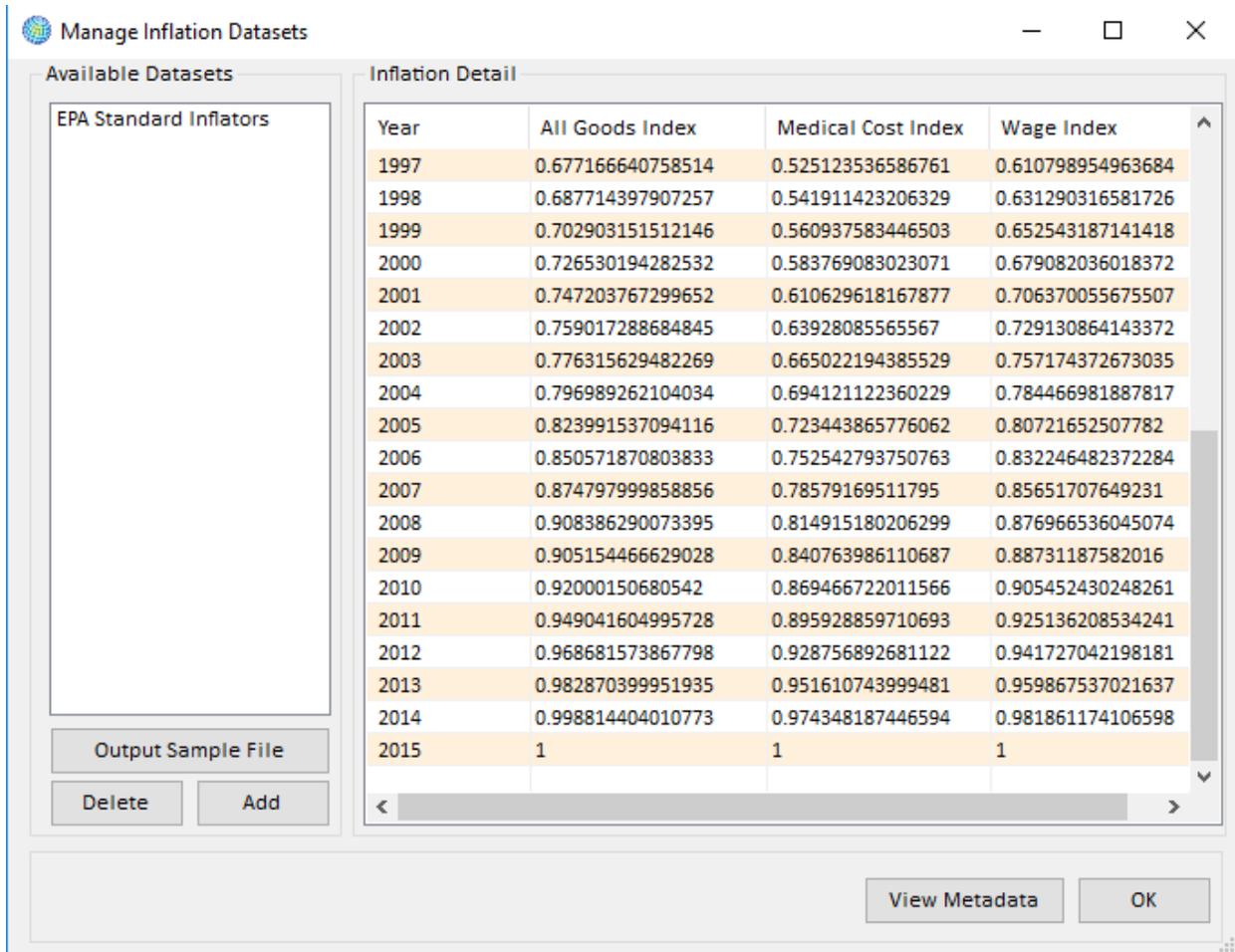
If a **Valuation Function** includes an estimate of wage income, for example, this value could be multiplied by the **Wage Index** adjustment factor to get the specified currency year. For example, in valuing work loss days, the *United States* setup uses a function like the following: $DailyWage * WageIndex$, where the *DailyWage* is specified in year 2015 dollars. In the **Inflation Dataset**, the **WageIndex** scales this **DailyWage** value up or down depending on the currency **Year** you have chosen. If the currency **Year** is 2015, then the **WageIndex** has a value of 1 and no change is made to the **DailyWage**. If the

currency **Year** is specified to, say, 2020, then the **WageIndex** will have a value greater than 1 because of the inflation that has occurred between 2015 and 2020.

4.1.8.1. Add Inflation Data

To add inflation data to BenMAP-CE, click on the **Manage** button below the **Inflation Datasets** box in the **Modify Datasets** window. The **Manage Inflation Datasets** window will appear. In this window you may **Add**, **Edit**, and **Delete** datasets. The section on the left under **Available Datasets** lists the **Inflation Datasets** that are currently in the setup database. The section on the right under **Inflation Detail** presents the inflation factors in a selected dataset.

Click on the **Add** button. In the **Load Inflation Dataset** window, type in the name of the dataset in the **Inflation Dataset Name** box, and then click on the **Browse** button to the right of the **Database** box to choose the dataset that you want to import and click **Open**. You can click the **Validate** button to ensure the file is properly formatted before importing. BenMAP-CE's validation tool will review the file format (column names, required columns, and data types) and provide a report with any errors or warnings. In addition, you can add metadata to the file, to include references and descriptions, by clicking the **View Metadata** button. Click **OK**. The **Manage Inflation Datasets** window will appear. Here you may view the data that you just loaded.



At this point you may add more data by clicking **Add**, or you may view and edit the Metadata for a specific dataset. This is done by selecting an **Available Dataset** and an entry in the **Inflation Detail** box and then clicking the **View Metadata** button. If you are satisfied with all import data, you can complete this step by clicking **OK**. Clicking **OK** takes you to the **Modify Datasets** window, where you should see an entry for the inflation dataset that you just loaded under the **Inflation Datasets** box.

4.1.8.2 Format for Inflation Data

Table 4-13a presents the variables that can be used in **Inflation Datasets**, and Table 4-13b presents a sample of what a dataset might look like. Note that if you are loading your own inflation data, you can use different names than the ones specified below. Instead of specifying *AllGoodsIndex* you could have a variable called *General Index* — this is fine as long as you make sure that your valuation functions properly reference these inflation variables.

Table 4-13a. Inflation Dataset Variables in U.S. Setup

Variable	Type	Required	Notes
Year	Integer	Yes	The year of the data. Note that this will typically only include historical estimates.
AllGoodsIndex	Integer	No	Example: All goods inflation index value.
MedicalCostIndex	Integer	No	Example: Medical cost inflation index value.
WageIndex	Integer	No	Example: Wage inflation index value.
<Variable Name>	Integer	No	Additional indices can be specified.

Table 4-13b. Sample Inflation Dataset

YEAR	AllGoodsIndex	MedicalCostIndex	WageIndex
2005	0.75	0.62	0.71
2006	0.79	0.67	0.74
2007	0.81	0.72	0.76
2008	0.83	0.77	0.78
2009	0.86	0.8	0.81
2010	0.88	0.84	0.83
2011	0.91	0.87	0.86
2012	0.93	0.89	0.89
2013	0.94	0.92	0.92
2014	0.96	0.96	0.96
2015	1	1	1
2016	1.02	1.04	1.03

4.1.9 Valuation Data

BenMAP-CE allows the valuation estimates to vary by **Endpoint Group, Endpoint, and Age** (note that multiple estimates may be provided for a particular combination). BenMAP-CE allows the valuation function to be quite detailed, and allows an uncertain parameter (A) with a user-specified distribution. You can modify the valuation function with a number of constant values (B, C, and D) that might represent an adjustment for inflation, income growth, income elasticity, or, say, purchasing power parity. Finally, BenMAP-CE has two fields to more clearly identify the valuation function (i.e., **Qualifier and Reference**).

When reviewing the economic literature to develop a valuation database or to simply add valuation functions to an existing database, it is important to have an economist assist. For an overview of valuation, see the Overview of Valuation section in Chapter 7: Aggregating, Pooling, and Valuing.

Fundamental Concept – Valuation

Valuation Functions are used by BenMAP-CE 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 endpoints, BenMAP-CE estimates monetized benefits by using either **Willingness to Pay (WTP)** or **Cost of Illness (COI)**-based valuation functions. **WTP** reflects the willingness of individuals to exchange money for a reduction in his or her risk of illness or death and is viewed by economists as the most complete and appropriate measure of the value of a risk reduction. **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 does not include the value of avoided pain and suffering.

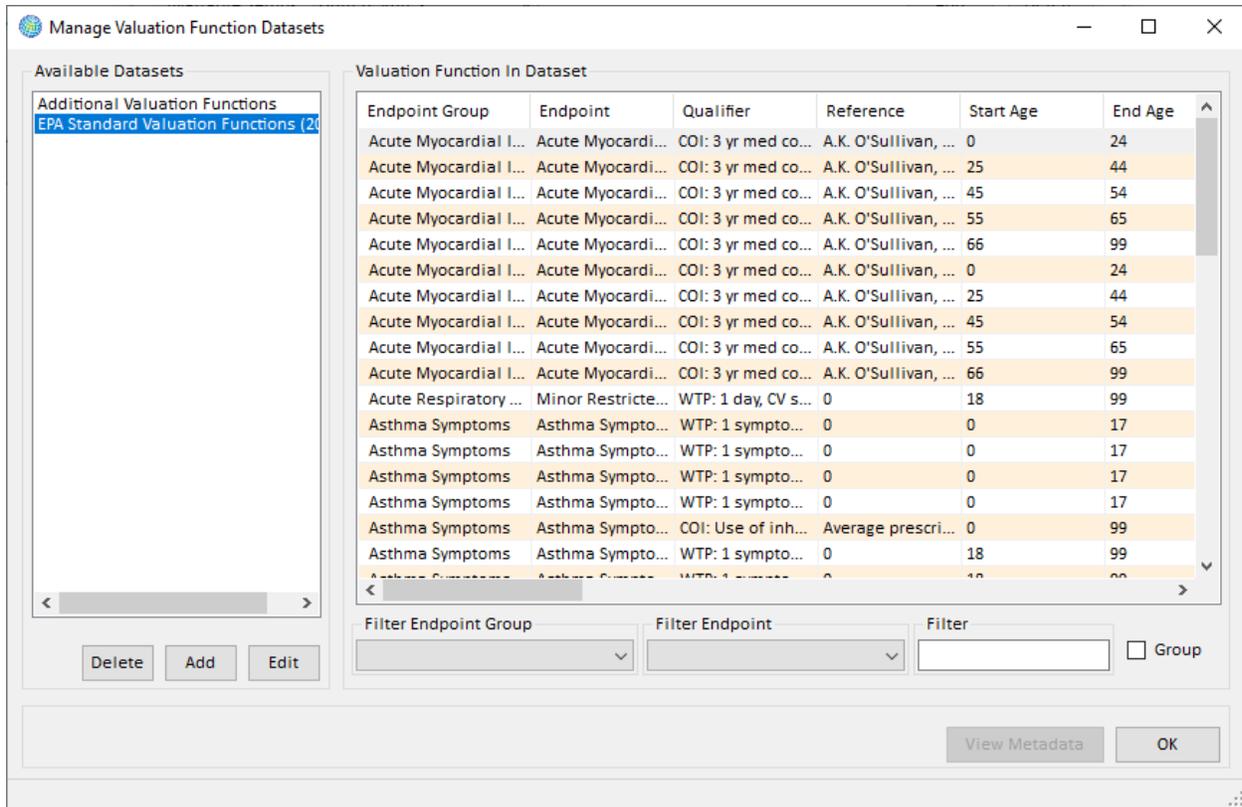
For mortality endpoints, BenMAP-CE generally estimates monetized benefits by using the **Value of Statistical Life (VSL)**, a WTP-based estimate derived from an extensive literature of observed or elicited estimates of the monetary value that an individual is willing to exchange for small reductions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

4.1.9.1 Add Valuation Data

To add valuation functions to BenMAP-CE, click on the **Manage** button below the **Valuation Datasets** box in the **Modify Datasets** window. The **Manage Valuation Function Datasets** window will appear.

Warning

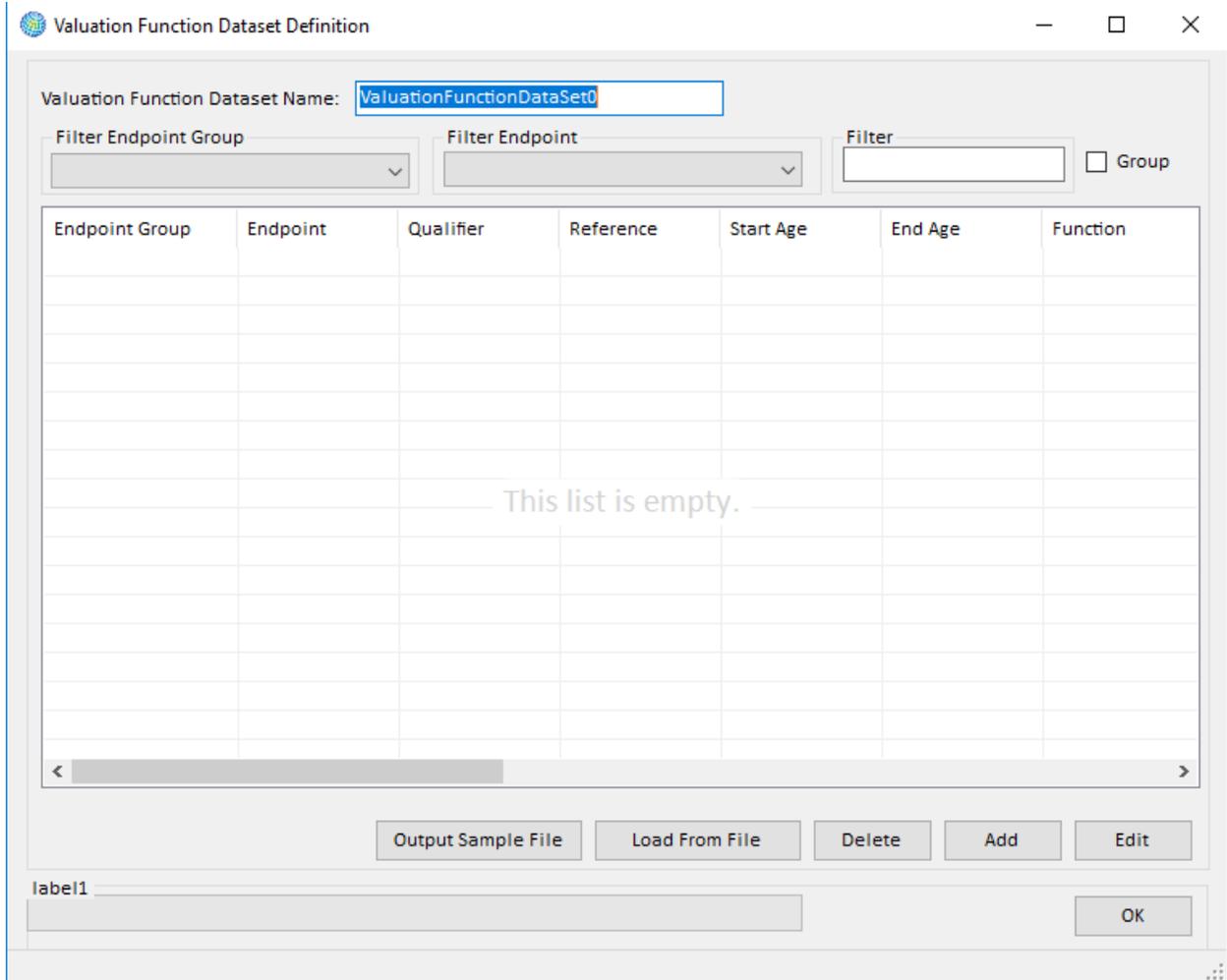
In order to use a valuation function with a particular health incidence result, the **Endpoint Group** of the valuation function must match the **Endpoint Group** of the health incidence result exactly.



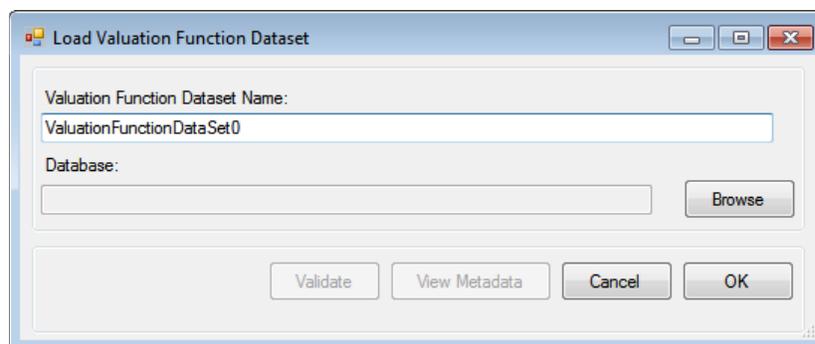
In this window you may **Add**, **Edit**, and **Delete** datasets. The section on the left under **Available Datasets** lists the valuation datasets that are currently in the setup database. The section on the right under the **Valuation Function In Dataset** lets you view the valuation factors in a selected dataset.

If the dataset is large, there are filters available to view a subset of the list by selecting a value from the **Filter Endpoint Group** and/or **Filter Endpoint** drop-down lists. Or, you can type a value in the **Filter** box to search for a particular word or phrase. You can also group data by **Endpoint Group** by selecting the *Group* option.

To add a dataset, click on **Add**. This will display the **Valuation Function Dataset Definition** window.



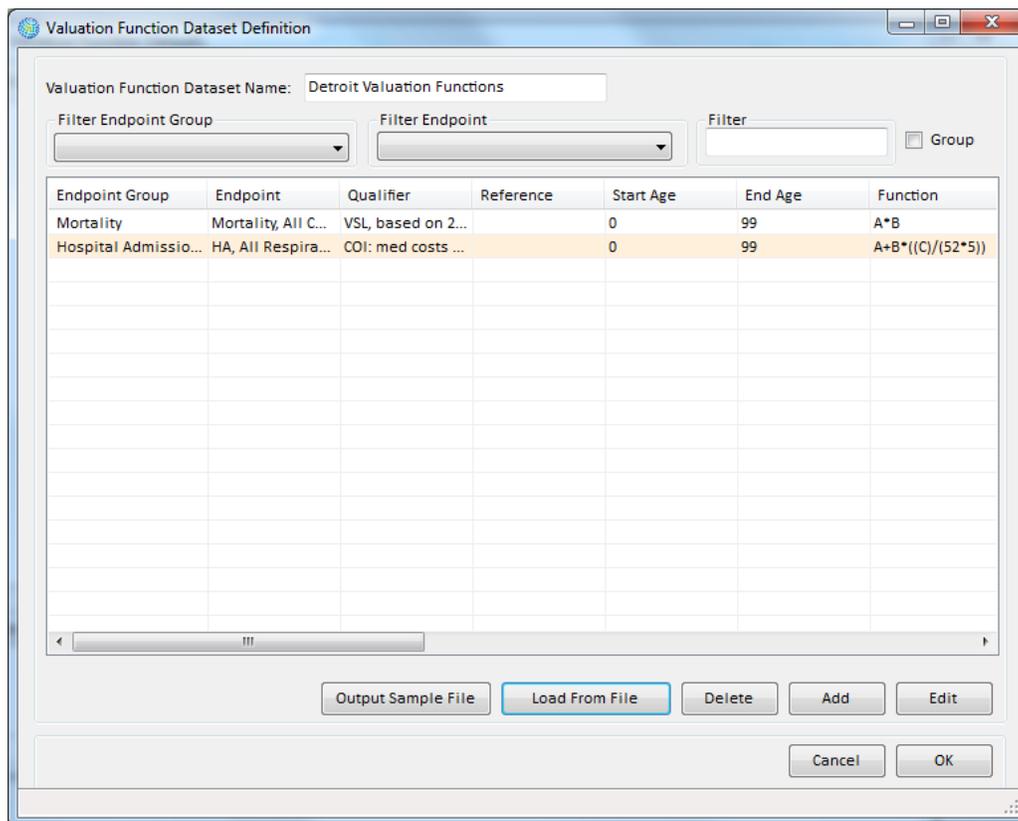
You may load valuation data with an externally-created data file, or you may add individual valuation functions from within BenMAP-CE. To import valuation functions, click on the **Load From File** button. This will bring you to the **Load Valuation Function Dataset** window.



In the **Load Valuation Function Dataset** window provide a name for the valuation function dataset. Use the **Browse** button to choose the valuation database and click **Open**. Once again, you can click the **Validate** button before the file is imported. In

addition, you can create metadata, which includes references and descriptions of the file, by clicking on the **View Metadata** button. Click **OK** on the **Load Valuation Function Dataset** window. This will bring you back to the **Valuation Function Dataset Definition** window. Here you can view the valuation functions that you have in your database.

The columns within each of the list boxes can be rearranged in order to provide the most useful display. (Note that rearranging the columns is only for display and has no effect on the underlying Valuation Function dataset.) You can also change how you view the list by specifying a text string to **Filter** the records (e.g., Asthma), or create groupings of records by their Endpoint Group (click the **Group** option box).



In the **Valuation Function Dataset Definition** window, you can also edit the functions already existing in your dataset by highlighting a particular **Valuation Function** and then clicking the **Edit** button.

If the dataset is large, there are filters available to view a subset of the list by selecting a value from the **Filter Endpoint Group** and/or **Filter Endpoint** drop-down lists. When you are finished, click **OK**.

From the **Valuation Function Dataset Definition** window you can also manually define a new **Valuation Function**. Click the **Add** button to open the **Valuation Function Definition** window where you can then create a new **Valuation Function**. (See Appendix M: Function Editor for additional information about the syntax for developing functions with this editor.)

After defining the new **Valuation Function**, click **OK**. This will take you back to the **Valuation Function Dataset Definition** window. When you are finished, click **OK**.

This will return you to the **Manage Valuation Function Datasets** window. From this form you can **Add**, **Delete**, or **Edit** the available datasets. In addition, you can view or edit the previously created metadata for a file by selecting an **Available Dataset** and **Valuation Function** and then click the **View Metadata** button. When you are satisfied with the inputs, click **OK**. The **Modify Datasets** window will appear. Here in the **Valuation Function** datasets box you should see any updates to the **Valuation Function** dataset that you just made.

4.1.9.2 Format for Valuation Data

Table 4-14 presents the variables that can be used in **Valuation Datasets**.

Table 4-14. Valuation Dataset Variables

Field Name	Type	Required	Notes
Endpoint Group	Text	Yes	If this doesn't reference an already-defined Endpoint Group, one will be added.
Endpoint	Text	Yes	If this doesn't reference an already-defined Endpoint for the Endpoint Group, one will be added.
Qualifier	Text	No	Provides additional information to identify a particular valuation function.
Reference	Text	No	Bibliographic reference, included to identify the source in the economic literature.
Start Age	Integer	No	Specifies the low and high ages, inclusive. For example, Start Age of '0' and End Age of '1' includes infants through the first 12 months of life and all one-year old infants.
End Age	Integer	No	
Point Estimate	Numeric (double)	Yes	Central estimate of the unit value.
Function	Text	Yes	The functional form, interpreted (executed) by BenMAP-CE when running an analysis.
A Description	Text	No	Description of variable A.
A	Numeric (double)	No	Mean of the A distribution.
A Distribution	Text	No	If A has no distribution, any value is acceptable. Otherwise, should be one of: <i>Normal, Triangular, Poisson, Binomial, LogNormal, Uniform, Exponential, Geometric, Weibull, Gamma, Logistic, Beta, Pareto, Cauchy, Custom</i> .
A Parameter 1	Numeric (double)	No	Parameter 1 of the A distribution (meaning depends on the distribution - for <i>Normal</i> distributions this represents the standard deviation).
A Parameter 2	Numeric (double)	No	Parameter 2 of the A distribution (meaning depends on the distribution - for <i>Normal</i> distributions this is not required).
Constant Description	Text	No	Description of variables, B, C, and D.
Constant Value	Numeric (double)	No	A constant value (denoted by B, C, and/or D) which can be referenced by the Function.

4.1.10 Income Growth Data

According to economic theory, willingness-to-pay (WTP) to avoid air-pollution related morbidity effects and premature mortality should grow as real income increases. BenMAP-CE allows users to adjust the WTP estimates to account for the growth in income over time. This adjustment is a combination of data on income growth and estimated income elasticity of demand, which measures the responsiveness of the quantity demanded of a good to the change in the income of the people demanding the good; this is distinct from elasticity of demand, which quantifies the change in demand for goods and services as a result of changes in price for those goods and services. This section describes how to load the data adjusting for income growth and how EPA developed these adjustment factors.

Fundamental Concept – Income Growth Adjustment

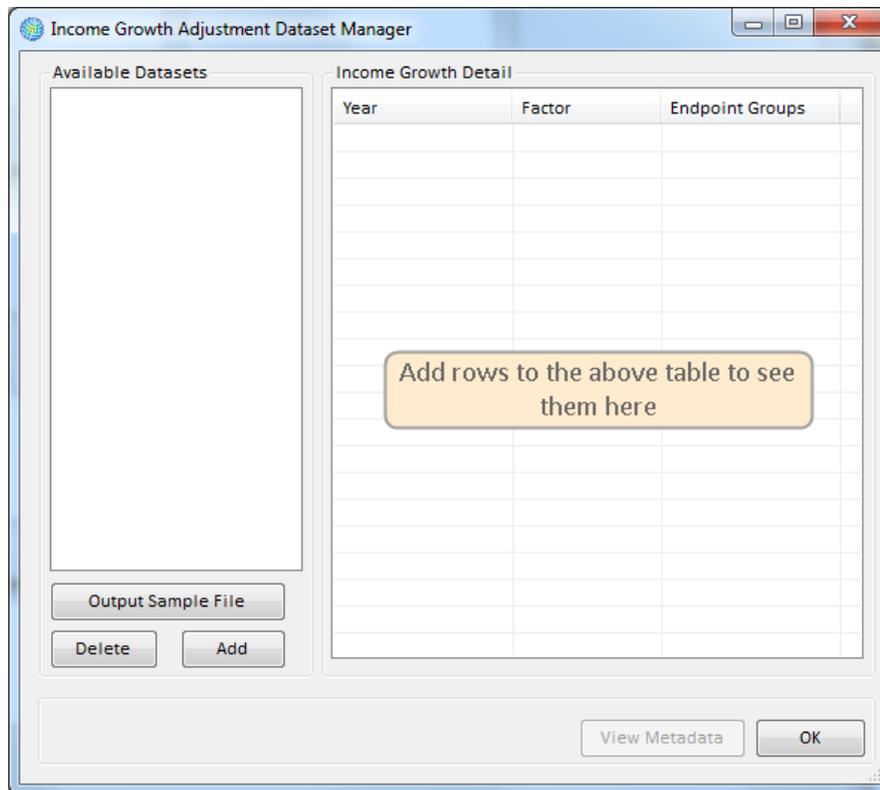
Income Growth Adjustment is the adjustment of the output of certain valuation functions to reflect the impact of increases in real income over time on WTP values. Generally, an increase in real income is expected to result in an increase in the willingness to pay (WTP). Users may select alternative values for the sensitivity of WTP to these changes.

Note that the WTP estimates in the default valuation functions in the *United States* setup are assumed to be based on 1990 income, so the income growth adjustments are all relative to 1990. That is, the income growth data has a value of 1 in 1990, and because income has generally increased over time in the U.S., the income growth values are typically greater than 1 after 1990. (An exception is 1991, when incomes declined slightly in the U.S.)

If you load in your own valuation functions and/or income growth adjustment factors, be sure that you have carefully considered the income year. For example, if your valuation functions are based on income in the year 2005, then the income growth adjustment should have a value of 1.0 in 2005, because no adjustment is necessary. As you forecast into the future, under the assumption that incomes go up over time, then your income growth adjustment factors would have values greater than 1.0 for years past 2005, and would have values less than 1.0 for years prior to 2005.

4.1.10.1 Add Income Growth Data

To add income growth data to BenMAP-CE, click on the **Manage** button below the **Income Growth Adjustments** box in the **Modify Datasets** window. The **Income Growth Adjustment Dataset Manager** window will appear.



In this window you may **Add** and **Delete** datasets. The section on the left under **Available Datasets** lists the **Income Growth Adjustments Datasets** that are currently in the setup database. The section on the right under **Income Growth Detail** presents the income growth adjustment factors in a selected dataset.

Click on the **Add** button. In the **Load Income Growth Adjustment Factors Dataset** window, type in the name of the dataset in the **Income Growth Adjustment Dataset Name** box, and then click on the **Browse** button to the right of the **Database** box to choose the dataset that you want to import and click **Open**. To confirm the file has proper formatting, click the **Validation** button before importing the file. In addition, you can edit the metadata for the imported file, to include references and descriptions, by clicking the **View Metadata** button.

Click **OK**. The **Income Growth Adjustment Dataset Manager** window will appear. Here you may view the data that you just loaded. From this window, you can also view or edit metadata by selecting an **Available Dataset** and **Income Growth Detail** and then clicking the **View Metadata** button.

4.1.10.2 Format for Income Growth Adjustment Data

Table 4-15a presents the variables that can be used in **Income Growth Adjustment Datasets** and Table 4-15b presents a sample of what a dataset might look like.

Table 4-15a. Income Growth Adjustment Dataset Variables

Variable	Type	Required	Notes
Year	Integer	Yes	The year of the data. Note that this will include historical estimates as well as forecasts.
Mean	Numeric (double)	Yes	Mean income growth adjustment factor.
EndPointGroup	Text	Yes	Endpoint group (e.g., <i>Chronic Asthma</i>).

Table 4-15b. Sample Income Growth Adjustment Dataset

Year	Mean	EndpointGroup
1990	1	Acute Bronchitis
1991	0.99788719	Acute Bronchitis
1992	1.0010463	Acute Bronchitis
1993	1.00313532	Acute Bronchitis
1994	1.00725281	Acute Bronchitis
1995	1.00950849	Acute Bronchitis
1996	1.01338995	Acute Bronchitis
1997	1.01823652	Acute Bronchitis
1998	1.02308881	Acute Bronchitis
1999	1.02832949	Acute Bronchitis
2000	1.03276563	Acute Bronchitis
2001	1.0327363	Acute Bronchitis
2002	1.03402805	Acute Bronchitis
2003	1.03695309	Acute Bronchitis
2004	1.04123259	Acute Bronchitis
2005	1.04486656	Acute Bronchitis
2006	1.04742563	Acute Bronchitis
2007	1.04867125	Acute Bronchitis
2008	1.04677248	Acute Bronchitis
2009	1.04110742	Acute Bronchitis
2010	1.04366136	Acute Bronchitis
2011	1.0449264	Acute Bronchitis
2012	1.04712892	Acute Bronchitis
2013	1.04852092	Acute Bronchitis
2014	1.05097616	Acute Bronchitis
2015	1.05361962	Acute Bronchitis
2016	1.05460298	Acute Bronchitis
2017	1.05676126	Acute Bronchitis
2018	1.0586977	Acute Bronchitis
2019	1.05993581	Acute Bronchitis
2020	1.06099141	Acute Bronchitis
2021	1.06246126	Acute Bronchitis
2022	1.06402493	Acute Bronchitis
2023	1.06561422	Acute Bronchitis
2024	1.06718934	Acute Bronchitis
2025	1.0687362	Acute Bronchitis
2026	1.07025254	Acute Bronchitis

4.2 Export and Import Setups

BenMAP-CE allows you to export and import entire databases (all **Available Setups**), individual setups (e.g., *United States, China*), and parts of individual setups (e.g. all **Grid Definitions**, or individual **Health Impact Function** datasets). This functionality can be used to archive data, share data with other BenMAP-CE users, as well as to allow you to move databases between computers or between versions of BenMAP (currently only supported for versions 1.3 and 1.4). In particular, all of the steps involved in creating a

setup can be done just once, after which the data can be exported and then imported on other computers. See Chapter 9: Tools Menu for more information about the *Database Export* and *Database Import* tools.

Warning

Please note that your existing Setups will not be automatically transferred to newly installed versions of BenMAP. If you wish to preserve your setups, please see instructions for exporting and importing databases in Chapter 9.

4.3 Frequently Asked Questions

I've loaded new baseline incidence rates, but BenMAP-CE won't let me select it in the configuration stage.

When formatting these data for importing to BenMAP-CE, take special care to ensure that you have specified the health endpoints correctly. The baseline incidence rate must be associated with a specific health endpoint and endpoint group in BenMAP-CE. Be sure that you have recorded the endpoint group and endpoint exactly as it is recorded in BenMAP-CE. For example, if the baseline incidence rate is for asthma-related hospital admissions, be sure you have recorded the endpoint group as '*Hospital Admissions, Respiratory*' and the endpoint as '*HA, Asthma*'.

I've loaded a new grid and new population data into BenMAP-CE but I can't seem to use these new data.

Be sure to load the new grid definition first. Next, load the population dataset and be sure to select your new grid definition.

How do I generate a population dataset for a new grid definition?

You can generate a population dataset using a variety of approaches. The key is that you need to have a shapefile of your area of interest (e.g., Census tracts in a city) and you need to have census data matching your area of interest. One source for both a shapefile and the associated population data is the U.S. Census Bureau. (A variety of other agencies have census data, and you need to check around for your area of interest.) Another option for U.S. population data is to use the PopGrid software application, mentioned in Section 4.1.5 and described in Appendix J on Population Data for the U.S. Setup. Using PopGrid, you still need to have a shapefile for your area of interest.

Can I edit a population configuration?

No, you cannot edit a population configuration. You can only view a population configuration. If the population configuration does not match your data, you need to either create a new population configuration to match your data, or reshape your data so that it matches the population configuration.

Chapter 5

Creating Air Quality Surfaces

In this chapter...

- **Define air quality grids**
- **Create air quality surfaces using different methods.**
- **Learn how to structure input files.**
- **Learn how to interpolate monitoring data with Closest Monitor, Voronoi Neighborhood Averaging, or Fixed Radius.**
- **Learn about the Monitor Rollback feature.**

BenMAP-CE is not an air quality model and it cannot simulate changes in air quality. Instead it relies on the model-simulated or monitored air quality inputs given to it. To estimate population exposure to air pollution, BenMAP-CE uses air quality surfaces that it generates from input air quality data (modeling or monitoring data). The program uses a grid structure, where each cell contains a model or monitored air quality value.

BenMAP-CE creates air quality surfaces to estimate the average exposure to ambient air pollution of people living in a “grid.” These grids are either regularly shaped areas like those used by air quality models, or irregular shapes, like provinces, local government areas, cities, or nations. BenMAP-CE does not estimate personal exposure. Instead, the program calculates average pollutant concentration to which people are exposed in each grid cell. BenMAP-CE then uses these average values to calculate health impact functions.

To create air quality surfaces, BenMAP-CE uses a number of inputs, including modeling data or monitoring data. You may enter your own modeling and monitoring data, provided that the data are in a format recognized by BenMAP-CE.

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-CE uses one air quality surface to represent the Baseline scenario and a second surface to represent the Control scenario. These baseline and control surfaces must use the same air quality grid. The program calculates the difference between Baseline and Control surfaces as an input to the health impact functions. Air quality surfaces are stored in files with an .aggx file extension.

Fundamental Concept – Baseline and Control Scenarios

BenMAP-CE requires both a **Baseline Scenario** air quality surface and **Control Scenario** air quality surface to estimate the effects of a change in air quality (**Delta**). Both Baseline and Control air quality surfaces are required regardless of whether you use modeling data or monitoring data.

- The **Baseline Scenario** characterizes the air quality levels observed or expected in the absence of the policy change you are evaluating. The baseline is sometimes referred to as “Business as Usual.” The baseline scenario is usually considered to be the reference scenario against which to compare a potential scenario characterized by the implementation of regulations.
- The **Control Scenario** in BenMAP is the scenario in which emissions from one or more source sectors are changed (increased or decreased) from the Baseline scenario. The Control scenario usually represents expected air quality levels after a new regulation or set of regulations has been implemented.

The air quality **Delta** is the change in air pollution between the Baseline air quality grid and the Control air quality grid (Baseline minus Control). BenMAP-CE uses the air quality **Delta** as the input to the health impact function.

To start the grid creation process, locate **Air Quality Surfaces** on the BenMAP-CE tree menu. Under this header, double-click **Pollutant**. On the selection window, click to select a

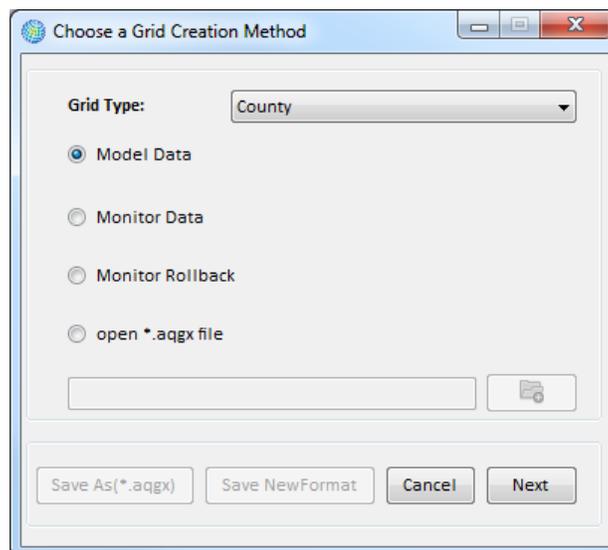
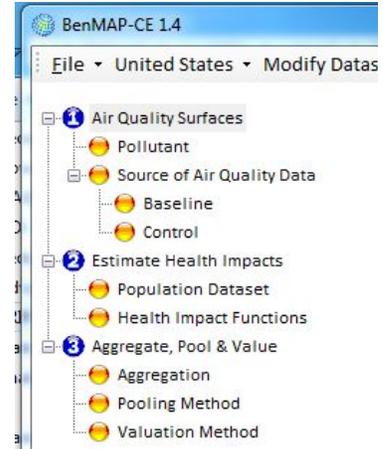
pollutant from the left site and click “Add” button to add it to the right side. You may also click, hold and drag the pollutant of choice from the left side to the right side. Click **OK**.

To remove a selected pollutant, click the Remove button or double click the pollutant name to remove it from the left site.

Next, double-click **Baseline** on the BenMAP-CE tree menu to open the **Grid Creation Method** window.

BenMAP-CE will then ask which **Grid Type** (which you select from a previously loaded shapefile) to use and which of the following types of air quality data you wish to use:

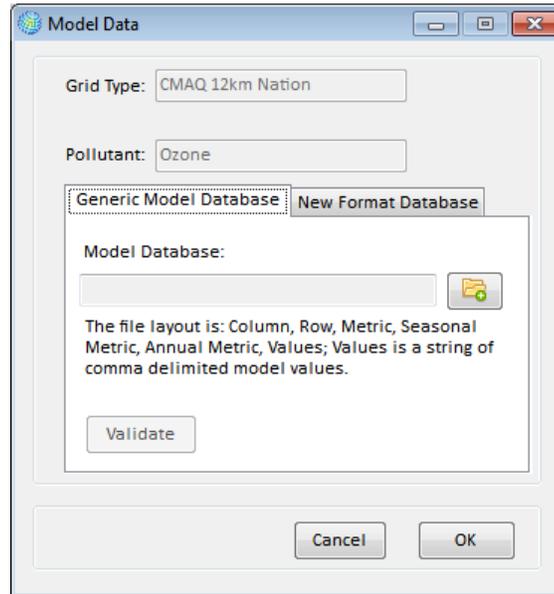
- *Model Data*. Choose this option if you have air quality modeling data that you wish to use directly. Table 5-1 below describes the input format for modeling data.
- *Monitor Data*. Choose this option if you wish to use point source monitoring data (measured observations).
- *Monitor Rollback*. Choose this option if you want to reduce monitor levels by a specified amount.
- *Open *.aqgx file*. Choose this option to import a file that has already been created.



Select your **Grid Type** and then click **Next**. BenMAP-CE will direct you through the necessary steps for each option (described below).

5.1 Model Data

After choosing the *Model Data* option, use the **Generic Model Database** tab to load grid-definition-based modeling data (e.g., CMAQ or CAMx).¹⁹ For model files being imported from an Excel workbook with multiple tabs, BenMAP-CE will prompt you to select the tab containing the data for import.



The **Model Database** specifies the location of the air quality model results that you want to import. Table 5-1 presents the structure that these files must have, and Table 5-2 presents a sample data file with a variety of metrics. (For more information on air quality models, the EPA website has detailed descriptions of a variety of models at <https://www.epa.gov/scram>.)

Table 5-1. Air Modeling Data File Variables

Variable	Type	Required	Notes
Column	Integer	Yes	The column and the row uniquely identify each set of modeling values and link the modeling data with cells in a Grid Definition.
Row	Integer	Yes	
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., 1-hour daily maximum) for the appropriate Pollutant.

¹⁹ Community Multi-scale Air Quality (CMAQ) Model is available at: <http://www.epa.gov/amad/Research/RIA/cmaq.html> or <https://www.cmascenter.org/cmaq/>. Comprehensive Air Quality Model with Extensions (CAMx) is available at: <http://www.camx.com/>.

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).
Annual Metric	Text	No	This variable is either blank (signifying that the values are not an annual metric) or must be one of: <i>None, Mean, Median, Max, Min, Sum</i> (e.g., mean of the 1-hour maximum values for the year)
Values	Comma Separated Values (Text) ²⁰	Yes	If Metric is blank, values are supplied as a comma-delimited string of values for the year [e.g., 365 or 366 (leap year) values for daily data, 8760 or 8784 (leap year) values for hourly data]. If Metric is defined, but Seasonal Metric and Annual Metric are blank, 365 or 366 metric values. If Seasonal Metric is defined, but Statistic is blank, <i>n</i> seasonal metric values. If Annual Metric is defined, one annual statistic value for either the Metric (if Seasonal Metric is blank) or the Seasonal Metric. Missing values are signified with a period ('.').

Table 5-2. Sample Air Modeling Data File

Column	Row	Metric	Seasonal Metric	Annual Metric	Values
24	101			,5.90,,,11,...
24	102				19.80,,,3.5,,,14.70,,,,,4.90
24	103				11.5,,,1.5,,,10.20,,,2.5,,,2.6
24	104				12.60,,,4.60,,,7.30,,,,,9.20
24	105	DailyAverage		,5.90,,,11,...
24	106	DailyAverage			19.80,,,3.5,,,14.70,,,,,4.90
24	107	DailyAverage			11.5,,,1.5,,,10.20,,,2.5,,,2.6
24	108	DailyAverage			12.60,,,4.60,,,7.30,,,,,9.20
24	109	DailyAverage	QuarterlyAverage		11.82,13.24,18.79,14.25
24	110	DailyAverage	QuarterlyAverage		12.31,13.89,18.27,21.19
25	101	DailyAverage	QuarterlyAverage		12.33,15.68,18.49,15.96
25	102	DailyAverage	QuarterlyAverage		13.52,13.69,19.04,22.43
25	103	DailyAverage		Mean	14.52
25	104	DailyAverage		Mean	16.41
25	105	DailyAverage		Mean	15.62
25	106	DailyAverage		Mean	17.17
25	107	DailyAverage	QuarterlyAverage	Mean	14.29
25	108	DailyAverage	QuarterlyAverage	Mean	18.97
25	109	DailyAverage	QuarterlyAverage	Mean	20.14
25	110	DailyAverage	QuarterlyAverage	Mean	16.46

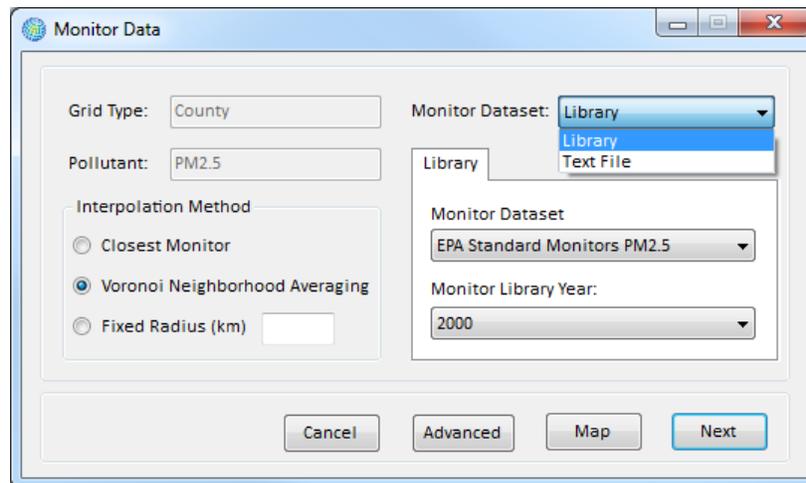
²⁰ The list of comma-separated values must be surrounded by quotation marks, otherwise only the first value in the list will be used.

5.2 Monitor Data

Using the *Monitor Data* grid creation option, you create an air quality grid directly from air pollution monitoring data. At the top left of the **Monitor Data** window, you will see the previously selected grid definition in the **Grid Type** field, and the previously selected pollutant in the **Pollutant** field. Below the **Pollutant** field of the **Monitor Data** window, you are asked to select an **Interpolation Method**. The interpolation method is used to move from point-based monitor data to grid cell-based air quality data. That is, some grid cells will have many monitors in them, some will have just one, and some will have none. BenMAP-CE uses the interpolation methods to generate representative air quality metric values for each grid cell from monitor data for all of these cases.

Fundamental Concept - Interpolation

Interpolation is the process of estimating the air quality level in an unmonitored area using data from one or more nearby air quality monitors. BenMAP-CE includes three types of interpolation options: one assigns the value from the closest monitor; a second assigns the weighted average of all monitors within a user-specified fixed radius, and the third applies the Voronoi Neighbor Averaging method. These interpolation methods are discussed in more detail below and in Appendix B.



BenMAP-CE includes three **Interpolation Methods**. The *Closest Monitor* method simply uses the monitor closest to a grid cell's center as its representative value. The *Voronoi Neighborhood Averaging* method takes an inverse-distance weighted average of a set of the monitors surrounding a grid cell's center as its representative value. The *Fixed Radius* method averages all of the monitors within a fixed (user-specified) radius measured from the center of the grid cell. Each method is described below. For more detail, also see Appendix B on Air Pollution Exposure Estimation Algorithms.

On the right side of the **Monitor Data** window, you can specify a source for your monitor data. Using the **Library** tab, you may select from data that you have already loaded into BenMAP-CE. Choose the **Monitor Dataset** and **Monitor Library Year** from the drop-down lists.

If you want to load your own monitor data, choose *Text File* from the **Monitor Dataset** drop-down list. You can then browse to locate the data file you want to load. See Chapter 4 for more information about formatting monitor datasets.

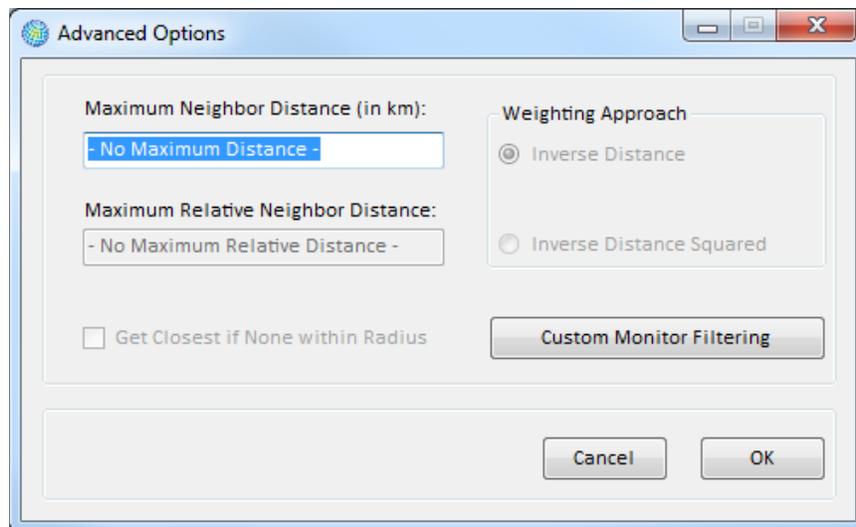
At the bottom of the **Monitor Data** window, there is a **Map** button. The **Map** button opens the **Monitor Map** window, allowing you to preview the map that you are about to load.

Decision Point – Monitor Data

Monitor data may be stored as part of a BenMAP-CE setup *or* loaded as a text file for a particular analysis. If you have already loaded your monitor data into a setup, choose *Library* from the **Monitor Dataset** drop-down list and select the appropriate monitor data in the **Library** tab. If you want to load your own monitor data, choose *Text File* from the **Monitor Dataset** drop-down list and browse to locate the data file you want to load.

5.2.1 Closest Monitor for Monitor Data

If you choose the *Closest Monitor* option, BenMAP-CE identifies the monitor closest to each grid cell's center, and then assigns that monitor's data to the grid cell. *Closest Monitor* interpolation can be modified by clicking on the **Advanced** button at the bottom of the window and typing in a **Maximum Neighbor Distance (in km)**.



The **Maximum Neighbor Distance** specifies the maximum distance (measured in kilometers) that a monitor may be from the center of a grid cell. Cells without any monitors within this distance will not be included in the resultant air quality grid. The default setting is infinite (i.e., no limit to the monitor's distance from the center of the grid cell).

Note: The **Maximum Relative Neighbor Distance** and the **Weighting Approach** options are irrelevant (and are therefore disabled) when using the *Closest Monitor* method, since BenMAP-CE is only choosing a single monitor to assign to any given grid cell.

5.2.2 Voronoi Neighborhood Averaging (VNA) for Monitor Data

If you choose the *Voronoi Neighborhood Averaging* option, BenMAP-CE first identifies the set of monitors that "surround" each grid cell's center (these monitors are referred to as the grid cell's neighbors), and then BenMAP-CE calculates an inverse-distance weighted average of these neighboring monitors. In this section, we provide some examples of the different ways that BenMAP-CE calculates the average of the neighboring monitors. See Appendix B on Air Pollution Exposure Estimation Algorithms for an expanded discussion of VNA, including how the VNA algorithm actually chooses the neighbor monitors, as well as the different ways that it may be used.

VNA interpolation has three advanced interpolation options, which can be modified by clicking on the **Advanced** button at the bottom of the window:

- **Maximum Neighbor Distance (in km)** specifies the maximum distance that a monitor may be from the center of a grid cell, and still be included in the set of neighbor monitors used to calculate air pollution exposure at a particular grid cell. The default setting is infinite (i.e., no limit to the monitor's distance to the center of the grid cell).
- **Maximum Relative Neighbor Distance** specifies the maximum ratio for the distance of each included monitor to the distance of the closest monitor. The default setting is infinite.
- **Weighting Approach** specifies whether BenMAP-CE should use inverse-distance weighting for the monitors, or inverse-distance-squared weighting of the monitors. The default setting is inverse-distance weighting.

The following examples illustrate how varying these options affects the final average concentration estimate.

Example 1: Monitor Data VNA method

Default options

Consider the following example at a hypothetical rural grid cell, where there are relatively few monitors, and where the distance from a monitor to the center of a grid cell can be fairly large. With *VNA*, BenMAP-CE first identifies the set of "neighbor" monitors for each grid cell. The number of neighbors is usually in the range of about three to eight. In this case, assume that there are five monitors at distances of 25, 50, 100, 200, and 400 km from the grid cell, with annual PM_{2.5} levels of 8, 13, 12, 18, and 15 µg/m³, respectively. BenMAP-CE would calculate an inverse-distance weighted average of the monitor values as follows:

$$PM_{2.5} \text{ average} = \frac{\frac{1}{25} \cdot 8 + \frac{1}{50} \cdot 13 + \frac{1}{100} \cdot 12 + \frac{1}{200} \cdot 18 + \frac{1}{400} \cdot 15}{\frac{1}{25} + \frac{1}{50} + \frac{1}{100} + \frac{1}{200} + \frac{1}{400}} = 10.68$$

Example 2: Monitor Data VNA method

Maximum Neighbor Distance = 75

Using the same example that we used above, let us say you have specified a **Maximum Neighbor Distance** of 75 km, and left unchanged the default options (infinite value) for **Maximum Relative Neighbor Distance**. BenMAP-CE would only consider the first two monitors, and would calculate an inverse-distance weighted average of the monitor values as follows:

$$PM_{2.5} \text{ average} = \frac{\frac{1}{25} \cdot 8 + \frac{1}{50} \cdot 13}{\frac{1}{25} + \frac{1}{50}} = 9.67$$

Example 3: Monitor Data VNA method

Maximum Relative Neighbor Distance = 10

Alternatively, if you left the **Maximum Neighbor Distance** at the default (infinite), but have set the **Maximum Relative Neighbor Distance** to 10, then BenMAP-CE would calculate the ratio of the distance for each monitor to distance of the closest monitor. In this case, the ratios would be 1 (=25/25), 2 (=50/25), 4 (=100/25), 8 (=200/25), and 16 (=400/25), and BenMAP-CE would drop the monitor with a ratio of 16. BenMAP-CE would then calculate an inverse- distance weighted average of the monitor values as follows:

$$PM_{2.5} \text{ average} = \frac{\frac{1}{25} \cdot 8 + \frac{1}{50} \cdot 13 + \frac{1}{100} \cdot 12 + \frac{1}{200} \cdot 18}{\frac{1}{25} + \frac{1}{50} + \frac{1}{100} + \frac{1}{200}} = 10.53$$

Example 4: Monitor Data VNA method

Inverse-distance squared neighbor weighting

In addition, you can specify an inverse-distance-squared weighting of the monitors. Let us say that you have left unchanged the defaults (infinite values) for **Maximum Neighbor Distance** and **Maximum Relative Neighbor Distance**, and specified that the

Weighting Approach is *Inverse Distance Squared*. BenMAP-CE would then calculate an inverse-distance-squared weighted average of the monitor values as follows:

$$PM_{2.5} \text{ average} = \frac{\frac{1}{625} \cdot 8 + \frac{1}{2,500} \cdot 13 + \frac{1}{10,000} \cdot 12 + \frac{1}{40,000} \cdot 18 + \frac{1}{160,000} \cdot 15}{\frac{1}{625} + \frac{1}{2,500} + \frac{1}{10,000} + \frac{1}{40,000} + \frac{1}{160,000}} = 9.26$$

Example 5: Monitor Data VNA method

Maximum Neighbor Distance = 75

Maximum Relative Neighbor Distance = 10

Inverse-distance squared Weighting Approach

Finally, you could specify changes to all three options: a **Maximum Neighbor Distance** of 75 km, a **Maximum Relative Neighbor Distance** of 10, and a **Weighting Approach** of *Inverse Distance Squared* weighting. BenMAP-CE would then calculate the following average:

$$PM_{2.5} \text{ average} = \frac{\frac{1}{625} \cdot 8 + \frac{1}{2,500} \cdot 13}{\frac{1}{625} + \frac{1}{2,500}} = 9.00$$

5.2.3 Fixed Radius for Monitor Data

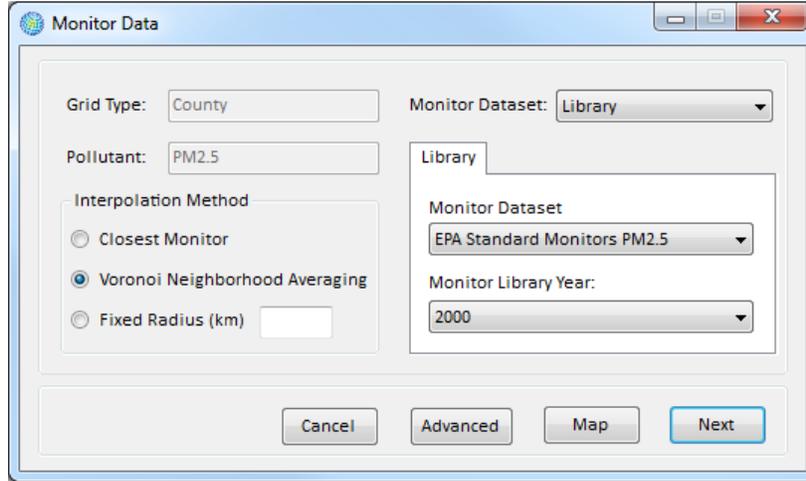
If you choose the *Fixed Radius (km)* option, BenMAP-CE averages all of the monitor values within a fixed radius (measured in kilometers) that you specify. The way that the monitor values are averaged depends on the **Weighting Approach** that you choose after clicking the **Advanced** button. You can choose either *Inverse Distance* or *Inverse Distance Squared* weighting.

Note that the default option with the *Fixed Radius* approach is that BenMAP-CE will not calculate an average for a grid cell if there are no monitors within the fixed radius (distance) that you specify. In the **Advanced Options** window, if you select *Get Closest if None within Radius*, then for those grid cells without any monitors within the fixed radius, BenMAP-CE will choose the nearest monitor (regardless of distance) and apply that value as the “average”.

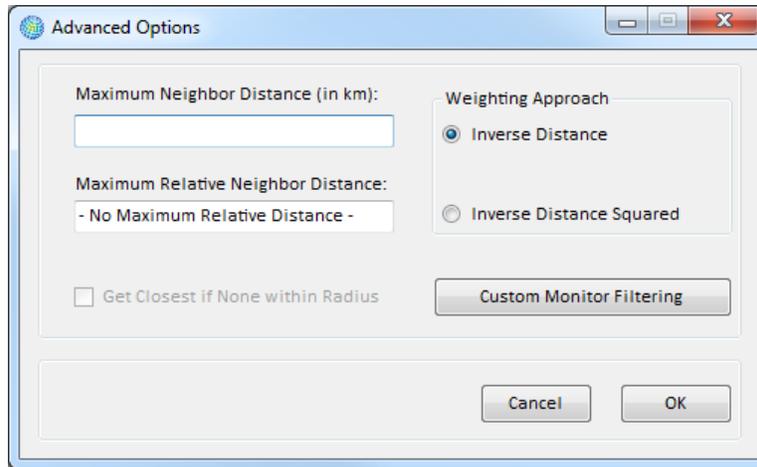
5.2.4 Custom Monitor Filtering for EPA Standard Monitors

Custom Monitoring Filtering options apply only to the EPA Standard Monitors library in the United States setup; these are the only monitoring values that include all of the variables that BenMAP-CE needs in order to filter the data properly. This tool allows you to filter, map, and export your monitor data. You can reach the **Custom Monitor**

Filtering tool by first choosing your pollutant, data source (e.g., monitor library) and year on the **Monitor Data** window.



Click the **Advanced** button. This will take you to the **Advanced Options** window.



Click the **Custom Monitor Filtering** button. This will take you to the **Filter Monitors** window.

Note that the first five options are essentially the same for each pollutant, and the sixth option depends on the pollutant. The example above shows what the form looks like with *PM_{2.5}* as the selected **Pollutant**.

1. Include specific monitors. Here you can specify particular monitor IDs that you want to include in your analysis. If this is left blank, then BenMAP-CE will include all monitors that meet the rest of the selection criteria.

2. Exclude specific monitors. Here you can exclude any particular monitor IDs from your analysis. Here again, if this option is left blank then BenMAP-CE will include all monitors that meet the rest of the selection criteria.

3. Restrict to particular states and/or latitude/longitude. You can choose monitors to include from particular states, by listing the two-character state abbreviation (e.g., CA = California). You can also choose monitors within particular latitude and longitude ranges. The default values for latitude (20 to 50) and longitude (-130 to -65) completely

include the continental U.S. Here again, if this option is left blank then BenMAP-CE will include all monitors that meet the rest of the selection criteria.

4. Parameter Occurrence Code (POC)²¹. Sometimes, multiple monitors are collocated at the same site measuring the same parameter (e.g., to check precision). The **Maximum POC** specifies the highest POC value allowed in the data. The default is a value of 4. And to choose one monitor when more than one monitor is in the same location, the POC Preference Order specifies the preferred ordering of POC codes.

5. Methods.²² The Method codes listed indicate U.S. EPA-defined methods for collecting and analyzing samples; these codes depend on the pollutant. In the case of PM_{2.5}, only federal-reference methods (FRM) are chosen by default -- specifically numbers 116 through 120. In the case of Ozone (O₃), all methods are checked by default.

6. Parameters Specific to the Pollutant. The default options vary by pollutant. Below, we have described the options that appear with *PM_{2.5}* and *ozone*.

- **PM_{2.5} Monitor Filter:** The **Number of Valid Observations Required per Quarter** specifies the number of days of data needed. The default is to require 11 observations per quarter. The **Data Types to Use** options specify whether to use data at *Local* conditions (parameter code 88101), *Standard* conditions (parameter code 81104), or *Both*.²³ The default for PM_{2.5} is to use data at *Local* conditions. When data at standard and local conditions are both available at the same monitor location, the **Preferred Type** allows you to choose which to use – the default is *Local*. The **Output Type** option is designed to allow you to make the data reasonably consistent when both local and standard condition data are used. The default is to use the *Local* output type, so *Standard* condition data will be converted to *Local*.
- **Ozone Monitor Filter:** The *ozone* specific options differ from *PM_{2.5}* in large part because ozone is monitored hourly in the United States. The **Number of Valid Hours** specifies the number of hours needed for a particular day of monitoring to be considered “valid.” BenMAP-CE counts the number of non-missing hourly values from the **Start Hour** through the **End Hour** and compares this number with that specified in the **Number of Valid Hours**.

The **Percent of Valid Days** specifies the percent of days between the **Start Date** and the **End Date** that need to be valid for the monitor itself to be considered valid. The

²¹ For information about data codes used in U.S. EPA’s Air Quality System, refer to the *AQS Data Coding Manual*: https://www.epa.gov/sites/production/files/2015-09/documents/aqs_data_coding_manual_0.pdf

²² For more information, refer to U.S. EPA’s *List of Designated Reference and Equivalent Methods*: <https://www3.epa.gov/ttnamti1/files/ambient/criteria/AMTIC%20List%20Dec%202016-2.pdf>

²³ Particulate concentrations are expressed in either local conditions (volume is at temperature and pressure of the ambient sample) or at standard conditions (where the volume has been converted to standard conditions, typically 20° C at 760 mm Hg).

default is 50 percent of the days between May 1 and September 30. The example below shows what the form looks like with *Ozone* as the selected **Pollutant**.

You can view a map of your data with the specified filter options by clicking the **Map** button on the bottom left side of the **Filter Monitors** window. This provides a brief preview of what will be shown once the data are loaded.

You can also export your data by clicking the **Export** button, which is found in the bottom left side of the **Filter Monitors** window. A **Save As** window will appear, allowing you to save the data as a CSV file.

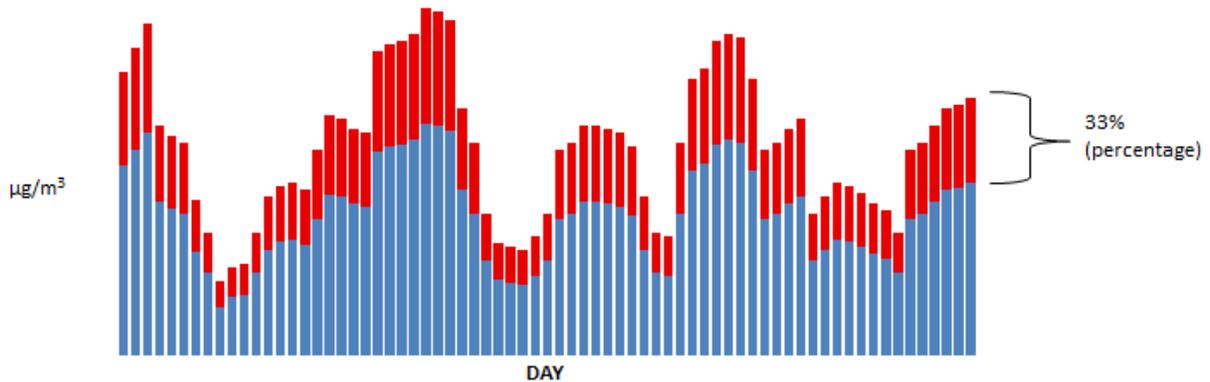
Fundamental Concept - Rollback

A **rollback** is a simplified type of BenMAP analysis in which all Baseline scenario monitor data are reduced in the Control scenario using a single, uniform rule. BenMAP-CE offers three rollback options: **Percentage rollback** reduces all monitor observations by the same percentage. **Incremental rollback** reduces all observations by the same increment. **Rollback to a standard** reduces monitor concentrations as necessary to ensure all monitor observations are at or below a user-specified air quality standard.

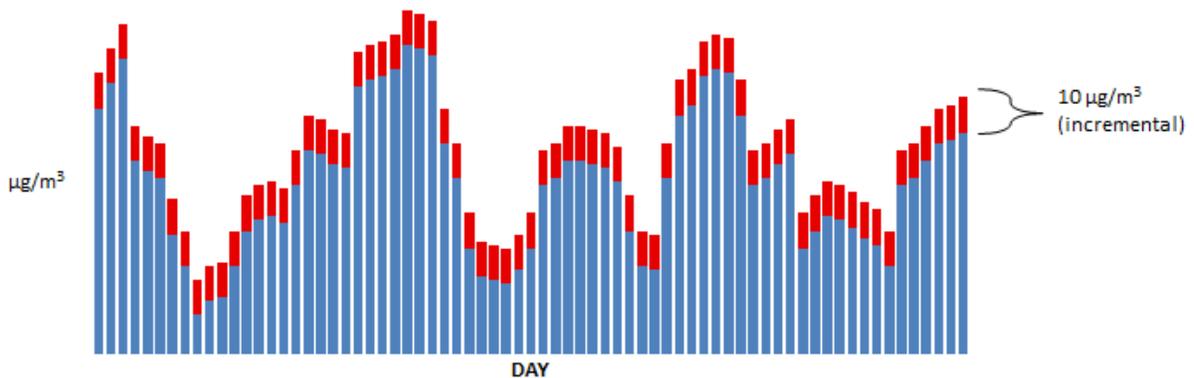
5.3 Monitor Rollback

The *Monitor Rollback* option allows you to quickly test what the benefits would be from reducing historical monitor levels. BenMAP-CE has three methods to reduce, or "roll back," monitor data: *Percentage Rollback*, *Incremental Rollback*, or *Rollback to a Standard*. Each of these methods is depicted below. Note that with each of these methods you can use the same two interpolation algorithms (*Closest Monitor* or *VNA*) as you can use with *Monitor Data*.

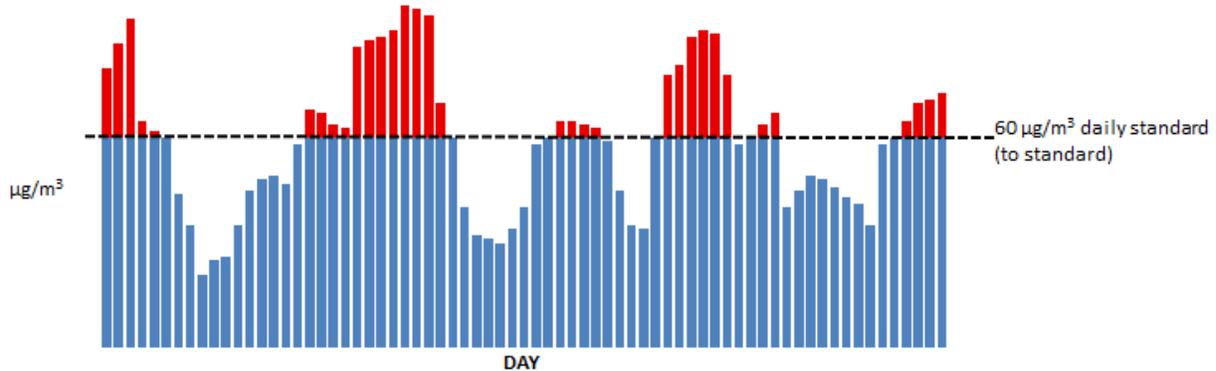
Percentage Rollback reduces all monitor observations by the same percentage:



Incremental Rollback reduces all observations by the same increment:



Rollback to a Standard lets you choose a standard, and then reduces any monitor observations exceeding the standard to the level of the standard:



5.3.1 Example: A Single Rollback in One Region

To apply a monitor rollback, first click the **Create Air Quality Grids** button. On the **Air Quality Grid Creation Method** window, choose *Monitor Rollback*. Click **Next**.

Monitor Rollback: (1) Select Monitors

Pollutant: PM2.5

Rollback Grid Type: State

Library Text File

Monitor Dataset: EPA Standard Monitors PM2.5

Monitor Library Year: 2006

Cancel Advanced Next

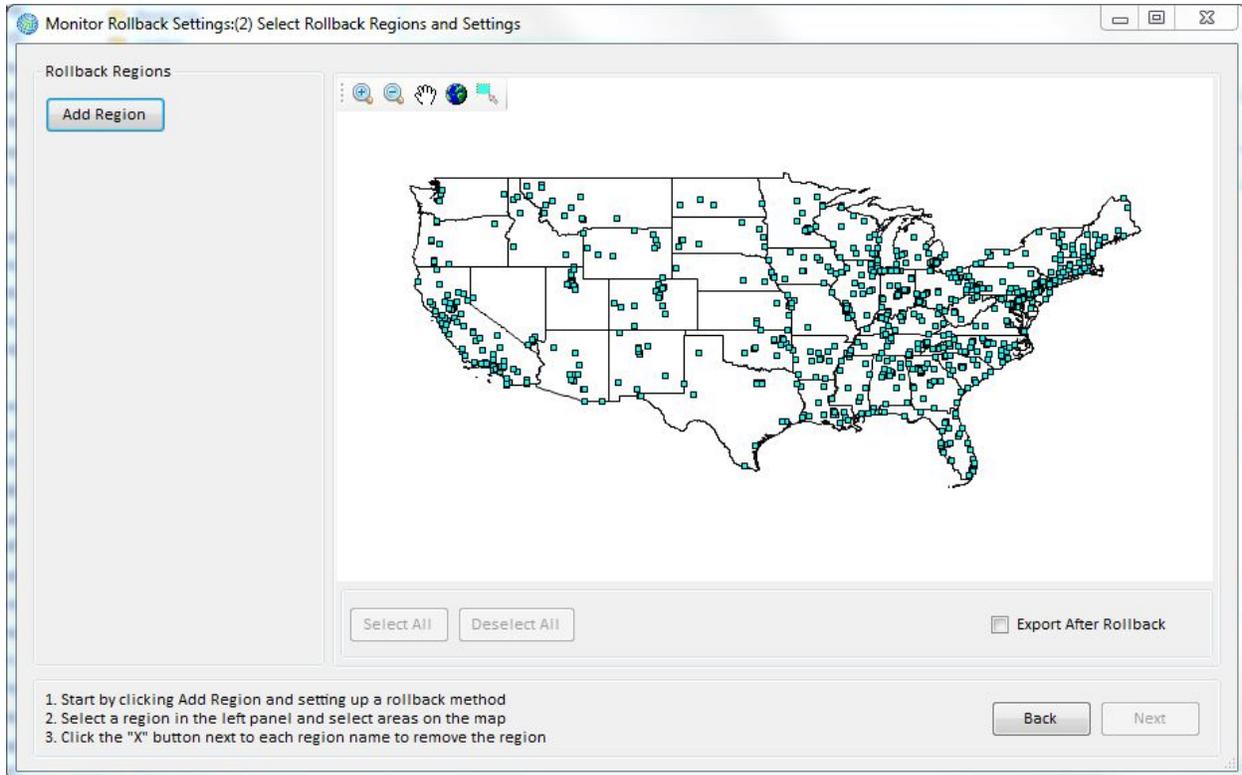
There are three steps to the *Monitor Rollback* method.

1. Select Monitors. Choose the **Rollback Grid Type** from the drop-down list. This allows you to determine how detailed the rollback scenario may be. If the whole region (e.g., United States) will have the same type of rollback then you may simply choose a

grid outlining the area of interest. If you are interested in different rollbacks within a region, then you should choose a more finely detailed grid definition (e.g., states).

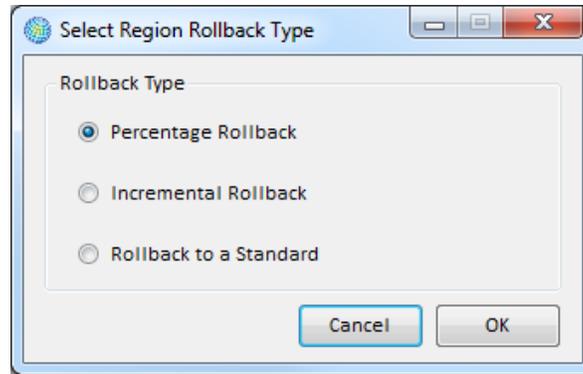
If you use data from an existing dataset, then choose the **Library** tab, and from the drop-down list choose the **Monitor Dataset** and the **Monitor Library Year**. If you want to use your own data, then choose the **Text File** tab. The file should have the monitor data format specified in Chapter 4: Loading Data.

When you have finished making your choices, click **Next**.



2. Select Rollback Regions and Settings. In this section, you can specify the type of the rollback method(s) that you would like to use, as well as the location of the monitors that you want to rollback.

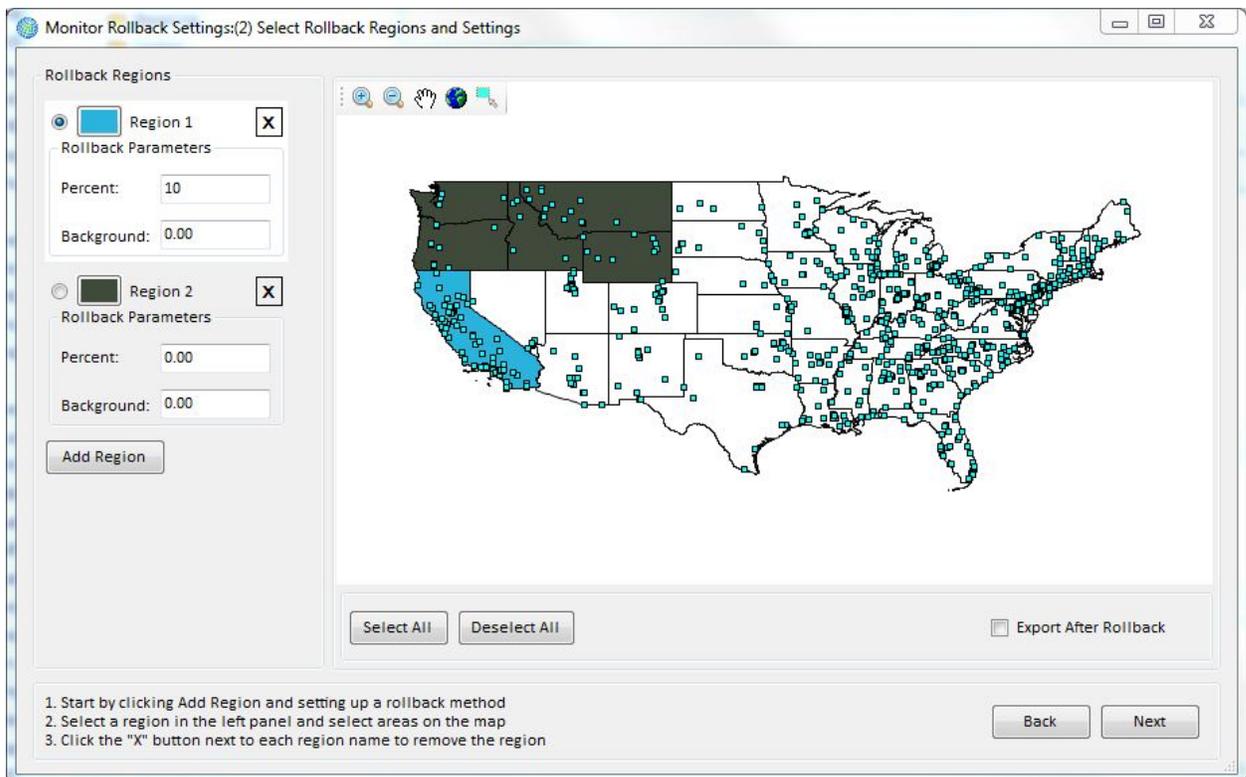
Click the **Add Region** button to display the three rollback options: *Percentage Rollback*, *Incremental Rollback*, and *Rollback to a Standard*.



Choosing the **Rollback Type** and click **OK**. Then, specify the amount of the rollback and the region to which you want to apply it. You can click on the map to select and deselect the states (or other defined areas depending on your rollback grid type) to add to the region.

At the top of the map are five GIS toolbar icons, typically seen in mapping programs. The first four tools allow you to zoom in and zoom out, and to focus on the particular groups of grid cells that interest you. The fifth tool allows you to select grid cells graphically, by clicking or dragging a box over them.

In this example, we specified a 10 percent reduction, a background of 0 ppb, and applied it to all monitors in the state of California (by clicking on the particular state in the GIS window).



To apply the rollback to all the states, you can simply click on the **Select All** button. To clear the selections, click the **Deselect All** button. At any time, you can change the grid cells that you have selected. To remove a region from the selection, click the “X” button next to the region ID. This particular example is quite simple, so we will use a more complicated example below.

If you want to export your monitor data to a CSV file, click the option for *Export After Rollback*. The exported data will be formatted in the same specific format required of BenMAP-CE **Monitor Data** import files discussed in Chapter 4.

After defining the **Rollback Regions** and setting the **Rollback Parameters**, click on the **Next** button. BenMAP-CE will then perform the rollback you specified on the monitors in the grid cells that you have chosen.

3. Additional Grid Settings.

The third stage is similar to the *Monitor Data* grid creation method. As with monitor data, you need to specify the **Interpolation Method** (*Closest Monitor*, *VNA*, or *Fixed Radius*) and the **Grid Type**.

By checking the option for *Make Baseline Grid (in addition to Control Grid)* you tell BenMAP-CE to create a baseline grid at the same time as the control grid. The baseline grid uses the same parameters as the control grid, with the exception of the rollback. That is, the baseline uses the same monitor data, interpolation method, and the same grid type. The two resultant grids will serve as both baseline and control scenarios and are automatically selected in the “Air Quality Surfaces” stage of the analysis.

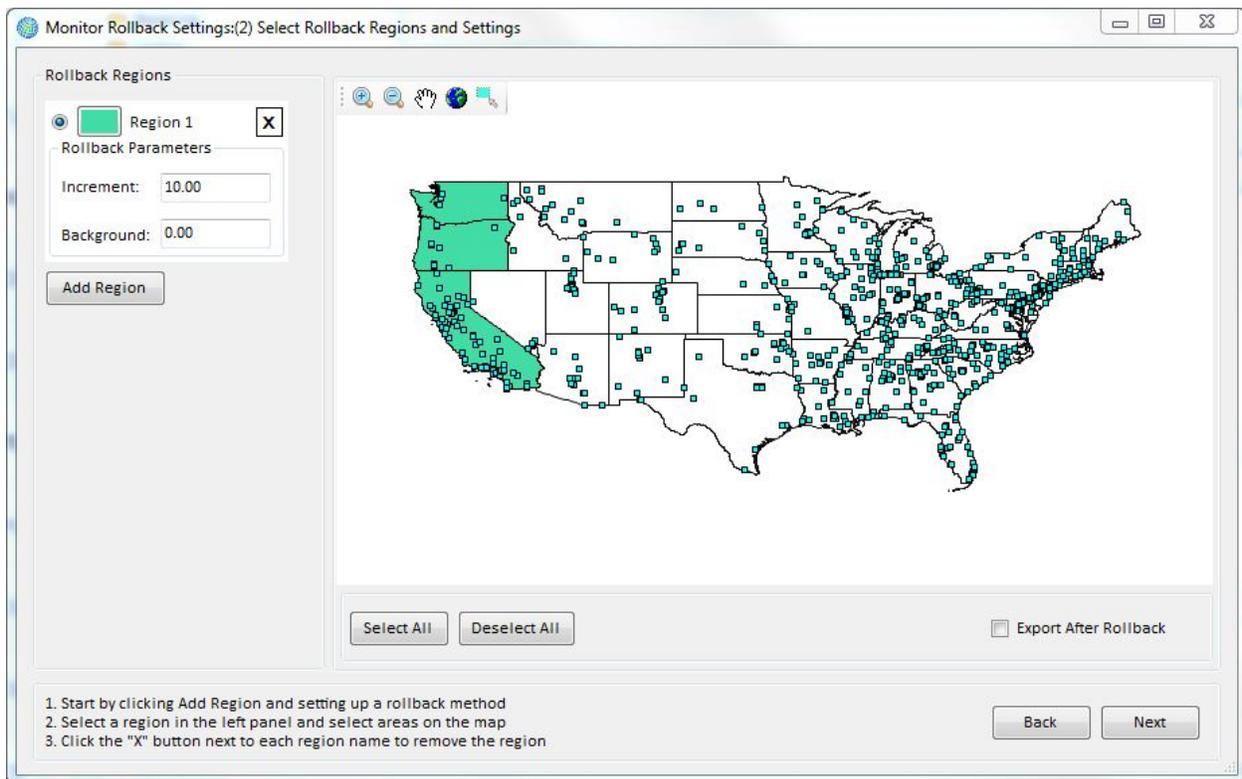
Note that there is an **Advanced** button that lets you select the **Maximum Neighbor Distance (in km)**, **Maximum Relative Neighbor Distance**, and **Weighting Approach**. The specific availability of advanced features depends on the interpolation method that you choose. The **Advanced Options** window is described in more detail in Section 5.2.4. You can view a map of the inputs to the rollback grids that you are creating, as well as the grids themselves by accessing the **Monitor Map**. To do this, click on the **Advanced** button, then the **Custom Monitor Filtering** button, and then the **Map** button (on the **Filter Monitors** window).

5.3.2 Example: Combining Three Rollback Approaches in Different Regions

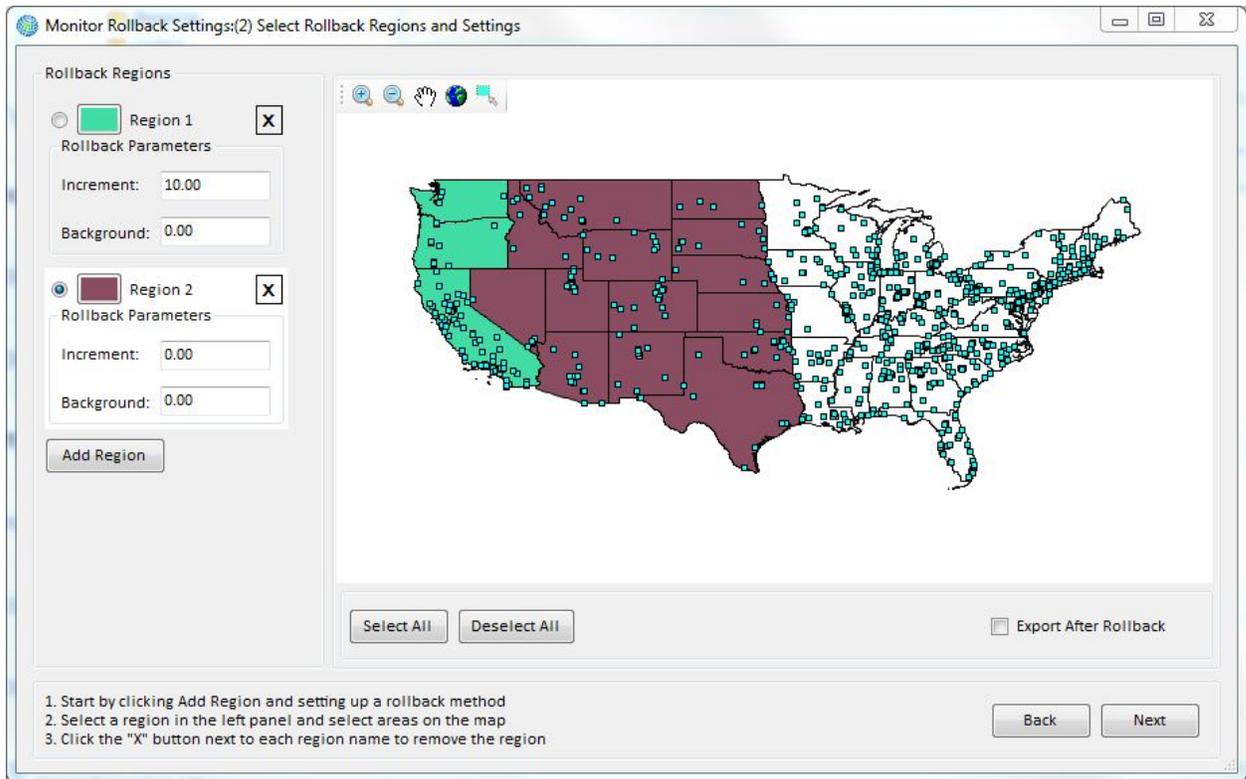
BenMAP-CE allows you to use different approaches to rolling back air quality data among regions. In this example, we'll use the *United States* setup to combine the three rollback types: *Percentage Rollback*, *Incremental Rollback*, and *Rollback to a Standard*.

Start by clicking on the **Pollutant** button in the left-hand pane of the main screen, and choosing *Ozone*. Next, click **Baseline** under **Source of Air Quality Data**. Select *Monitor Rollback* as the **Grid Creation Method**. On the **Monitor Rollback: (1) Select Monitors** window, select the **Rollback Grid Type** (*State*), **Monitor Dataset** (*EPA Standard Monitors O3*) and **Monitor Library Year** (*2000*), and click **Next**.

On the **Monitor Rollback Settings: (2) Select Rollback Regions and Settings** window, click the **Add Region** button and select the *Percentage Rollback* method. Click **OK**. Enter *10* for the **Percent**. In the previous example, we included only one state in the rollback region. In this example we want to create three regions. Click on the three western-most states to add them to the first region. The states you have added to the region will fill in, as in the picture below.



To add states with a second type of rollback, click on the **Add Region** button, choose the rollback type, and then click on the states to include in this second region, which BenMAP-CE denotes as Region 2. In this example, we have chosen an *Incremental Rollback* with an **Increment** of 5 and a **Background** of 0, and applied it to the 14 next western-most states.



The map now depicts two rollback regions. We can toggle back and forth between each region by clicking on the button on the legend on the left side of the map. Any states that have not yet been included in a region may be added to an existing region, or we may create one or more regions for these remaining states. Note that once states have been included in a rollback region, they cannot be included in a different rollback region. In our example, the three western-most states are highlighted in gray.

If you want to add or remove states from a defined region, make sure you select the appropriate region by clicking on the button to the left of that region before clicking on the map to select or deselect the state(s).

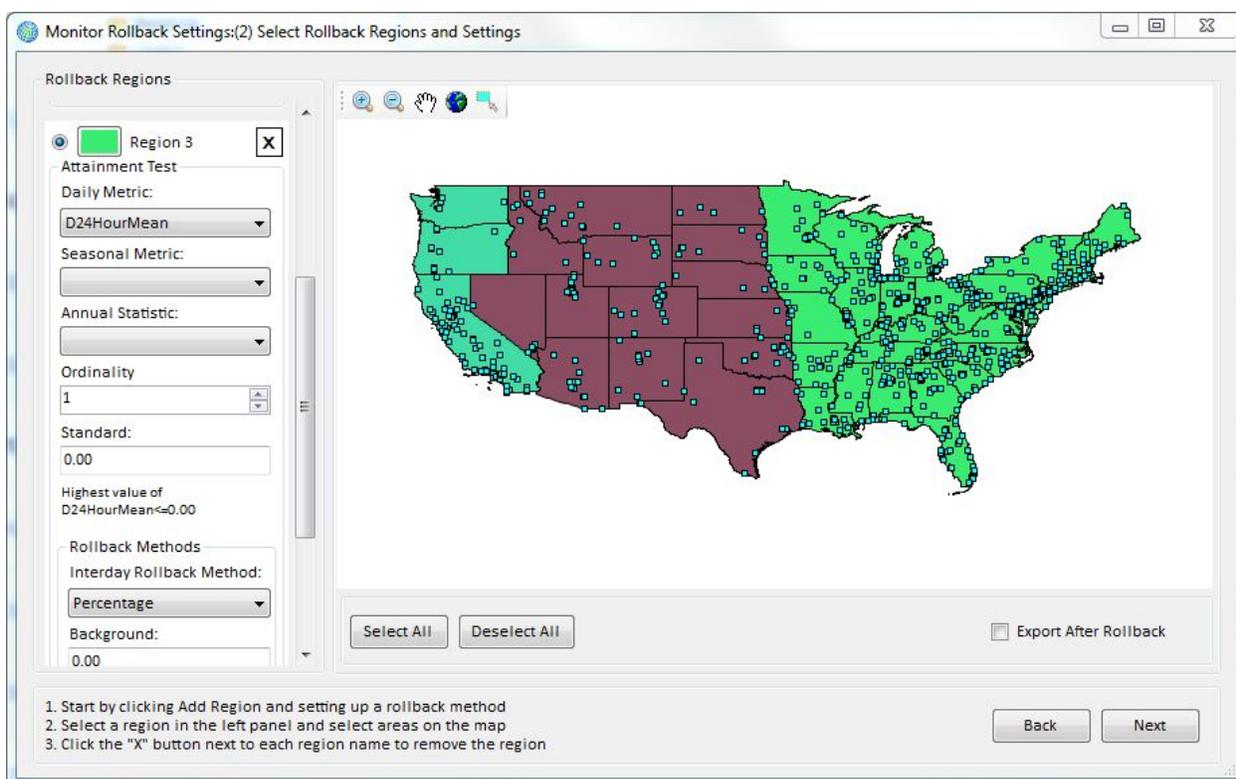
To add a third rollback type covering the rest of the states, click again on the **Add Region** button, and then choose the rollback type. However, instead of individually choosing the states, simply click the **Select All** button. This will select all of the states that are not yet included in a region, and these remaining states will now become Region 3.

In this third region, we have chosen a *Rollback to a Standard*, which involves two groups of parameters - those associated with the **Attainment Test**, which determines whether a monitor is in attainment (meets the standard), and those associated with the **Rollback Methods**, which are used to bring out-of-attainment monitors into attainment.

The **Attainment Test** parameters are **Daily Metric**, **Seasonal Metric**, **Annual Statistic**, **Ordinality**, and **Standard**. (Note: You will need to use the scroll bar to view more detail on the left side of the screen.)

In this step BenMAP-CE calculates the metric to be used to determine whether a monitor's values must be rolled back and, if so, how much (e.g., if **Metric** is *D8HourMax*, BenMAP-CE calculates the 8-hour daily maximum for each day at each monitor).

A monitor is considered in attainment if the n^{th} highest value of a daily metric specified by **Metric** is at or below the value specified by **Standard**, where n is the value specified by **Ordinality**. For example, if **Metric** is *D8HourMax*, **Ordinality** is 4, and **Standard** is 85, a monitor will be considered in attainment if the fourth highest value of the eight-hour daily maximum is at or below 85 ppb.



The **Attainment Test** can also be used for seasonal metrics (by choosing previously defined seasonal metrics from the drop-down list below **Seasonal Metric**), as well as for annual metrics (by using the drop-down list below **Annual Statistic**). For example, if you want the annual mean ozone level to stay below 60 ppb, then you would choose the daily 24 hour mean (*D24HourMean*) from the drop-down list below **Daily Metric**, choose *Mean* from the drop-down list below **Annual Statistic**, and set the **Standard** to 60. (Note that in this case **Ordinality** cannot be chosen because there is only a single annual value.)

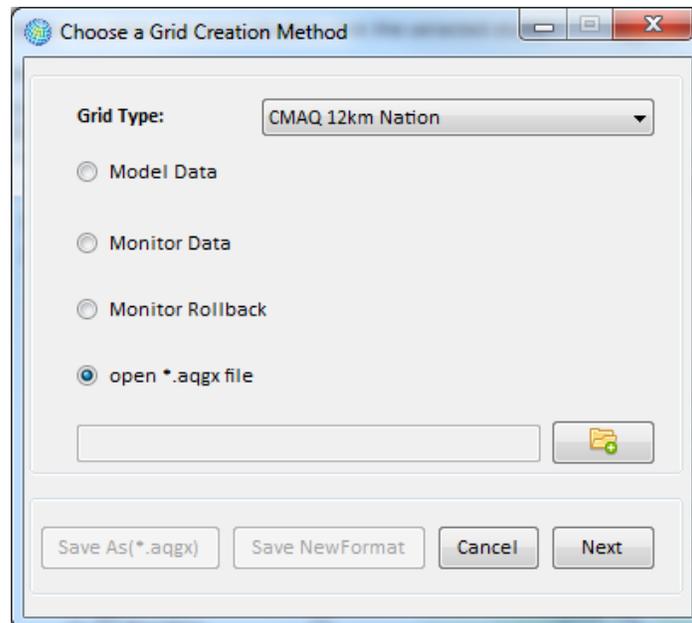
The **Rollback Methods** parameters determine the rollback procedures used to simulate out-of-attainment monitors coming into attainment:

- **Interday Rollback Method** (with associated **Background** level) – These are used to generate target values for the metric specified by the **Attainment Test**. Method types include *Percentage*, *Incremental*, and *Peak Shaving*.
- **Intraday Rollback Method** (with associated **Background** level) – These are used to adjust hourly observations to meet the target metric values generated in the previous step. Method types include *Percentage* and *Incremental*.

The methods involved for each can be somewhat complicated, so we have included a section in Appendix A: Monitor Rollback Algorithms which goes through several examples.

5.4 Open *.aqgx File

The final option for uploading an **Air Quality Grid** is to select the *Open *.aqgx File* option from the **Choose a Grid Creation Method** window.

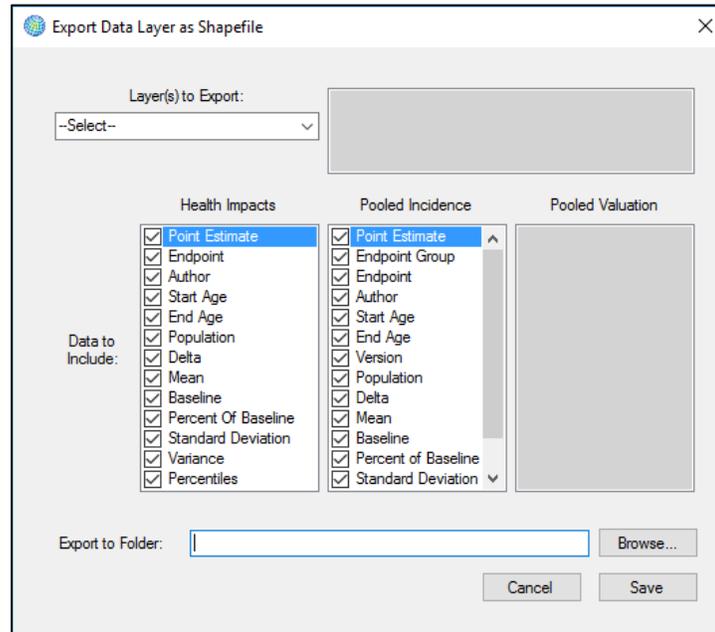


Choosing this option will activate the **Open File Browser** button located directly below this option. Click the **Open File Browser** button. This will cause an **Open** window to appear, allowing you to search for an **Air Quality Grid** (*.aqgx file) that has already been created. Select a file and click **Open**. The file path and name should appear in the box beside the **Open File Browser** button. Click **Next** to close the window and begin to create the map layer.

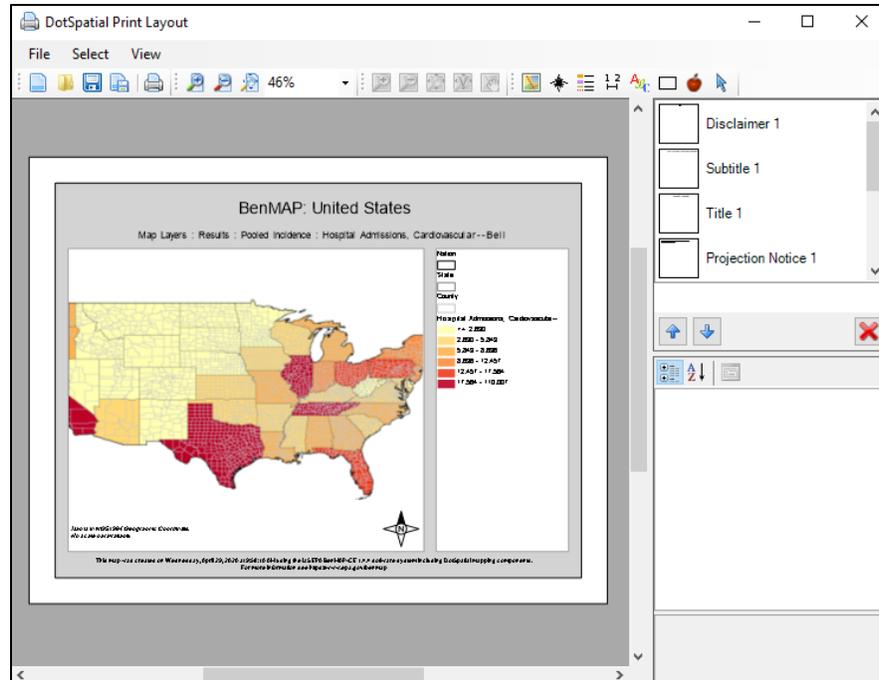
5.5 Frequently Asked Questions

How can I generate a map of an air quality grid and export it?

When viewing any of the displayed maps in the **GIS Map** tab (lower right frame of the main window), click on the GIS toolbar icon for **Save Shapefile** (looks like a 3.5-inch diskette). You can choose the Layer(s) to Export from the dropdown menu and select data to include in the exported shapefile. Follow the prompts to provide a name and location for the file. BenMAP-CE will export a set of files (.dbf, .prj, .shp, .shx) associated with the shapefile that you can use with any GIS viewer.



To export the map as an image, click the **Prepare Print Layout** icon (immediately above the **Save Shapefile** icon). This will use built-in DotSpatial GIS tools to allow you to save the map as a formatted image (.png) file.



For the Rollback to a Standard option, why are there Interday and Intraday rollback options?

The **Interday Rollback Method** option identifies the approach (e.g., *Percentage*) to reduce daily air pollution levels, in order to meet the specified standard. (In other words, there is more than one way to reduce daily pollution levels so as to meet the standard you have chosen, and BenMAP-CE lets you choose from among those approaches.) The **Background** level associated with the **Interday Rollback Method** specifies the bound, below which, BenMAP-CE will not make adjustments to daily levels.

The **Intraday Rollback Method** option is only relevant for hourly air pollution data, like ozone measurements. This option specifies the approach (e.g., *Percentage*) used by BenMAP-CE to reduce hourly air pollution levels to reach the target metric values. That

is, once you have chosen the approach to reduce daily air pollution levels, on any given day there is more than one way to reduce the hourly air pollution values to meet the targeted pollution level for that day. The **Background** level associated with the **Intraday Rollback Method** specifies the bound, below which, BenMAP-CE will not make adjustments to hourly levels.

The **Interday** and **Intraday** options are complicated. Appendix A on Monitor Rollback Algorithms explains these options in more detail and gives some numerical examples.

Can I use air quality grids based on different Grid Types in the baseline and control scenarios?

No. In any given analysis, you need to use the same Grid Type in the baseline and control scenarios.

Can I use air quality grids of the same Grid Type but based on different Grid Creation Methods?

Yes. In any given analysis, you may use air quality grids made with different methods. Air quality grids made with Model Data and Monitor Data may be used interchangeably, if desired. Similarly, air quality grids made with different interpolation methods may be compared. However, it generally is not recommended to create grids with different methods and use them in the same analysis.

Can I do an analysis with multiple pollutants?

You can currently only estimate impacts one pollutant at a time; however, BenMAP-CE allows you to aggregate the results of more than one pollutant. This is discussed in Chapter 7: Aggregating, Pooling, and Valuing.

Why does it take so long to generate an Ozone Air Quality grid if there are a lot of grid cells?

It can take a long time to create an air quality grid because the file being generated can be quite large. In some cases, air quality grids can be several hundred megabytes in size. (One reason the ozone files are large is that the definition of ozone has, by default, four metrics. If you do not need all of the default metrics for the health impact functions in your database, then delete the unneeded metrics and BenMAP-CE will run faster and generate smaller air quality grids. This is an advanced step, so do not do it if you are unsure.) The type of computer you use can also affect processing speeds. Refer to Chapter 1, Section 1.3 for recommended hardware specifications.

How do I access data in an Air Quality Surface?

You can access the data in an air quality grid by going to the **Tools** menu and choosing the **Export Air Quality Surface** option. (The **Tools** menu is available on the toolbar of the main BenMAP-CE window.) Locate the air quality grid from which you want to

export air quality data and then give a name to your exported file. BenMAP-CE will generate a text file that you can then examine. This is discussed in detail in Chapter 3, Section 3.2.1 (Tools).

How do I perform a rollback to simulate attainment with an annual *and* daily PM_{2.5} standard?

Unfortunately, BenMAP-CE will roll back to either an annual standard or a daily standard—but not both. If this feature is of interest to you, please contact the BenMAP-CE developers at benmap@epa.gov.

Chapter 6

Estimating Incidence

In this chapter...

- **Get an overview of how BenMAP-CE estimates the incidence of health outcomes.**
- **Learn to create a health impact configuration.**
- **Learn about baseline and control scenarios.**
- **Learn the difference between Point Mode and the Monte Carlo analysis options.**
- **Learn how to run, save, and re-open a configuration.**
- **Learn how to view and export Incidence Results.**

To estimate changes in the incidence (or, counts) of adverse health effects from air pollution, you will need to create and run a BenMAP-CE configuration file (*.cfgx). A configuration is a reusable file that specifies the air quality grids, health impact functions, population data, and other parameters necessary for an analysis. It is a record of the choices you make in estimating the change in adverse health effects between a baseline and control scenario. The choices include the following:

- The pollutant and air quality grids for the baseline and control scenarios;
- The year for the analysis;
- The population dataset for the analysis;
- The health impact functions to be used in estimating adverse health effects; and Whether the analysis will focus on a single "point" estimate (Point Mode), or a range of results that mirror the variability in the inputs to the health impact functions (Monte Carlo-generated percentiles).

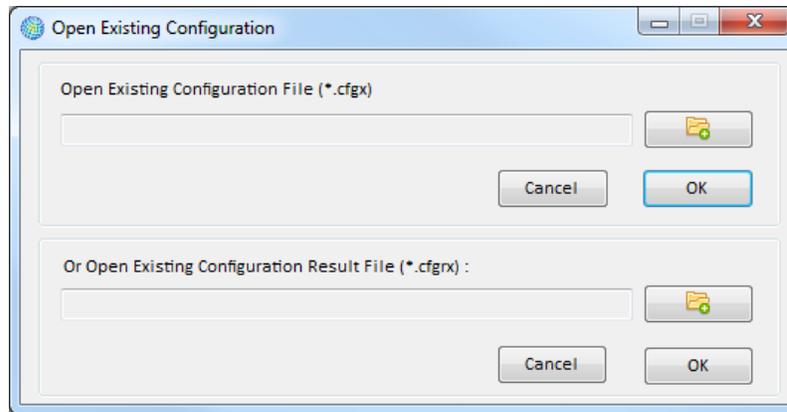
Fundamental Concept – Configuration Files

A **Health Impact Configuration** file (*.cfgx) includes all the user-specified data and choices for a BenMAP-CE run made prior to executing the health impact function step. This includes specified air quality grids, air quality surfaces, population data, health impact functions, and incidence datasets. This file is not saved automatically but can be saved by clicking the appropriate button. Configuration files can be useful for running BenMAP-CE in batch mode via the Command Line feature (see Appendix L).

Once you execute the Health Impact Functions, BenMAP will save a **Health Impact Configuration Results** file (*.cfgrx). This file contains the same information as the .cfgx file, plus the health impacts results from running the health impact functions on the specified air quality surfaces.

BenMAP-CE gives you flexibility in creating, editing, and saving configuration files. You can open an already existing configuration and proceed directly to estimate incidence. Or, you can create a new file, and then estimate incidence. In addition, you may save any edits made to existing configuration files. After calculating the change in adverse health effects, BenMAP-CE saves the results in a "configuration results" file with a *.cfgrx* extension. The results obtained from running a configuration are sometimes referred to as "raw" results because they represent the estimated change in incidence for each air quality grid cell in a given scenario; they have not been aggregated, pooled, or valued (see Chapter 7).

To load a previously saved configuration, click **Estimate Health Impacts**, which is beside **Step 2** in the BenMAP-CE tree menu (on the left side of the main window). An **Open Existing Configuration** window will appear, as shown below:



To open an existing Configuration file, click the top file browser button. To open an existing Configuration Result file, click the bottom file browser button. After selecting either of these options, search and select a file to load in the **Open** window. The file path and name should appear in the box next to the file browser button. Once you have selected the desired option, and are satisfied with the file selection, click **OK**.

6.1 Introduction to Estimating Health Incidence

Health impact functions relate a change in the concentration of a pollutant to a change in the incidence of a health endpoint (i.e., premature mortality or work-loss days). It is typically derived from the estimated relationship between the concentration of a pollutant and the adverse health effects suffered by a given population in an epidemiology study. For example, the pollutant concentration being measured may be particulate matter (PM_{2.5}), and the population response may be daily premature deaths. For the purposes of estimating benefits, we are not interested in the health impact function itself, but rather 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 10µg/m³, 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

Fundamental Concept: Health Impact Function or Concentration-Response Function

A **health impact function** calculates 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 (using a specified metric such as annual D24HourMean); 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.

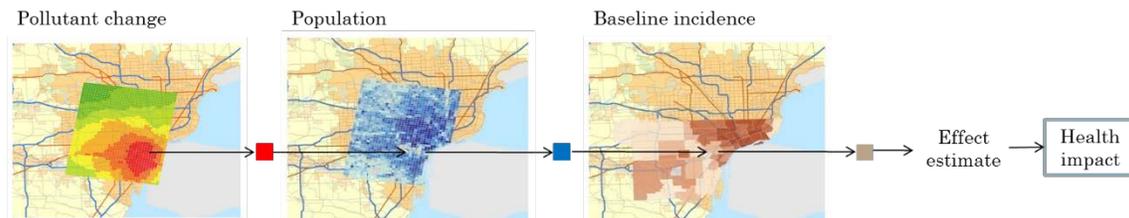
The coefficient for the health impact function is known as Beta (β) and is derived from epidemiological studies. The value of β typically represents the percent change in a given adverse health impact per unit of pollution.

Health impact functions are derived from **concentration-response (C-R) functions**, 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.

increment between the baseline scenario and the control scenario. This increment and a gridded population dataset are then used in health impact functions to calculate the change in health incidence that would result from this change in pollution. These functions are based on epidemiological studies and can be selected by the user. Typically, these health incidence results show the decrease in health incidence (e.g., the decrease in asthma, bronchitis, mortality) due to a decrease in pollution. In BenMAP-CE, the selected health impact functions are stored in configurations, which can be re-used over and over again.

Steps to Calculating Health Impacts

$$\Delta Y = Y_0 (1 - e^{-\beta \Delta PM}) * Pop$$



6.2 Create a Health Impact Configuration

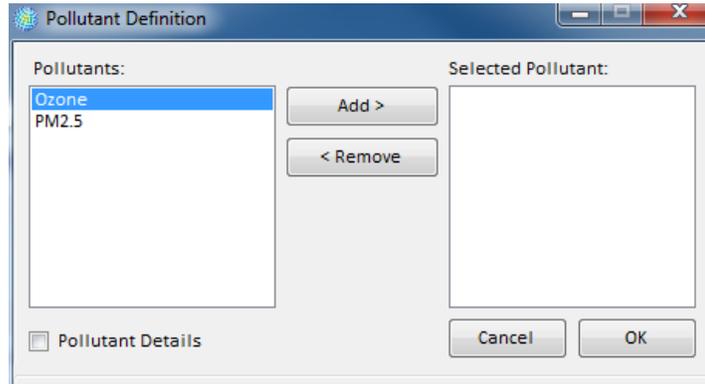
There are three major steps to creating a new configuration. First, select a **Pollutant** and specify the **Source of Air Quality Data** for the **Baseline** and the **Control** layers using the BenMAP-CE tree menu on the left side of the main window. Next, choose the **Population Dataset** and **Health Impact Functions** that you want to use in your analysis. Finally, using the **Advanced** button on the **Health Impact Functions** window, specify whether BenMAP-CE will run in **Point Mode** or perform a Monte-Carlo analysis (default setting). (See **Section 6.2.3 Advanced Configuration Settings** for additional detail.)

Note: As you move through the analysis steps, the BenMAP-CE tree menu will update its “**stoplight**” colors to reflect progress. **Yellow** indicates an operation has not yet started. **Green** indicates that an operation has been successfully completed. **Red** indicates that an operation completed, but you may need to re-run this step.

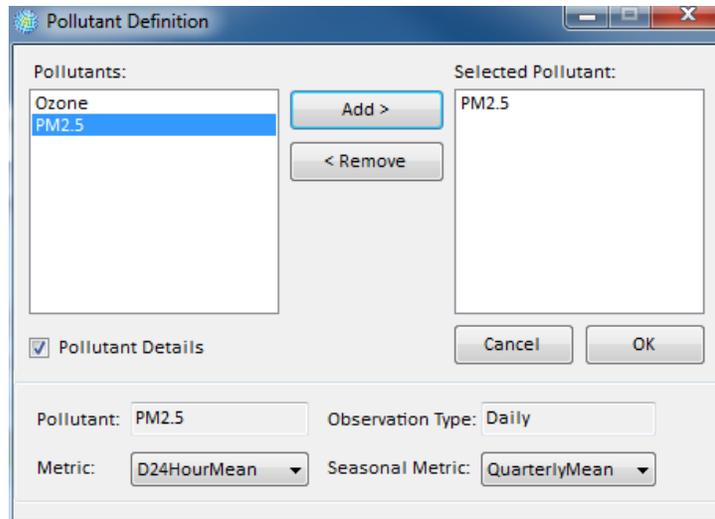
6.2.1 Air Quality Surfaces – Pollutants and Air Quality Grids

Using the BenMAP-CE tree menu, specify the pollutant(s) of interest. Click the **Pollutant** tree menu item to open the **Pollutant Definition** window. Select the pollutant(s) of interest by clicking on an item in the **Pollutants** box (left side) and then clicking the **Add** button to move the pollutant(s) to the **Selected Pollutant** box. To deselect a pollutant and remove it from the **Selected Pollutant** box, click on the item in

the **Selected Pollutant** box and click the **Remove** button. You can also add a pollutant by dragging it from the left-hand box to the right-hand box.

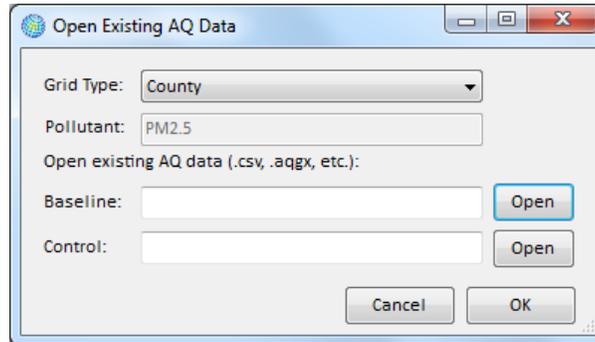


If you want to view or modify any pollutant details, click the box next to *Pollutant Details* and the window will expand to display details and options for the highlighted **Pollutant** (left box). Note that the details are based on the defined **Pollutant Definitions** (see Chapter 4: Loading Data).



Once your pollutant is selected, click **OK** to close the form. The stoplight for **Pollutant** will change from yellow to green when the operation is successfully completed.

Next, select air quality grids for the **Baseline** file and **Control** file. You may choose existing air quality grids by double-clicking the **Source of Air Quality Data** from the tree menu. The **Open Existing AQ Data** window will be displayed. Select air quality grids, designated with an '.aqgx' extension, for **Baseline** and **Control**. To create a new air quality grid, follow the steps outlined in Chapter 5: Creating Air Quality Grids.



The **Baseline** file contains the air quality metrics for the scenario assumed to occur without any change in policy. The **Control** file specifies the air quality metrics assuming that some type of policy or change has been implemented. The air quality grids should be for the same pollutant, and should also be based on the same **Grid Type**.

If you choose a particular **Grid Type** (e.g., *County*) for the **Baseline** file, then the same grid type must be used in the **Control** file. Conversely, it would not be possible to use *County* grid-type in the **Baseline** and a *Tract* grid-type in the **Control** file.

Fundamental Concept – Baseline and Control Scenarios

BenMAP-CE requires both a **Baseline Scenario** air quality surface and **Control Scenario** air quality surface to estimate the effects of a change in air quality (**Delta**). Both baseline and scenario air quality surfaces are required regardless of whether you use modeling data or monitoring data.

- The **Baseline Scenario** characterizes the air quality levels observed or expected in the absence of the policy change you are evaluating. The baseline is sometimes referred to as “Business as Usual.” The Baseline scenario is usually considered to be the reference scenario against which to compare a potential scenario characterized by the implementation of regulations.
- The **Control Scenario** in BenMAP is the scenario in which emissions from one or more source sectors are changed (increased or decreased) from the Baseline scenario. The Control scenario usually represents expected air quality levels after a new regulation or set of regulations has been implemented.

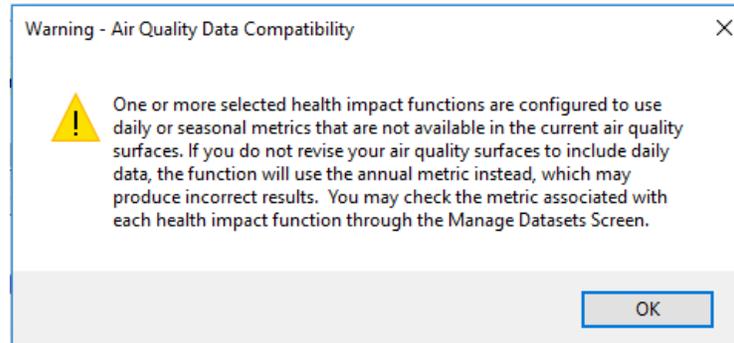
The air quality **Delta** is the change in air pollution between the Baseline air quality grid and the Control air quality grid (Baseline minus Control). BenMAP-CE uses the air quality **Delta** as the input to the health impact function.

The **Pollutant** specified in the air quality grids determines 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 one (or both) of the air quality grids (see

the information in Chapter 3 on monitor and model data formats for more information on how this can occur) **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 and the annual metric will be used instead.

Warning

BenMAP-CE will evaluate the compatibility between provided air quality metrics and health impact functions. For example, if the air quality surface provides an annual pollutant metric for a health impact function that uses a daily pollutant metric, BenMAP-CE will provide a warning message (shown below).

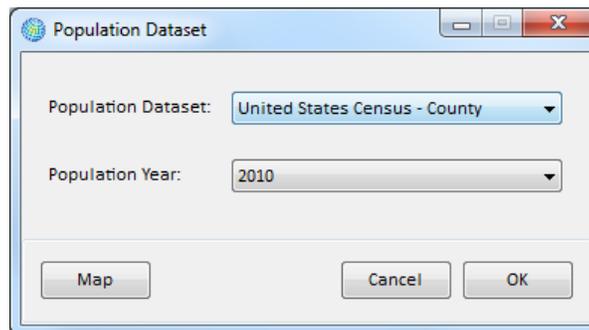


Once the **Baseline** and **Control** files are selected, click **OK** to close the form. The stoplights for each of these under **Source of Air Quality Data** will change from yellow to green when the operation is successfully completed.

6.2.2 Estimate Health Impacts – Population and Health Impact Functions

The second step in creating a health impact configuration is to select the **Population Dataset**, **Population Year** and **Health Impact Functions**. If you want to open an existing configuration, double-click the **Estimate Health Impacts** tree menu item and select the file to open. Follow instructions from **Section 3.1.2 Estimate Health Impacts**.

To continue creating a new configuration, double-click **Population Dataset** in the tree menu. Here, you can choose the **Population Dataset** and **Population Year** that will be used in the analysis. The values in the drop-down list for **Population Year** depend on the range of values in your **Population Dataset**. (See Chapter 4: Loading Data.)



Clicking the **Map** option in the bottom left hand corner will open a separate **Population Map** window. (Warning: this procedure can require a significant amount of time to complete as it creates the population grid and loads the population growth weights). This provides a map of your selected population. If you want to view a different population subgroup based on race, gender, ethnicity, or age range, choose from the available drop-down list (this runs fairly quickly as it uses the same population grid already loaded). The map will refresh automatically.

To change the **Population Dataset** or **Population Year**, click on their respective drop-down list and click **Draw** to update the map and display new results. Warning: This requires BenMAP-CE to recalculate the grid.

Click the close ('x') button at the top of the **Population Map** window to close it.

Clicking **OK** on the **Population Dataset** window will close the window and change the stoplight color to green in the tree menu.

Next, double-click **Health Impact Functions** on the tree menu to display the **Health Impact Functions** window.

Health Impact Functions

Selected Health Impact Functions

Filter Dataset: EPA Standard Health Functions (2021) | Filter Endpoint Group: All | Filter: | Groups | Add Selected

Endpoint Group	Endpoint	Pollutant	Geographic Area	Author	Start Age	End Age	Beta Distribution	Metric	Season
Acute Myocardial Infarction, Nonfatal [25 items]									
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Peters e...	18	24	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	45	54	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	25	44	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	18	24	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	99	99	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	65	64	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	45	54	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	25	44	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	18	24	Normal	D24HourMe...	

Selected Health Impact Functions

Endpoint Group	Endpoint	Pollutant	Geographic Area	Author	Start Age	End Age	Race	Ethnicity	Gender	Incidence Dataset
Selected items to be shown here										

Delete Selected | Advanced | Cancel | Save As (*.cfgx) | Run

The **Health Impact Functions** window is split into two display frames. The upper frame presents the **Available Health Impact Functions**, which you may select, and the lower frame shows the **Selected Health Impact Functions** that you have already selected. Both frames have a tree structure and the ability to change the order of the fields for easy viewing of the functions.

Above the **Available Health Impact Functions** frame, there are a series of buttons that give you the option to select different datasets and filter options for each one.

To add studies to your configuration, simply click to select the health impact functions of interest in the top frame and drag them down to the lower frame. You can do this for blocks of health impact functions by selecting the *Groups* option, clicking on the header of an endpoint group, and dragging the entire endpoint group into the lower frame. And, you can use the options to **Filter Dataset**, **Filter Endpoint Group**, and **Filter** (by keyword) to filter the list according to your preference. Once you are satisfied with the filter, click the **Add Selected** button to apply the selection to the list of selected functions.

BenMAP-CE Guidance and Best Practices

When selecting HIFs for your analysis, one option is to use the ozone and PM_{2.5} configurations used by EPA. These are available on the BenMAP-CE website (<http://www2.epa.gov/benmap/benmap-community-edition>). These functions are derived from the epidemiology literature described in the appendices to this user manual.

If BenMAP-CE does not have pre-installed functions for a pollutant or a health impact that you would like to study, you may need to develop your own health impact function(s) which involves a careful review of the epidemiological literature. We recommend consulting with an epidemiologist or other public health professional if you need to develop your own functions.

Health Impact Functions

Selected Health Impact Functions

Filter Dataset: EPA Standard Health Functions (2021) | Filter Endpoint Group: All | Filter: | Groups | Add Selected

Endpoint Group	Endpoint	Pollutant	Geographic Area	Author	Start Age	End Age	Beta Distribution	Metric	Season
Acute Myocardial Infarction, Nonfatal [25 items]									
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Peters e...	18	24	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	45	54	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	25	44	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	18	24	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	65	99	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	55	64	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	45	54	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	25	44	Normal	D24HourMe...	
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5		Zanobe...	18	24	Normal	D24HourMe...	

Selected Health Impact Functions (3)

Endpoint Group	Endpoint	Pollutant	Geographic Area	Author	Start Age	End Age	Race	Ethnicity	Gender	Incidence Dataset
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5	Everywhere	Peters e...	18	24				Other Incidence (201
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5	Everywhere	Pope et ...	18	24				Other Incidence (201
Acute Myocardial Infa...	Acute Myocardial ...	PM2.5	Everywhere	Zanobe...	18	24				Other Incidence (201

Delete Selected | Advanced | Cancel | Save As (*.cfgx) | Run

If you want to delete some of the health impact functions that you added to your configuration, just highlight the studies of interest and hit the **Delete** key on your keyboard (or click the **Delete Selected** button on the form).

BenMAP-CE displays information for selected studies in two broad categories: **Function Identification** (column headings in black text) and **Function Parameters** (column headings in pink text). The **Function Identification** includes information such as the **Endpoint Group**, **Endpoint**, **Metric**, **Location**, and other variables. This identification information is useful when distinguishing between multiple health impact functions. The **Function Parameters** include those variables that you may directly edit: **Race**, **Ethnicity**, **Gender**, **Start Age**, **End Age**, **Geographic Area**, **Incidence Dataset**, **Prevalence Dataset**, and **Variable Dataset**.

Clicking on a column header will sort the health impact functions according to that variable. For example, clicking the **Start Age** column will sort the functions by youngest to oldest **Start Age**. You can add the same study to the selection multiple times, and then make edits, in order to be able to calculate the impact of changes in these variables of the function. For example, you can perform an analysis using multiple versions of the same function with different age ranges. To edit the default **Start Age** and **End Age**, just highlight the appropriate cell and type in the desired age values. Keep in mind that these ages represent

inclusive age bounds, so if you type in '10' and '12' this will include all children ages ten, eleven, and twelve years old. If you want to apply a single age year (e.g., only children who are eleven years old), then type the same year (e.g., '11') in both the **Start Age** and

Warning

After selecting your health impact functions, Check the *Incidence Dataset* column in the bottom window and make sure that the correct incidence dataset is selected for each health impact function. This is a step which is often missed and can result in errors. Recall that in order to use a baseline incidence rate with a particular health impact function, the endpoint group and endpoint of the baseline incidence rate must match the endpoint group and endpoint of the health impact function exactly.

Fundamental Concepts – Incidence, Prevalence, and Variable Datasets

The **Incidence Dataset** contains **baseline incidence rates**, or the average number of health outcomes (e.g., number of hospital visits or deaths) per person, per unit of time (generally a day or a year)—from air pollution as well as all other causes. These rates are used to calculate the baseline number of adverse health effects that are occurring in a population.

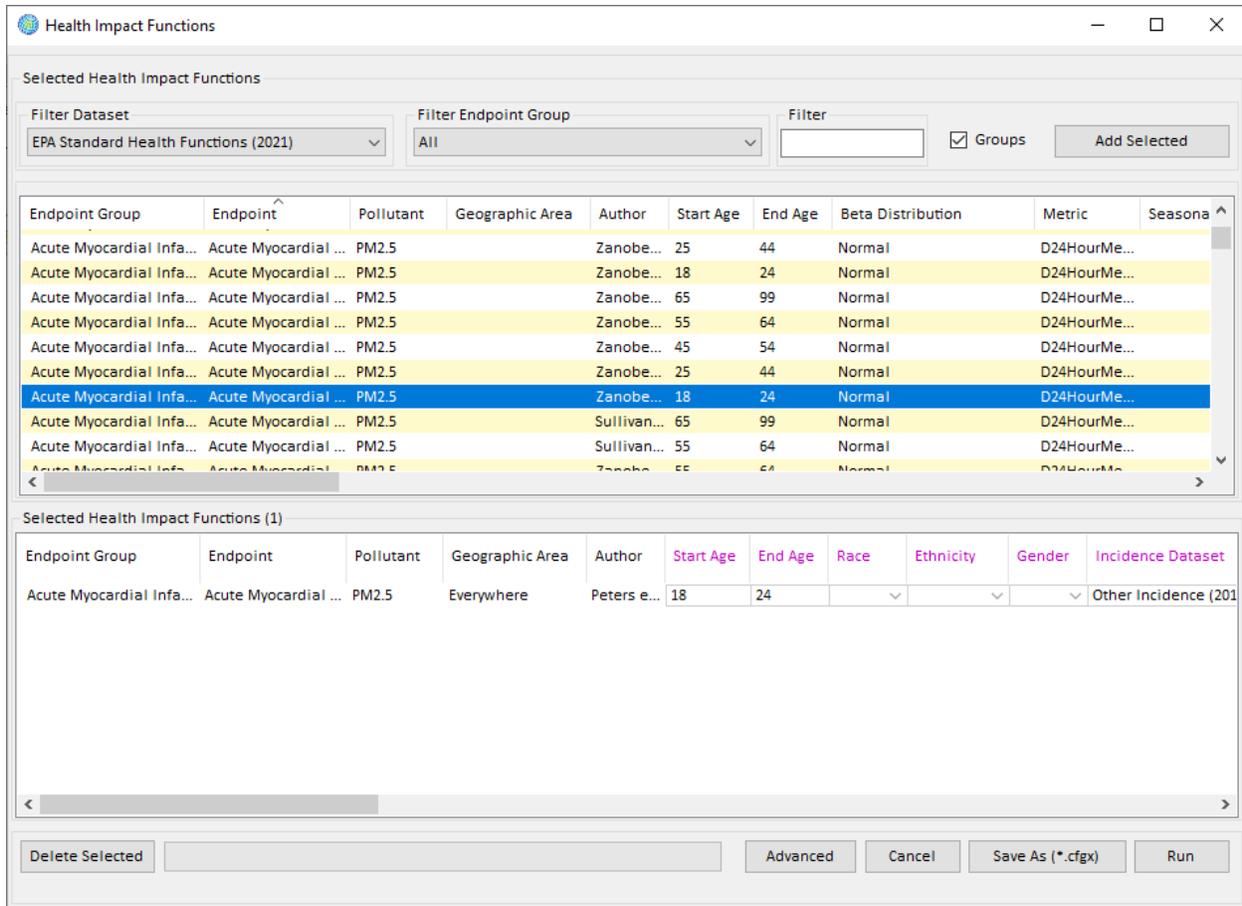
The **Prevalence Dataset** contains **prevalence rates**, or the percentage of individuals in a given population who already have a given adverse health condition. Prevalence rates are used to calculate changes in health conditions among those who already have a health condition, such as asthmatics experiencing asthma symptoms exacerbated by air pollution.

The **Variable Dataset** contains sociodemographic and economic data not contained in other setup datasets that may be used in health impact functions and valuation functions. Examples of socioeconomic variables that might be included in **Variable Data** include median household income and percentage of population living below the poverty line. Variables included in the dataset must be associated with a particular **Grid Definition**).

Note that the accuracy of the populations calculated for these age ranges will depend on the specificity of the population data present in your selected population dataset. Use the drop-down lists in the **Incidence Dataset**, **Prevalence Dataset**, and **Variable Dataset** fields.

Added in version 1.4, the **Geographic Area** field allows users to assign health impact functions to either the entire area of analysis (“Everywhere”), to a specific subset of that area

(defined by a specific grid definition), or to all areas outside of one or more specified geographic areas (“Elsewhere”). Note that if an air quality cell intersects multiple geographic areas, BenMAP-CE will calculate health impacts for the geographic area containing the majority of the air quality cell. To access these options, select the drop-down list under the **Geographic Area** header. Please note that users must specify whether grid definitions can be linked to health impact functions when defining new grid definitions or modifying existing grid definitions.



6.2.3 Advanced Configuration Settings

Clicking the **Advanced** button at the bottom of the **Health Impact Functions** window gives you access to a number of options for tailoring your assessment.

The Point Mode and Monte Carlo options allow you to generate an average incidence estimate, or a range of results that mirror the uncertainty in the inputs to the health impact functions. By default, BenMAP will run in Monte Carlo mode. If you select the option for *Run in Point Mode*, BenMAP-CE uses the mean values of the inputs to the health impact functions, and generates a single "point estimate" of the change in adverse health effects.

With the Monte Carlo (default) option, you can generate a number of estimates that mirror the variability in the inputs to the health impact functions. The Monte Carlo option allows you to generate specific percentiles along the estimated incidence distribution. For example, if you specify **20 Percentiles** (default value), then BenMAP-CE will generate incidence estimates of the 2.5th percentile, 7.5th percentile, and so on, up through the 97.5th percentile. The number of points suggested in the drop down menu for **Percentiles** varies between 10 and 100. The greater the number of chosen

points, the greater the amount of time BenMAP-CE will need to process the results. The relationship between the number of points and time to process is essentially linear, so a doubling of the number of points would double the processing time.

In the Monte Carlo (default) option, the program will perform 10,000 iterations to calculate uncertainty. You may change this number, but the greater the number of iterations, the longer the processing time.

If you choose to *Run in Point Mode*, the field for **Percentile** points for Monte Carlo sampling is disabled and will be ignored (treated as zero). However, with the default Monte Carlo option, the program will still report a point estimate. As discussed in Chapter 7 on Aggregation, Pooling, and Valuation, by choosing the Point Mode, you limit your ability to pool the results. You cannot conduct fixed effect/random effects pooling, or any other procedure that depends on knowing the distribution, or the range of variability of the incidence estimates.

The **Air Quality Threshold** indicates the minimum air quality value that BenMAP-CE will use to quantify health impacts. That is, air quality metrics below the threshold will be replaced with the threshold value. With a threshold of zero, there is no impact on the estimates generated by the health impact functions. However, as the threshold increases, then it will have a progressively larger impact on the incidence estimate. The **Air Quality Threshold** option allows you to explore the impact of any given threshold on the incidence estimate. This can also be useful for scenarios where you might want to know the incidence associated with changes in air quality occurring only above a standard.

If you are estimating impacts for specific population subgroups (e.g., stratification by gender, age, race, or ethnicity), BenMAP-CE provides two options for calculating baseline incidence:

- 1) You can use incidence rates averaged over gender, race, and ethnicity (default). (This option must be selected for the rates supplied with BenMAP-CE, as they are not stratified by gender/race/ethnicity).
- 2) You can use incidence rates that match the gender, race, and ethnicity selected with your health impact function. (You must import stratified rates for use with this option). If you select this option and have not imported rates that match each subgroup, the groups without incidence rates will not be included in the calculations and will have point estimates of zero.

It is recommended that users select this second option if your incidence/prevalence data include overlapping groups. For example, the asthma exacerbation prevalence rate for ages 5-17 has two race groups, one is “ALL” with value 0.107 and the other is “BLACK” with value 0.177.

If the first option (above) is selected, BenMAP will use the arithmetic mean of both values (0.142) as the prevalence rate in its calculations, regardless of whether users select “ALL” or “BLACK” as the race category—or left the race category blank.

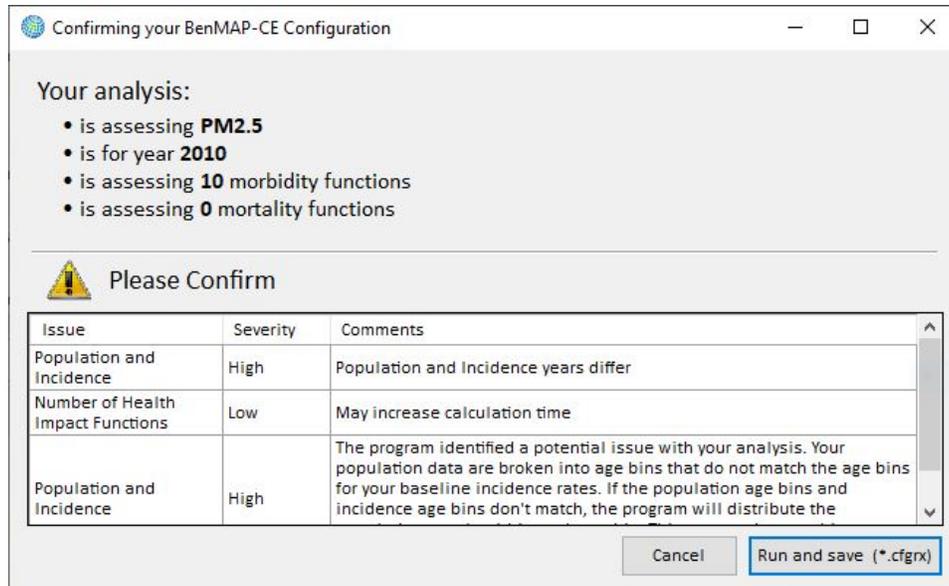
If the second option is selected, and if users select “ALL” as the race category (or leave it blank), BenMAP will first check if this dataset has overlapping race groups to avoid double counting of individuals in incidence or prevalence calculations. In this example, BenMAP will use the “ALL” prevalence rate of 0.107, which exactly matches the name of the selected race. Thus, it is recommended that users select the second option unless the user is certain that incidence or prevalence rate categories do not overlap.

After making selections for calculating impact functions, BenMAP-CE allows you to save the configuration for future use. Click the **Save As (*.cfgx)** button and specify a file with a *.cfgx* extension. Advanced settings options will be displayed in the Audit Trail.

6.3 Run Health Impact Configuration

To execute the calculation of incidence for the health impact functions in the configuration, click the **Run** button on the bottom right-hand corner of the **Health Impact Functions** window.

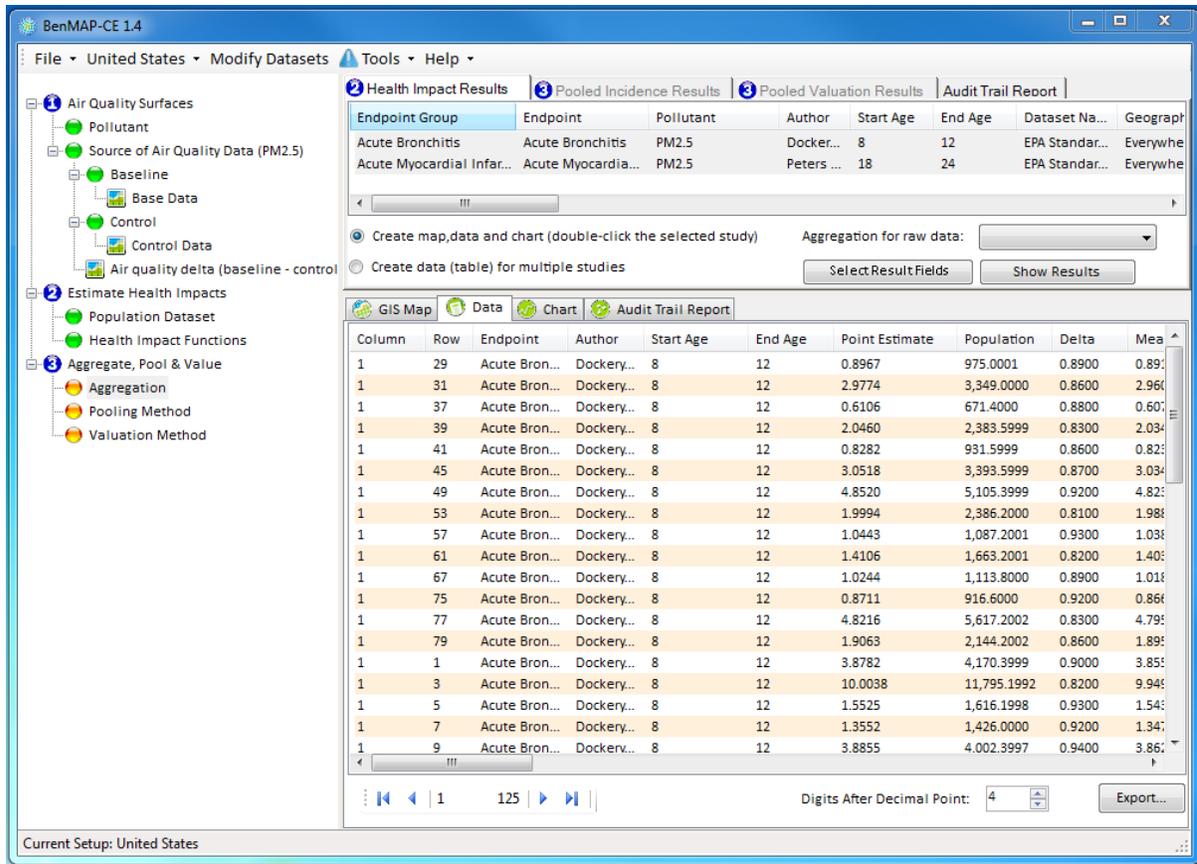
A confirmation window will pop up to notify users of key data points and raise potential issues with the run, shown below. To proceed, weigh these limitations and click **Run and save (*.cgfrx)**. BenMAP-CE will require that you specify a file in which to save the results, with a *.cgfrx* extension.



6.4 View and Export Health Impact Results

The **Health Impact Results** report gives you the opportunity to examine the incidence results of each health impact function applied at the grid-cell level, or temporarily aggregate them to, say, the state or national level. The configuration results files (*.cgrx*) contain “raw” health impact estimates that you have not yet aggregated, pooled, or valued.

To begin, click the **Health Impact Results** tab in the upper portion of the main window. To display incidence results for a single study (i.e., health impact function), double-click on the study of interest (or select the study and click the **Show Results** button). Your results will be generated and displayed in a results table (on the **Data** tab). If you choose a single study, you will also have options to view results on the **GIS Map** tab and in a simple bar chart (view in **Chart** tab.) To change the selected study, double-click on a different study choice or select the study and click the **Show Results** button to refresh the display.



The Health Impact Results tab provides a simple tool to aggregate the raw incidence results. You may select an aggregation level from the **Aggregation for raw data** drop-down list. If you do so, the **Data** table will refresh to display the incidence results aggregated to the selected level.

If you check the option to *Create data (table) for multiple studies*, results will only be available in the **Data** tab. For example, you might want to select three different studies and view aggregated results at the national level. As you modify your choices, the display will be updated accordingly.

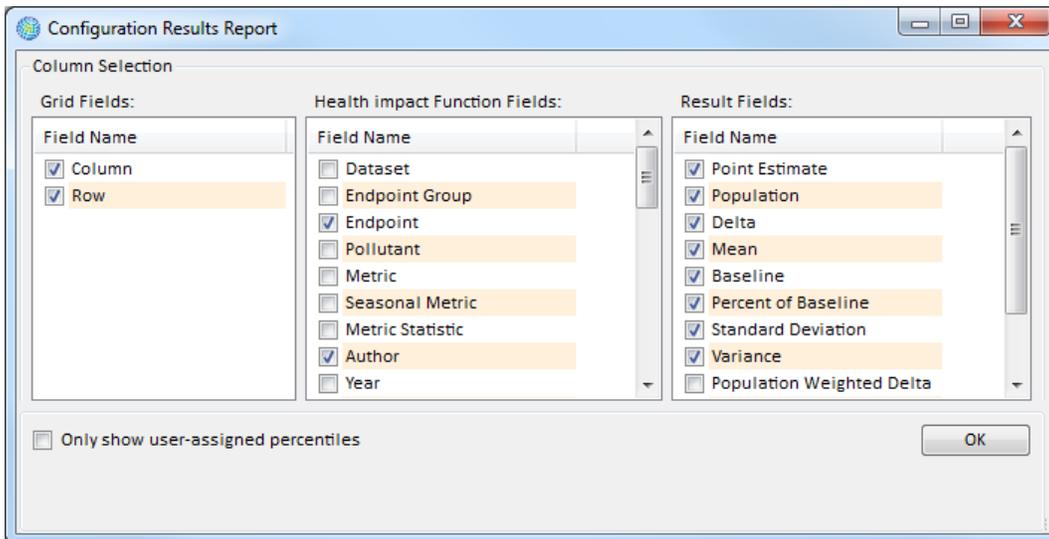
On the **Data** tab, clicking the **Select Result Fields** button opens a **Configuration Results Report** window that allows you to choose the columns that will appear in the results table.

BenMAP-CE Guidance and Best Practices

Below is a list of common culprits that may result in BenMAP-CE calculating a point estimate of zero. If you are generating zero results, begin by double checking the following:

- Did you choose the correct pollutant?
- Does the pollutant definition contain analogous metrics as the health impact function?
- Does the air quality delta (baseline – control) show a change in air quality concentrations? Does it show the necessary pollutant metrics based on the HIF?
- Does the Control scenario have lower air quality values than the Baseline scenario?
- Did you do less than 100% monitor rollback?
- Does the Population Dataset match the location of the Grid Definition?
- Does the Baseline Incidence dataset match the Population Dataset?

- **Grid Fields** permit the inclusion of *Column* and *Row* fields, which can be helpful in identifying the grid-cell of a particular line in the report. For example, when results have been temporarily aggregated to the national level.
- **Health Impact Function Fields** permit the inclusion of various fields which describe or define a function (e.g., *Endpoint Group*, *Endpoint*, *Pollutant*, *Metric*, *Author*, *Year*, *Start Age*, *End Age*, *Gender*, *Race*, *Ethnicity*, *Beta*, *Beta Distribution*). These fields can be helpful in identifying the health impact function associated with a particular line in the report.
- **Result Fields** permit the inclusion of fields associated with results of this analysis (e.g., *Point Estimate*, *Population*, *Delta*, *Mean*, *Percentiles*).



At the bottom of the **Data** tab, there is also an option to specify the number of digits that appear after the decimal point (click the up or down arrows to edit the number beside the **Digits After Decimal Point** field, or type a number directly in the box). Also at the bottom of the **Data** tab is an option to display **All Percentiles** rather than the default 2.5th and 97.5th percentiles.

Table 6-1 provides a summary of the optional fields that have not been previously described in this section.

Table 6-1. Selected Variables in the Reports Based on the APVR file

Variable	Description
Column	The column of the grid cell of the result. For grid cell level results, this is the column of the grid cell. For county and state level results, this is the state FIPS code. For national results, this is always 1.
Row	The row of the grid cell of the result. For grid cell level results, this is the row of the grid cell. For U.S. county results, this is the county FIPS code. For U.S. state and national results, this is always 1.
Dataset	Specifies the dataset from which a health impact function was chosen.
Population	Population provides the number of persons used in the health impact function calculation.
Delta	The difference between the baseline and control scenarios for the metric used in the health impact function. Calculated by subtracting the metric value in the control scenario from the metric value in the baseline scenario.
Point Estimate	The point estimate for the result from the health impact function. The point estimate is generally based on the mean estimate of the "Beta" from the health impact function.
Mean	Mean of the points in the Monte Carlo-generated distribution for this result. The mean is set to missing if the Point Mode option is chosen.
Baseline	Estimate based on the baseline function, which typically estimates health impacts due to all causes (not just air pollution-related causes).
Percent of Baseline	Estimates the percentage change in health impacts (e.g., hospital admissions) due to the change in air quality from the baseline to the control scenario. Calculated by dividing the Point Estimate by the Baseline.
Standard Deviation	Standard deviation calculated based on the points in the Monte Carlo-generated percentiles for this result.
Variance	Variance calculated based on the points in the Monte Carlo-generated percentiles for this result.
Percentiles	The number of percentiles depends on the number of points in Monte Carlo-generated percentiles for this result.

Once all of the options have been selected for your report, you can export the **Health Impact Results** data. First select the **Data** tab then click the **Export** button. This will bring up a window allowing you to name the file you want to save. Note that by default BenMAP-CE will export the file to the CFGR folder. Carefully name the file that you are generating so that you will recognize it in the future!

Decision Point

Once you generate a CFGRX file, BenMAP-CE has quantified the endpoint-specific health benefits associated with your Control scenario. You have completed your BenMAP-CE analysis if:

3. You do not want to pool your health incidence results. For many of the health endpoints (e.g., mortality), BenMAP-CE contains several different health impact functions from different studies that you may choose to include in your configuration. Pooling refers to combining the results of two or more health impact functions into a single, integrated result.
4. You do not want to monetize the health benefits in the valuation step.

You may export your health impact results by selecting your desired aggregation level from the *Aggregation for raw data* drop-down list and clicking the **Export** button from the **Data** tab. If you also want to pool your health incidence results and/or monetize the health benefits, proceed to Ch. 7 "Aggregating, Pooling, and Valuing."

6.5 Frequently Asked Questions

How do I know which health impact functions to use? Which functions does EPA use?

One option regarding the choice of health impact functions is to work with someone, say another BenMAP-CE user, who is familiar with the epidemiological literature and develop your own set of health impact functions. Reviewing the epidemiological literature can be time consuming, though in some situations, this might be the best option. For example, it would be worthwhile to develop health impact functions to estimate the impacts of carbon monoxide exposure, for which BenMAP-CE does not have pre-installed functions.

Another option is to use the ozone and PM_{2.5} configurations used by EPA. These are available on the BenMAP-CE website (<http://www2.epa.gov/benmap/benmap-community-edition>). These functions are derived from the epidemiology literature described in the appendices to this user manual. If desired you can edit this configuration and then save it under a different file name—it is always a good idea to keep the original version, so you can go back to it if needed!

How do I edit or add other health impact functions?

To edit or add health impact functions you need to go to **Modify Datasets** window available from the BenMAP-CE main menu. See the health impact function section in Chapter 4: Loading Data for details on how to do this.

How do I learn more about the population data in BenMAP-CE?

Appendix J describes the population data for the *United States* setup in detail.

Why did I not get results for a given geographic area that I wanted in my analysis?

Check to see if your air quality grids mapped properly.

How do I determine what the Column and Row refer to in the result table?

The **Column** and **Row** are variables designed to uniquely identify each grid cell in the grid definition. In the case of the *U.S. County* grid definition, the **Column** refers to the *state FIPS code* and the **Row** refers to the *county FIPS code*. One way to get a good sense of the **Column** and **Row** variables is to create a map (discussed in the next chapter) and then view where particular **Column** and **Row** variables occur in the map.

Chapter 7

Aggregating, Pooling, and Valuing

In this chapter...

- **Get an overview of valuation, discounting, and pooling.**
- **Configure an Aggregation, Pooling, and Valuation (APV) file.**
- **Sort and pool incidence results.**
- **Learn the differences between the pooling methods.**
- **Assign economic values to incidence results.**
- **Aggregate incidence results and valuations.**
- **Run, save, and re-open an APV configuration.**
- **View and export APV results.**

This section presents an introduction to valuation, discounting, and pooling. Most BenMAP-CE users find this portion of the program the most complex to understand and use. You may find yourself referring to this chapter frequently.

Once you have created a configuration results file with incidence results based on your two air quality grids (refer to Chapter 6), you can use the Aggregate, Pool, and Value feature to combine the incidence results and place an economic value on the combined results. You have two options.

- **Create a New Configuration to Aggregate, Pool, and Value results.**

You can create a new type of configuration, termed an Aggregation, Pooling, and Valuation (APV) Configuration. This allows you to (1) specify the geographic level at which you want to report your results, (2) specify how you might want to combine or "pool" the incidence results, and (3) specify how to assign an economic value to the health incidence results. These selections can be saved in an APV Configuration file (.apvx) and used to calculate results, which are stored in an APV Results file (.apvrx).

Fundamental Concept – APVX and APVRX Files

An **Aggregation, Pooling, and Valuation (APV) Configuration** file (*.apvx) stores your user-specified BenMAP-CE preferences regarding how to aggregate your results, whether and how to pool your results, and any economic valuation functions you have applied. For example, an APV file might aggregate your estimated change in incidence to the U.S. county level, it might pool across multiple hospital admission health impact functions, and it could include an economic valuation function.

Results derived from an APV configuration will be stored along with the configuration details in an **Aggregation, Pooling, and Valuation (APV) Configuration Results** file (*.apvrx). The results in this file are the aggregated, pooled, and/or valued results for a given scenario.

- **Open Existing Configuration for Aggregation, Pooling, and Valuation.** You can load an existing APV Configuration file, edit the configuration, save it with the same or a different name, and then proceed to calculating the results.

7.1 Introduction to Valuation, Discounting, and Pooling

Valuation generally refers to placing a monetary value on estimated health incidence. In the example below, we discuss U.S. dollar values and provide a brief introduction to discounting, which has to do with placing less weight on things occurring in the future than on things occurring today. Finally, we discuss pooling, which has to do with combining comparable results.

7.1.1 Overview of Economic Valuation

Improvements in ambient air quality generally lower the risk of developing an adverse health effect by a fairly small amount across a large population. A lower risk for everyone means that fewer cases of the adverse health effect are expected, although we cannot predict which people would be spared. Therefore, the health benefits conferred on individuals by a reduction in pollution are actually reductions in the risk of having to

endure certain health problems. Monetizing the benefits of a reduction in air pollution involves estimating society's willingness to pay (WTP) for these reductions in risk, or the observed Cost of Illness (COI) for an effect, and is typically referred to as valuation. BenMAP-CE uses valuation functions to estimate the monetized 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.

Fundamental Concepts – Valuation

Valuation Functions are used by BenMAP-CE 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 endpoints, BenMAP-CE estimates monetized benefits by using either **Willingness to Pay (WTP)** or **Cost of Illness (COI)**-based valuation functions. **WTP** reflects the willingness of individuals to exchange money for a reduction in his or her risk of illness or death and is viewed by economists as the most complete and appropriate measure of the value of a risk reduction. **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 does not include the value of avoided pain and suffering.

For mortality endpoints, BenMAP-CE generally estimates monetized benefits by 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.

7.1.1.1 Monetizing 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 from that 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 \$100, then the value of a statistical life is $10,000 \times \$100$, or \$1 million. In general, the ex-ante WTP for a risk reduction of x can be converted into an ex-post value of a statistical case avoided by dividing the average individual's WTP for the risk reduction of x by x (e.g. $\$100/0.0001 = \$1,000,000$). The same type of calculation can produce values for statistical incidences of other health endpoints.

Sometimes those economic values come from contingent valuation studies, in which study participants are queried about their WTP to avoid a specific adverse health effect.

When estimates of WTP are not available, it can be approximated by other measures, most notably COI measures.

An individual's WTP to avoid an adverse health effect will include, at a minimum, 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 total value to society of an individual's avoidance of an adverse health effect, then, might be thought of as having two components: (1) the COI to society, including the total value of the medical resources used (some portion of which will be paid by the individual), 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.

The COI approach attempts to estimate the total value of the medical resources used up as well as the value of the individual's time lost as a result of the illness. 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 (and conjoint analysis) attempts to elicit from people what they would be willing to pay (WTP) 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 the 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 the benefit analyst chooses to value a particular health endpoint will depend in part on what data are available. The unit values currently available for use in BenMAP-CE 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.

7.1.1.2 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-CE currently offers a variety of options reflecting the uncertainty surrounding

the unit value for premature mortality. See the Appendices H and I for more detail on the valuation functions available in BenMAP-CE.

Monetary estimates of changes in premature mortality risk are often expressed in terms of the Value of a Statistical Life (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 marginal changes in the risk of death and should not be used to value more significant 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.

7.1.2 Overview of Discounting

What is discounting?

In general, people prefer current consumption to future consumption. In other words, a \$1 today is worth more today than a \$1 tomorrow is worth today, 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 that 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** of benefits.

A basic discounting function is as follows:

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

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

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

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

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 in order to compare those future benefits with current benefits.
2. For a cost-benefit analysis, benefits 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 core BenMAP-CE 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.²⁴ Discounting can be used to compare the future benefits with benefits occurring during the analysis year.

Under which scenarios would I need to discount benefits?

Health benefits may occur three different ways after the analysis year specified in BenMAP-CE.

1. Certain health endpoints accrue medical expenses or lost earnings for multiple years. The future medical expenses would need to be discounted to compare with expenses occurring in the analysis year.
2. Pollution exposure and the resulting health effects do not occur within the same year (a.k.a. a cession lag). The monetized benefits of future health effects would need to be discounted to compare with the benefits of health effects that occur during the analysis year.
3. In some analyses, you may want to estimate a stream of benefits occurring over multiple analysis years instead of just one analysis year. In this scenario, you would need to discount the future benefits occurring in each year analyzed back to the present year in order to present the cumulative total estimate of benefits (i.e., the net present value of a stream of benefits).

²⁴ The PopSim tool estimates the change in population mortality risk over a multi-year period, but it is not yet possible to estimate the economic value of these impacts in BenMAP-CE.

When would we not discount benefits?

In many instances, it is not necessary to discount the benefits estimates generated by BenMAP-CE. If the health effect and the monetized value of all the medical expenses, lost earnings, and suffering occur entirely in the analysis year, then you may not need to discount your benefits. For example, school loss days occur within the analysis year, and all monetized expenses occur within the analysis year. It is important that you understand the assumptions within the health and valuation functions before you decide whether you need to discount. (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.)

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 and mercury.)

There are various economic arguments in support of and in opposition to various discount rates. To comply with OMB and EPA's recommendations, EPA currently uses discount rates of 3% and 7% for benefit analyses. For more details, see EPA [1999; 2000] listed in Chapter 1, Section 1.7 (Sources for More Information).

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

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

- Chronic bronchitis is assumed to last from the initial onset of the illness throughout the rest of the individual's life. BenMAP-CE currently includes one WTP function as well as two COI functions representing the two discount rates for chronic bronchitis.

- 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-CE. AMIs are assumed to accrue costs over five years. Although WTP functions for AMIs are not available, BenMAP-CE 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.

See Appendix G for details on the discounting assumptions within the valuation functions.

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

You should not discount the health incidence for any of the scenarios mentioned above. Changes in the lag assumptions do not change the total number of estimated deaths, for example, but rather the timing of those deaths. If you discounted the health incidence along with valuation, you would essentially be discounting twice.

Which health endpoints do not occur in the same year as exposure?

In many cases, the health effect from exposure to air pollution occurs shortly after exposure, but there can be a significant lag between exposure and the health effect. The cessation lag can be a matter of hours or days, but some health effects may lag exposure by much longer. 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 function currently in BenMAP-CE that falls into this category is PM_{2.5}-related premature mortality. Discounting PM-related premature mortality is controversial because the lag structure is unknown, but scientific literature on similar adverse health effects and new intervention studies suggest that premature mortality 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-CE 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 provided a lag structure that could be assumed until additional research has been completed. See Chapter 5 of the PM Regulatory Impact Analysis for more detail on assumed lag structures for PM_{2.5}-related premature mortality (<http://www.epa.gov/ttn/ecas/regdata/RIAs/finalria.pdf>). Some example lag structures from the PM RIA are shown in Figures 7-1 and 7-2 below. Currently, BenMAP-CE does not have the capability to do this type of discounting, so you must discount outside of BenMAP-CE.

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.

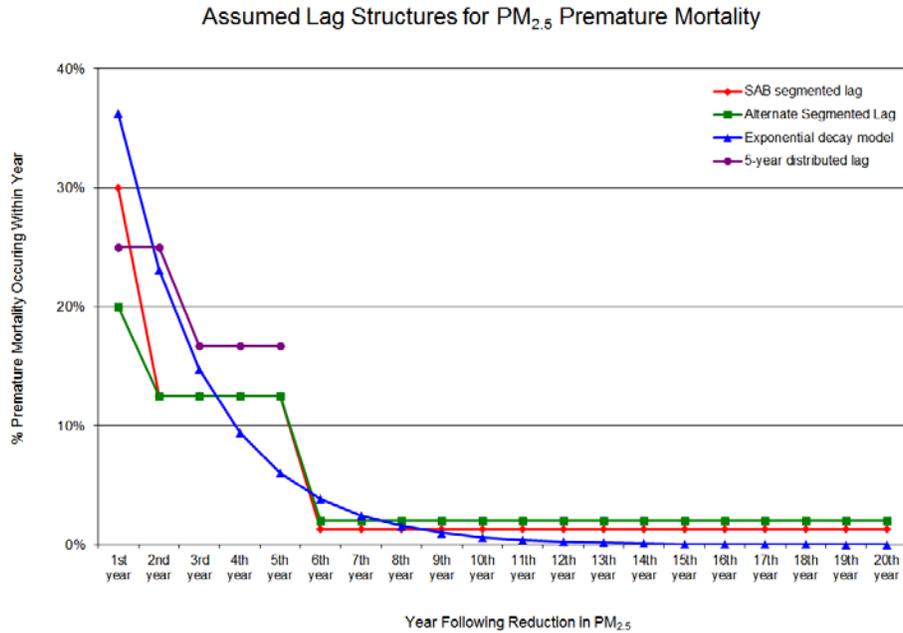


Figure 7-1. Graphical representation of assumed lag structures analyzed in EPA's PM RIA as sensitivity analyses

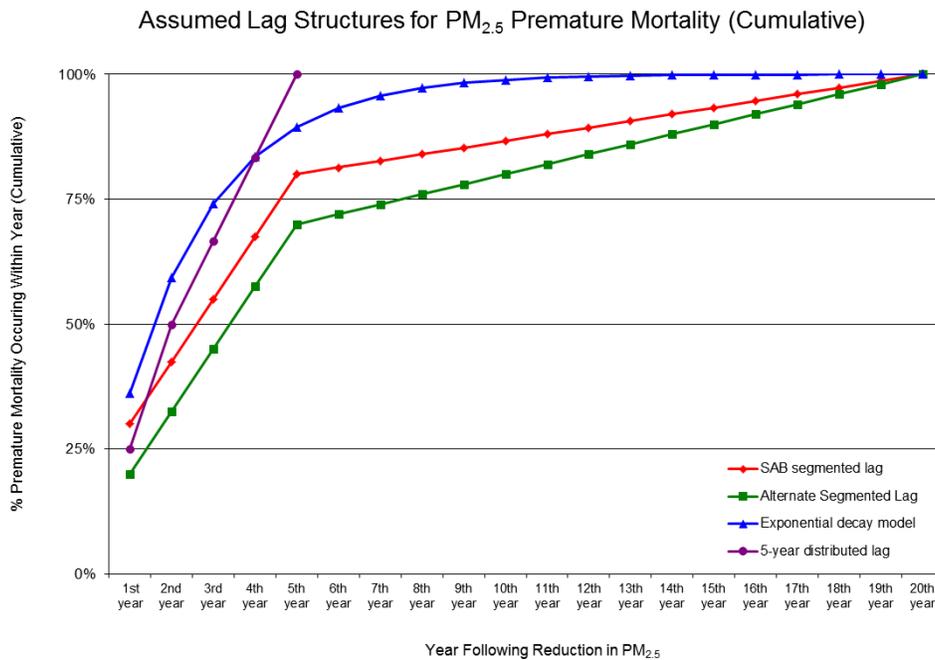


Figure 7-2. Graphical representation of cumulative assumed lag structures analyzed in EPA's PM RIA as sensitivity analyses

7.1.3 Overview of Pooling

For many of the health endpoints (e.g., respiratory hospital admissions), BenMAP-CE contains many different functions from different studies that you could choose to include in your configuration. Combining data from several comparable studies in order to analyze them together is often referred to as meta-analysis. For a number of reasons, it is often impractical or impossible to combine the original data sets. Combining the results of studies provides a second-best way to synthesize information. This is referred to as pooling.

BenMAP-CE allows users to pool the estimated incidence changes predicted by several studies for the same pollutant-health endpoint group combination (e.g., PM_{2.5}-related cardiovascular hospital admissions). It also allows the pooling of the corresponding study-specific estimates of monetary benefits.

Fundamental Concept - Pooling

Pooling refers to the methods of combining of different sets of results generated by multiple health impact functions or valuation functions. For example, BenMAP-CE allows you to pool the estimated incidence changes predicted by several health impact functions for the same pollutant-health endpoint group combination (e.g., PM_{2.5}-related cardiovascular hospital admissions) so that you can present a single value integrating the information generated by all those functions. BenMAP also allows you to pool the economic benefits estimates, e.g. to generate an average valuation from two different studies for ED visits for asthma.

BenMAP-CE includes several pooling methods, which are described in more detail below and in Appendix K.

Why would you want to pool results?

There are two good reasons to pool across study results, one practical and one methodological. Pooling allows you to

- Combine or aggregate multiple study estimates into a single estimate. This combined estimate is easier to report.
- Certain types of pooling—including random effects techniques—account for heterogeneity in the risk estimates reported in the epidemiological literature used to construct the health impact functions you used to calculate incidence.

However, as we discuss below, pooling may not be such a good option if

- You don't know a great deal about the studies used to quantify health impacts; you'll need to know a lot about epidemiological studies used to construct the health impact functions in order to pool properly.

Decision Point - Pooling

When might you want to pool your health impact estimates?

- You have multiple studies of the same health endpoint in the same population group and wish to report a single estimate reflecting the contributions of all the studies.
- You have multiple studies of different (non-overlapping) health endpoints in the same population group that you want to combine to simplify reporting of results (useful for HAs or ED visits).
- You have multiple studies of partially overlapping health endpoints and want to isolate the estimate of a specific health endpoint that appears in one HIF but not the other (via subtraction).

- You think it is important to convey the variability across incidence estimates for a given health endpoint.

BenMAP-CE allows you to pool in five different ways:

- Addition
- Subtraction
- User-assigned weights
- Random Effects
- Fixed Effect.

The examples that follow demonstrate each of these methods (Fixed and Random Effects are combined in one example.) In each of the examples below, the distribution of estimated health impacts are represented by a normally distributed probability density function (PDF) shaped as a bell curve. In each PDF, the mean health impact estimate is represented by a dashed line. While we illustrate these examples using a normally distributed PDF for ease of presentation, BenMAP-CE can accommodate several other types of distributions (e.g. Weibull, Triangular).

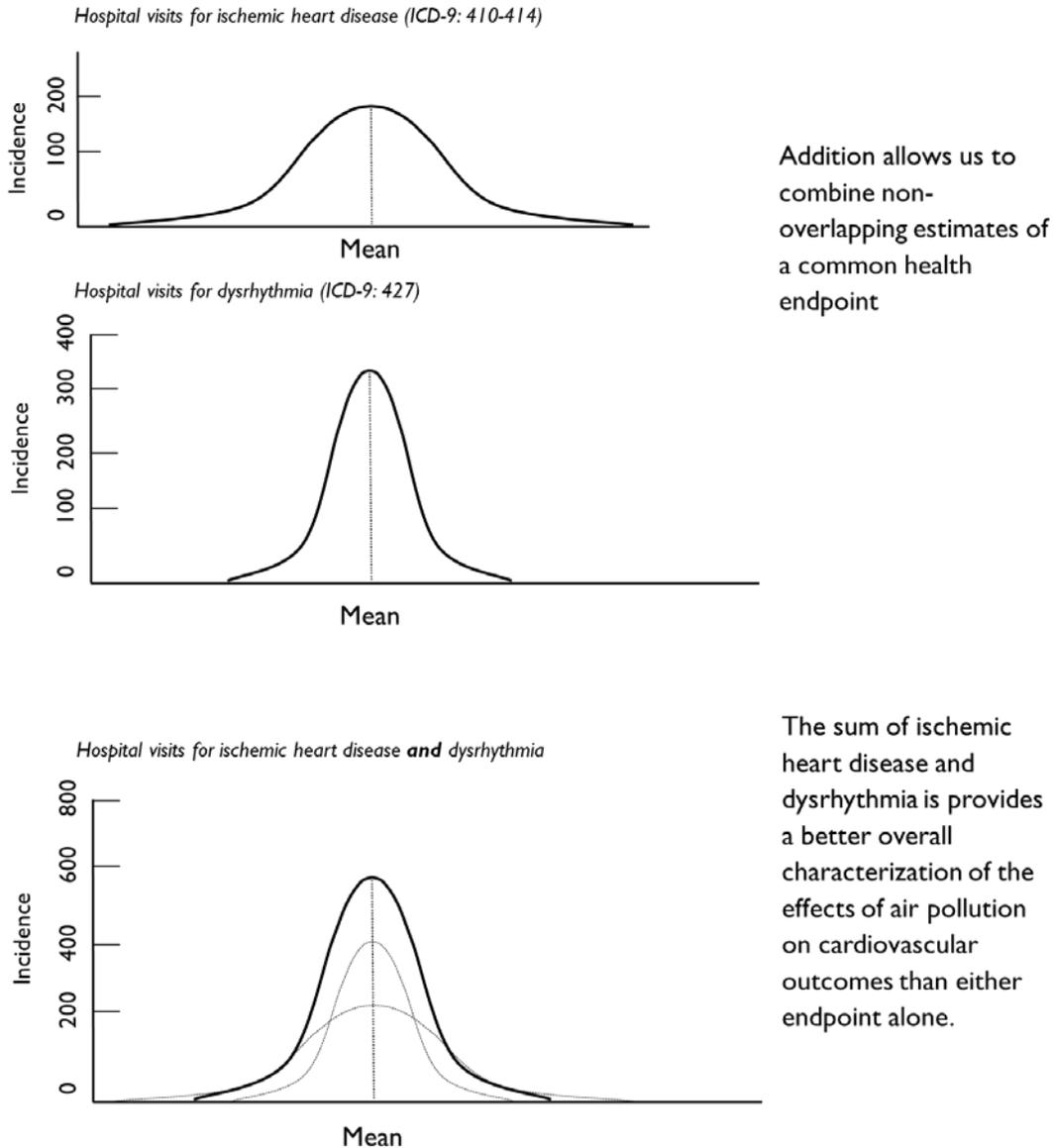
BenMAP-CE Use Example

Example scenarios for pooling health impact estimates:

- You ran two different HIFs for HA, All Cardiovascular for ages 65-99. They report different risk estimates and standard errors, and you'd like to combine them into a single estimate.
- You ran an HA, Ischemic Heart Disease HIF for ages 65-99 and an HA, Dysrhythmia HIF for ages 65-99 and want to combine the results into a single value for HA, cardiovascular outcomes.
- You ran an HA, All Cardiovascular HIF for ages 65-99 and an HA, All Cardiovascular, *less stroke* HIF for ages 65-99 and want to use the two functions to estimate the number of stroke-related HAS.

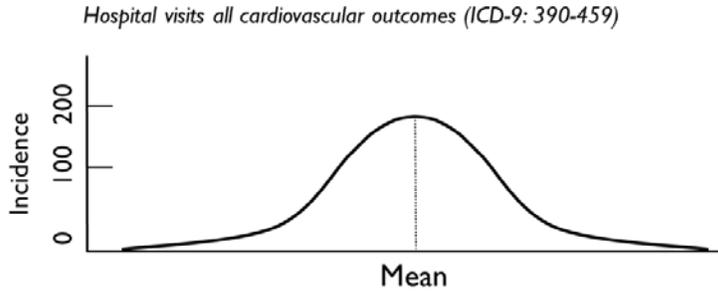
Example 1: Pooling by Addition

You might want to use the *Pooling by Addition* option if you would like to aggregate two outcomes that are non-overlapping. In the example below, we have estimated ischemic heart disease hospital admissions and dysrhythmia hospital admissions. You'll see that each endpoint is associated with unique, and non-overlapping, International Classification of Disease 9th edition (ICD-9) code (Slee 1978). Therefore, it's ok to add the two estimates, because doing so would not double-count impacts.

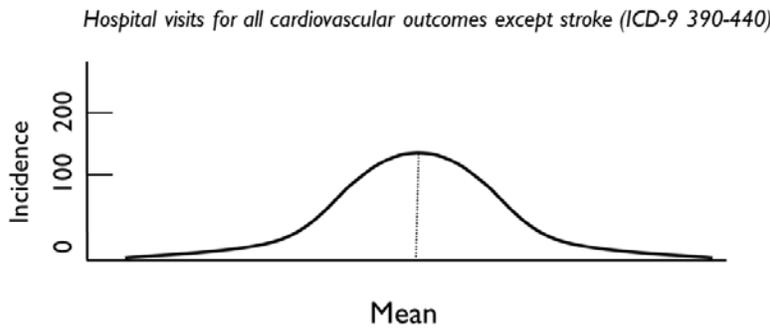


Example 2: Pooling by Subtraction

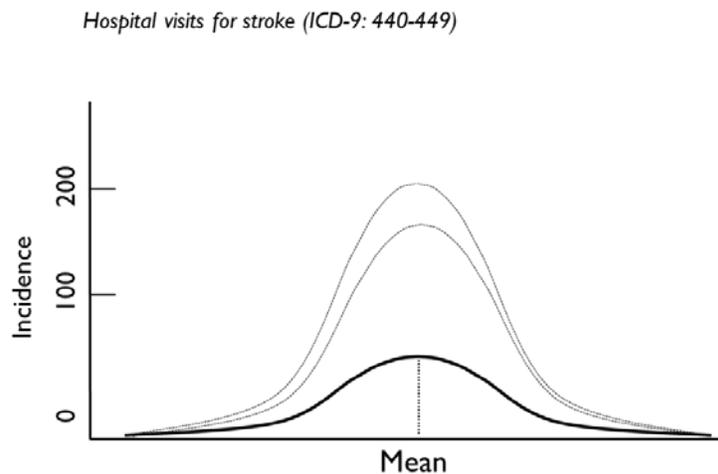
In this example, suppose that you have estimated the incidence of total cardiovascular hospital admissions using one health impact function. Suppose also that you estimated the incidence of total cardiovascular hospital admissions, *less stroke*. In this instance, you could subtract the second incidence estimate from the first incidence estimate to yield the number of stroke hospital admissions.



Subtraction allows us to “net out” the incidence of a health endpoint from two or more studies



In this example, the only difference between these two studies is that study one includes all cardiovascular outcomes, while study two excludes strokes

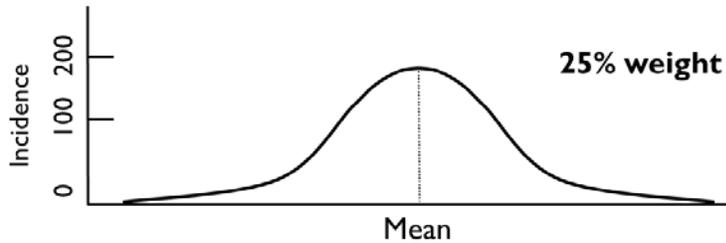


Subtracting the results of study two from study one yields an estimate of stroke

Example 3: Pooling with User-Assigned Weights

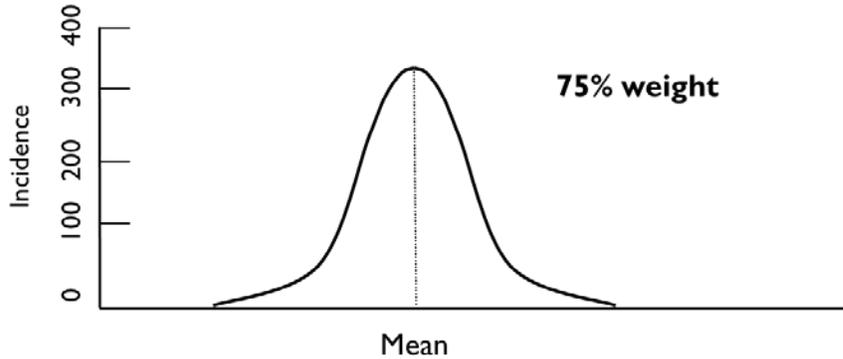
In this case, you might have estimated the change in incidence using two different health impact functions for the same health endpoint and would like to combine them using weights that you specify.

Peng et al. 2009 Multi-City Study of Cardio Hospital Admissions



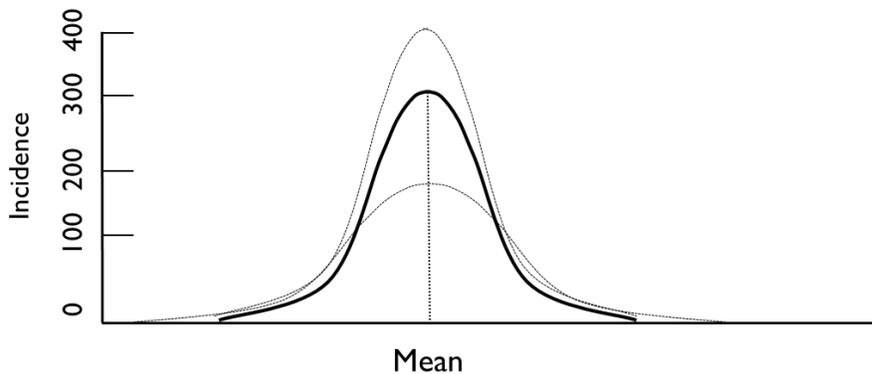
Some studies examine a common health endpoint and share a similar methodology, but may differ slightly in the populations examined

Bell et al. 2008 Multi-City Study of Cardio Hospital Admissions



Users may wish to combine these study estimates together using equal weights

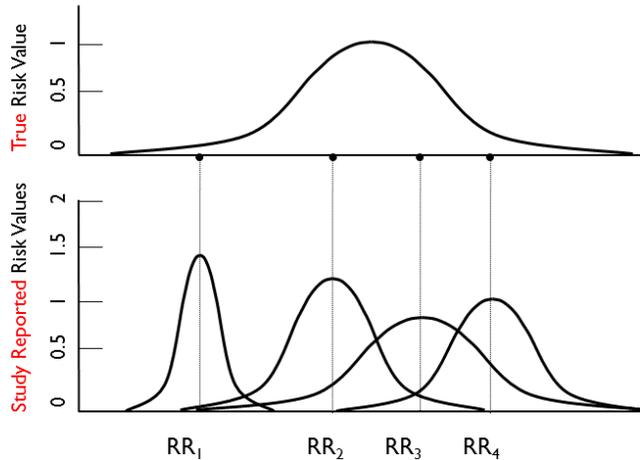
Pooled estimate of Peng & Bell



The pooled value reflects a weighted average of the two studies

Example 4: Pooling Using the Fixed Effect and Random Effects

The Fixed Effect and Random Effects pooling techniques are among the most complicated and are best applied only when you understand clearly the assumptions inherent in the method and its suitability to the incidence estimates. The example below describes the procedure for performing this technique.

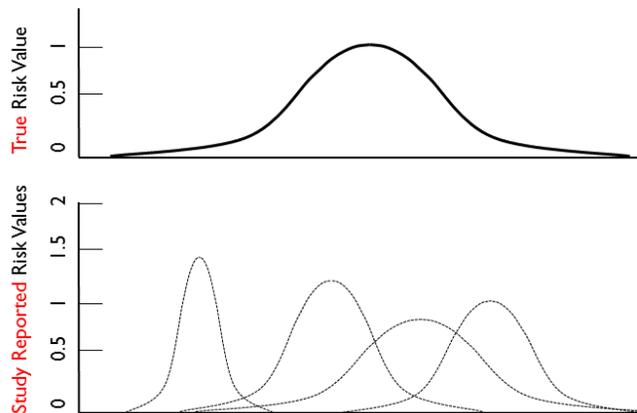


For the risks of a given health outcome there is a **true** but **unknown** distribution

The individual studies in the literature report individual risk estimates from that distribution

Random effects pooling accounts for heterogeneity in the individual risk estimates to generate a single mean risk estimate

Adapted from: Mosteller and Colditz (1996); Charles Poole EPID 731

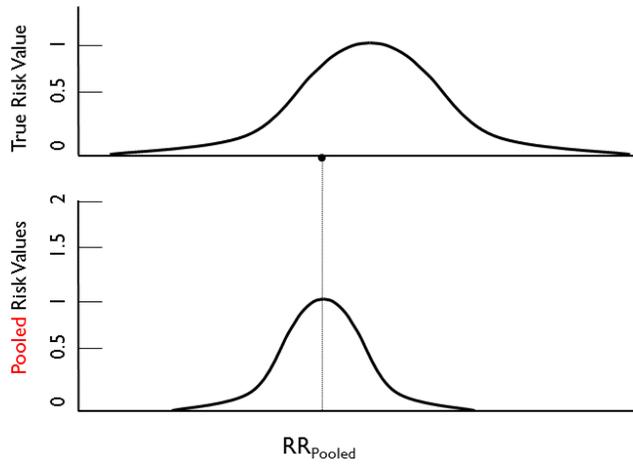


The Random-Effects model assigns each study a weight based on two factors:

1. The spread of estimates reported by each study (i.e. the variance)
2. How much that spread of estimates differs from spread reported by the other studies

Adapted from: Mosteller and Colditz (1996); Charles Poole EPID 731

Pooling: Random-Effects



The Random-Effects model assigns each study a weight based on two factors:

1. The spread of estimates reported by that study (i.e. the variance)
2. How much that spread of estimates differs from the other studies

Finally, the Random-Effects model calculates a weighted average of the studies

Table 7-1 summarizes the different types of pooling approaches, and Appendix I provides a detailed discussion of the approaches. Note that some pooling methods are only available in Monte Carlo mode. This is because these pooling methods attempt to combine distributions of results into new distributions, and no distributional information is available in Point Mode. The Pooling Method column will thus have different values in its drop-down list depending on the mode used to generate the incidence results being pooled.

Table 7-1. Pooling Approaches for Incidence and Valuation Results

Pooling Approach	Description of Pooling Approach	Availability	
		Point Mode	Monte Carlo
None	No pooling performed.	Yes	Yes
Sum (Dependent)	Results are summed assuming they are perfectly correlated. In Point Mode, this is just a simple sum. In Monte Carlo mode, BenMAP-CE chooses the first point from each result in the pooling and does a simple sum to generate the first point in the pooled result, and so on for all of the points in the distribution of results.	Yes	Yes
Sum (Independent)	Results are summed assuming that they are independent. A Monte Carlo simulation is used. At each iteration, a random point is chosen from the distribution for each result, and the sum of these values is put in a holding container. After some number of iterations, the holding container is sorted low to high and binned down to the appropriate number of percentile points.	No	Yes

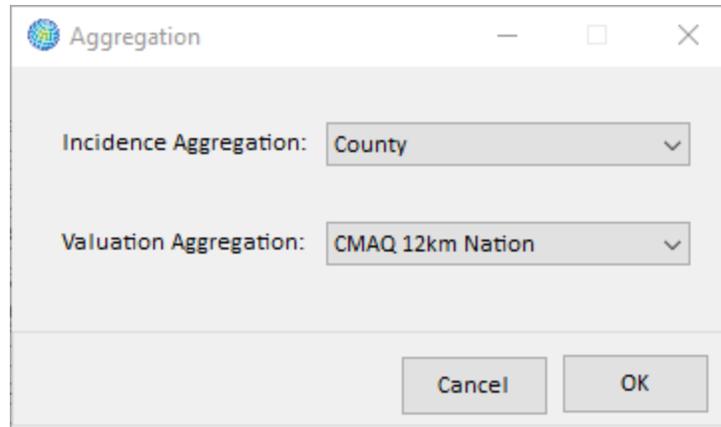
Pooling Approach	Description of Pooling Approach	Availability	
		Point Mode	Monte Carlo
Subtraction (Dependent)	Results are subtracted assuming they are perfectly correlated. All subsequent results are subtracted from the first result (the highest result in the display - to reorder results, simply click and hold a result and then drag it to its new position). In Point Mode, this is a simple subtraction. In Monte Carlo mode, BenMAP-CE chooses the first point from each result in the pooling and does a simple subtraction to generate the first point in the pooled result, and so on for all of the points in the distribution of results.	Yes	Yes
Subtraction (Independent)	Results are subtracted assuming that they are independent. A Monte Carlo simulation is used. At each iteration, a random point is chosen from the distribution for the first result, and then random points are chosen from the distribution for each subsequent result and subtracted from the first. The result is put into a holding container. After some number of iterations, the holding container is sorted low to high and binned down to the appropriate number of percentile points.	No	Yes
User defined Weights	Weights are specified by the user. In Point Mode, the new result is generated by a simple weighted sum of the input results. In Monte Carlo mode, the results are combined using the user specified weights with the <i>'Round Weights to Two Digits'</i> Advanced Pooling Method. Note that the weights you enter need not add up to one - BenMAP-CE will normalize them internally. Also note that BenMAP-CE initializes all the weights to $1/n$, where n is the number of results being pooled.	Yes	Yes

7.2 Create Aggregation, Pooling, and Valuation (APV) Configuration

Once you have run a configuration result file (see Chapter 6), you can begin creating your APV configuration. You will start with selecting the aggregation levels for incidence and valuation results, then move on to pooling and valuation. These processes are described in detail below.

7.2.1 Selecting Aggregation Levels

Double-clicking **Aggregation** from the BenMAP-CE tree menu item opens the window which lets you choose the level of aggregation for the incidence and valuation results.



7.2.2 Pooling Incidence Results

To begin pooling incidence results, double-click **Pooling Method** from the BenMAP-CE tree menu. In the top half of the **Select and Pool Incidence Estimates** window, you will find a list of **Available Incidence Results**. The results are represented by the health impact functions from which they were created. You may click on the **Select study fields** button to display additional information about each health impact function. This is recommended when using the geographic areas functionality (i.e., applying region-specific air pollution effect coefficients).

1. Select Available Incidence Results

Filter Dataset: [Dropdown] Filter Endpoint Group: [Dropdown] Filter: [Text] Groups Select study fields Check HIF Changes

Add to Pooling Window Add All

Endpoint	Endpoint Group	Dataset Name	Start Age	End Age	Author
<input type="checkbox"/> Acute Bronchitis	Acute Bronchitis	Additional Health Functions	8	12	Dockery et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Wheeze	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Norris et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Mar et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Slaughter et al.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Emergency Room Visits, Respiratory Pooling Levels: 0 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Acute Bronchitis: | Asthma Exacerbation: | **Emergency Room Visits, Respiratory** x

Studies, By Endpoint	Pooling Method	Nickname	Endpoint	Author	Qualifier	Study L...	Start A...	End Age	Age Ra...	Version	Year
Emergency Room Visits, R...	None	Emergency Room Visits, Respiratory	Emergency Room Visits, Asthma	Mar et al.; Slaug...	Entire st...	Greater ...	0	99	0-99		
Mar			Emergency Room Visits, Asthma	Mar et al.			0	99	0-99	1	2010
Slaughter			Emergency Room Visits, Asthma	Slaughter et al.			0	99	0-99	1	2005
Glad			Emergency Room Visits, Asthma	Glad et al.			0	99	0-99	1	2012

Preview Advanced Cancel Next

There are several steps to pooling your incidence results:

Step 1. Add incidence results to the pooling window

Using the available incidence results, you can drag individual incidence results to the pooling window, or select the result(s) using the checkboxes next to each study, and click **Add to Pooling Window** to move them to the pooling window. You do not have to add all of your incidence results to the pooling window, but note that only those results showing in the pooling window will be included in the pooled incidence or valuation results. As with health impact functions, in Chapter 6, there is a filter method below the **Available Incidences Results** for your convenience.

The bottom half of the pooling window consists of one or more tabs, each with an associated “pooling window”, where health impact functions can be added and specifications for how to pool them (if any) can be made. In version 1.5, by default, BenMAP-CE will automatically create a pooling window for each Endpoint Group for which one or more Health Impact Functions were selected in the Health Impact Function selection screen, and the Endpoint Group will be indicated on the tab. For example, there could be one pooling window for Mortality and one for Asthma Exacerbation. Health impact functions should be added to the pooling window corresponding to that function’s Endpoint Group. Additional pooling windows can be added if needed, as discussed below.

Incidence results are displayed in the pooling window in a tree structure determined by (1) the order of the columns, and (2) the values of the identifying variables of the **Health Impact Functions** from which the incidence results were generated (**Endpoint Group, Endpoint, etc.**).

BenMAP-CE Best Practices

Incidence results *must* be added to the pooling window in order to be included in the valuation step, whether or not you intend to pool them with other incidence results. If you would like to apply a valuation function to a single (unpooled) health incidence estimate, add the health incidence estimate to the pooling window and specify the **Pooling Method** as “None.”

BenMAP-CE Guidance and Best Practices

BenMAP-CE automatically generates a pooling window (i.e., tab) for each unique **Endpoint Group** present in your health incidence results.

Generally, you should add health impact functions to the pooling window corresponding to that function’s **Endpoint Group**. For example, you would likely want to add health incidence results for *Mortality, Respiratory* to the “Mortality” pooling window.

You may need multiple pooling windows for the same **Endpoint Group** if you want to pool and/or value the same incidence results in different ways. For example, you might have age-stratified incidence results and want to calculate age-specific pooled results as well as a pooled result across all ages. In this case, you would need to set up two pooling windows for the same **Endpoint Group**. You may use as many pooling windows as you need in your analysis; you should give each pooling window a unique name. See Step 4 for more information on adding pooling windows.

Select and Pool Incidence Estimates

1. Select Available Incidence Results

Filter Dataset: [] Filter Endpoint Group: [] Filter: [] Groups Select study fields Check HIF Changes

Add to Pooling Window Add All

Endpoint	Endpoint Group	Dataset Name	Start Age	End Age	Author
<input type="checkbox"/> Acute Bronchitis	Acute Bronchitis	Additional Health Functions	8	12	Dockery et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Wheeze	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	17	Norris et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Mar et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Slaughter et al.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Emergency Room Visits, Respiratory Pooling Levels: 0 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Acute Bronchitis | Asthma Exacerbation | **Emergency Room Visits, Respiratory** x

Studies, By Endpoint	Pooling Method	Nickname	Endpoint	Author	Qualifier	Study L...	Start A...	End Age	Age Ra...	Version	Year
Emergency Room Visits, R...	None	Emergency Room Visits, Respiratory	Emergency Room Visits, Asthma	Mar et al.; Slaug...			0	99	0-99		
Mar			Emergency Room Visits, Asthma	Mar et al.	Entire st...	Greater...	0	99	0-99	1	2010
Slaughter			Emergency Room Visits, Asthma	Slaughter et al.		Spokan...	0	99	0-99	1	2005
Glad			Emergency Room Visits, Asthma	Glad et al.		Pittsbu...	0	99	0-99	1	2012

Preview Advanced Cancel Next

Each line in the pooling window represents a node in the tree structure, with each node representing either an individual incidence result or a collection of incidence results which have common values for their leftmost identifying variables. The tree structure is generated by comparing the leftmost values of the incidence result's identifying variables. High level nodes in the tree are formed when results have common values for identifying variables, and branches in the tree occur when the values differ.

Select and Pool Incidence Estimates

1. Select Available Incidence Results

Filter Dataset: Filter Endpoint Group: Filter: Groups

Endpoint	Endpoint Group	Dataset Name	Start Age	End Age	Author
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Wheeze	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.

2. Select Pooling Methods

Pooling Window Name: Pooling Levels: Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Acute Bronchitis: **Asthma Exacerbation** x Emergency Room Visits, Respiratory x

Studies, By Endpoint	Pooling Method	Nickname	Endpoint	Author	Qualifier	Study L...	Start A...	End Age	Age Ra...	Vers
Asthma Exacerbation	None	Asthma Exacerbation	Asthma Exacerbation, Cough Asthma Exace...	Mar et al.; Ostro ...			6	18	6-18	
Asthma Exacerbation, Cough	None	Asthma Exacerbation, Cough	Asthma Exacerbation, Cough	Mar et al.; Ostro ...			6	18	6-18	
Mar			Asthma Exacerbation, Cough	Mar et al.	Uses in...	Spokan...	6	18	6-18	1
Ostro			Asthma Exacerbation, Cough	Ostro et al.	Ostro st...	Los Ang...	6	18	6-18	1
Asthma Exacerbation, Shortness of Breath		Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation, Shortness of Breath	Ostro et al.			6	18	6-18	
Ostro			Asthma Exacerbation, Shortness of Breath	Ostro et al.	Ostro st...	Los Ang...	6	18	6-18	1
Asthma Exacerbation, Wheeze		Asthma Exacerbation, Wheeze	Asthma Exacerbation, Wheeze	Ostro et al.			6	18	6-18	
Ostro			Asthma Exacerbation, Wheeze	Ostro et al.	Ostro st...	Los Ang...	6	18	6-18	1

In the above example, four incidence results have been dragged into the pooling window. Each of the four health impact functions has **Endpoint Group** 'Asthma Exacerbation'. Thus, the top line, or root of the tree structure, represents all four incidence results. A branch then occurs in the tree structure, because two studies have **Endpoint** 'Asthma Exacerbation, Cough', while two others have **Endpoint** 'Asthma Exacerbation, Wheeze' and 'Endpoint Asthma Exacerbation, Shortness of Breath'. A further branch occurs within **Endpoint** 'Asthma Exacerbation, Cough' when **Author** of the two incidence results differs. Once a node has only a single incidence result, no further branching can occur.

Step 2. Sort results

After dragging incidence results into the pooling window, you can rearrange the order of the columns (variables), and thus change the tree structure. To do this, click on a column and hold the button down as you drag it to its new location. Note that the **Pooling Method** is always the first column after the **Studies, By Endpoint** column. All the other columns can be moved. To see how the order of the columns in the pooling window affects the tree structure, consider the following example:

1. Select Available Incidence Results

Endpoint	Endpoint Group	Dataset Name	Start Age	End Age	Author
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Wheeze	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Asthma Exacerbation Pooling Levels: 1 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Acute Bronchitis: Asthma Exacerbation x Emergency Room Visits, Respiratory x

Studies, By Endpoint	Pooling Method	Nickname	Author	Endpoint	Qualifier	Study L...	Start A...	End Age	Age Ra...	Vers
Asthma Exacerbation	None	Asthma Exacerbation	Mar et al.; Ostro ...	Asthma Exacerbation, Cough Asthma Exace...			6	18	6-18	
Mar et al.		Mar et al.	Mar et al.	Asthma Exacerbation, Cough			6	18	6-18	
Mar			Mar et al.	Asthma Exacerbation, Cough	Uses in...	Spokan...	6	18	6-18	1
Ostro et al.			Ostro et al.	Asthma Exacerbation, Cough Asthma Exace...			6	18	6-18	
Ostro			Ostro et al.	Asthma Exacerbation, Cough	Ostro st...	Los Ang...	6	18	6-18	1
Ostro			Ostro et al.	Asthma Exacerbation, Shortness of Breath	Ostro st...	Los Ang...	6	18	6-18	1
Ostro			Ostro et al.	Asthma Exacerbation, Wheeze	Ostro st...	Los Ang...	6	18	6-18	1

This example uses exactly the same incidence results as the previous example, but with the **Author** column (variable) immediately after the **Pooling Method** and **Nickname** columns. Note that the **Pooling Levels** drop-down is set to 1 because one criterion (**Author**) is used to group studies for pooling. Multiple criteria (e.g., **Endpoint** and **Author**) may be used to group studies for pooling if desired.

BenMAP-CE Guidance and Best Practices

Once you have added health impact results to a pooling window, your pooling tree structure is determined by:

- (1) the number of **Pooling Levels** selected (up to 3), which determines the number of criteria used to group studies for pooling,
- (2) the order of the **Pooling Level** columns in the pooling window, and
- (3) the values of the relevant **Pooling Level** meta-data of the Health Impact Functions from which the incidence results were generated (**Endpoint Group**, **Endpoint**, etc.).

You may alter your pooling tree structure by modifying (1) using the drop-down menu and (2) by dragging and dropping the **Pooling Level** into the desired grouping hierarchy.

Step 3. Select pooling methods

Once the tree structure is set up in the pooling window, you are ready to select your pooling methods. Essentially each **Pooling Method** involves a different method of combining input incidence results to generate new incidence results. Results can be pooled any time a branch occurs in the tree structure — that is, any time two or more results share common values for their leftmost variables. BenMAP-CE helps you to identify these spots by inserting a value of *None* in the **Pooling Method** column at each spot where pooling is possible. These spots may change depending on the ordering of the columns and the selected number of **Pooling Levels**.

When a row that represents a group of incidence results is selected, an arrow appears at the right of the **Pooling Method** column, indicating the availability of a drop-down menu where you can select the desired **Pooling Method**. If you select **User Defined Weights** from the **Pooling Method** menu, a **Weight** column will appear to the right of the **Pooling Method** menu. All studies are assigned equal weight by default unless you update them. The sum of all **User Defined Weights** must equal 1.

If a **Pooling Method** is selected, you can view the calculated weights before executing the pooling by pressing the **Preview** button. In the case of the **Random or Fixed Effects** pooling method, the **Preview** button will reveal whether random or fixed effects are being applied.

Step 4. Create additional pooling windows if needed

Within a given pooling window, you can have only one ordering of the columns (variables). As we have seen, however, the ordering of the columns determines the structure of the tree used to pool results. It may thus sometimes be necessary for analyses to have multiple tree structures to handle the various pooling trees they require. To facilitate this, BenMAP allows additional pooling windows to be added and deleted. The pooling windows are displayed in a tabbed format.

To open a new pooling window, simply click on the rightmost tab marked with **+**. You may do this as many times as needed to accommodate different sort orders. You can add the same incidence results to as many different pooling windows as you like.

As needed, you can also delete a pooling window by clicking on the window you wish to delete and clicking the **x** to the right of the tab name.

Select and Pool Incidence Estimates

1. Select Available Incidence Results

Filter Dataset: [Dropdown] Filter Endpoint Group: [Dropdown] Filter: [Text] Groups Select study fields Check HIF Changes

Add to Pooling Window Add All

Endpoint	Endpoint Group	Dataset Name	Start Age	End Age	Author
<input type="checkbox"/> Acute Bronchitis	Acute Bronchitis	Additional Health Functions	8	12	Dockery et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Cough	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Mar et al.
<input type="checkbox"/> Asthma Exacerbation, Wheeze	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	Additional Health Functions	6	18	Ostro et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	17	Norris et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Mar et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Slaughter et al.
<input type="checkbox"/> Emergency Room Visits, Asthma	Emergency Room Visits, Respiratory	Additional Health Functions	0	99	Glad et al.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Acute Bronchitis Pooling Levels: 0 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

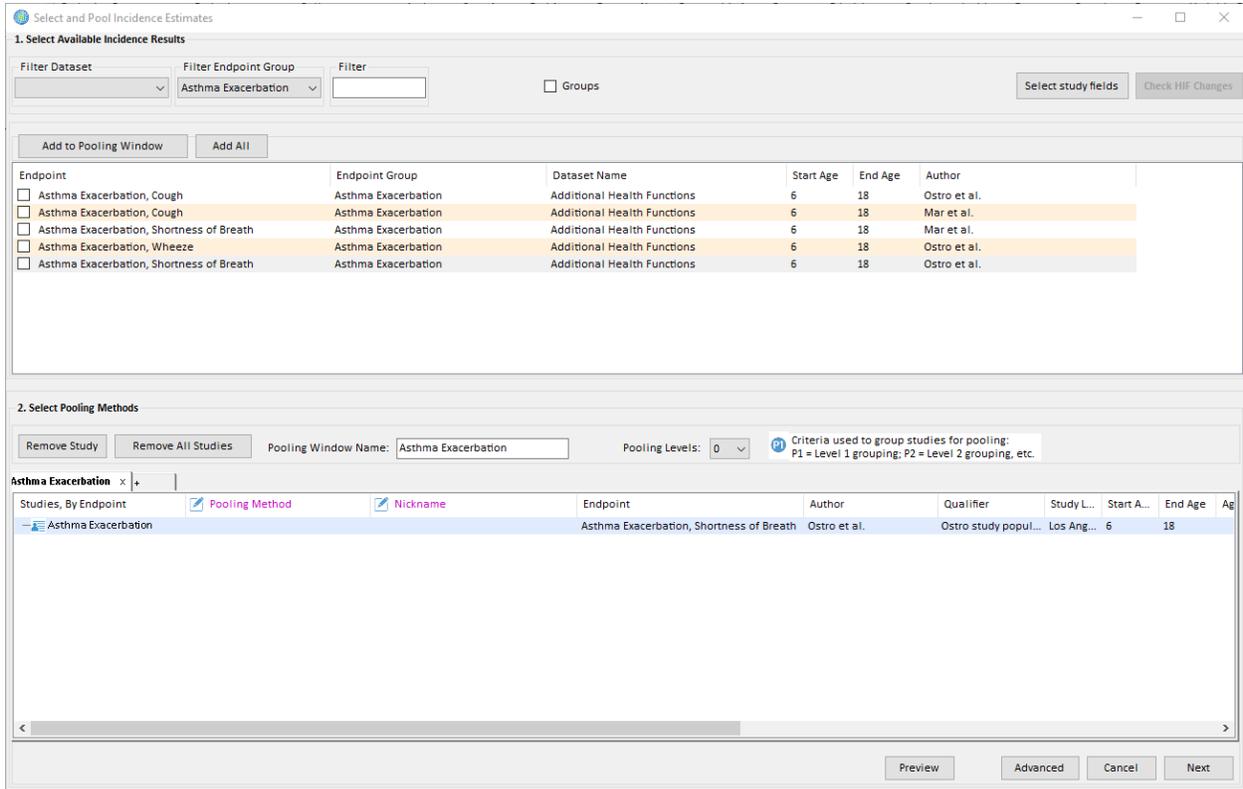
Acute Bronchitis x Asthma Exacerbation x Emergency Room Visits, Respiratory x +

Studies, By Endpoint	Endpoint	Author	Qualifier	Study L...	Start A...	End Age	Age Ra...	Ver
<input checked="" type="checkbox"/> Acute Bronchitis	Acute Bronchitis	Dockery et al.		24 com...	8	12		1

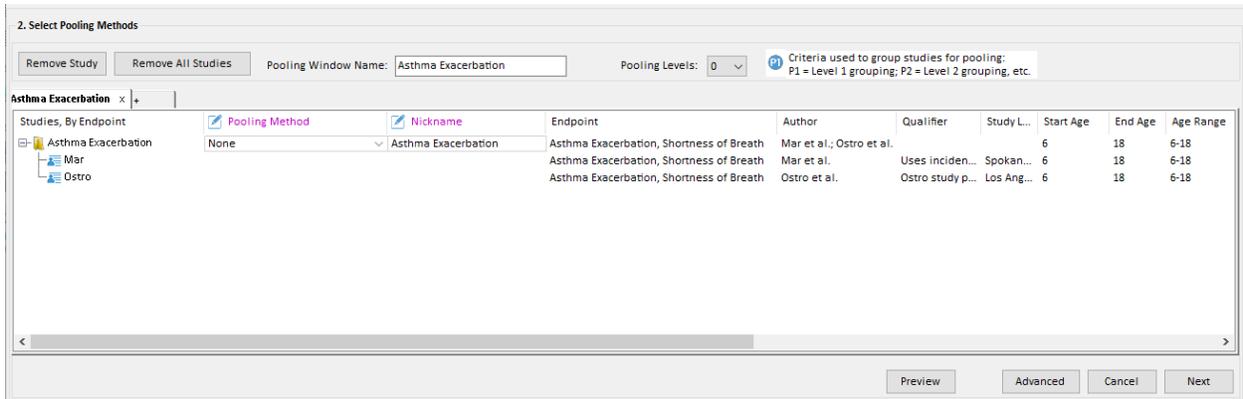
Preview Advanced Cancel Next

Example: Simple Sorting & Pooling

If you add a single incidence result to the pooling window, you will see just one line, and therefore no opportunities to pool. This is shown in the example below:



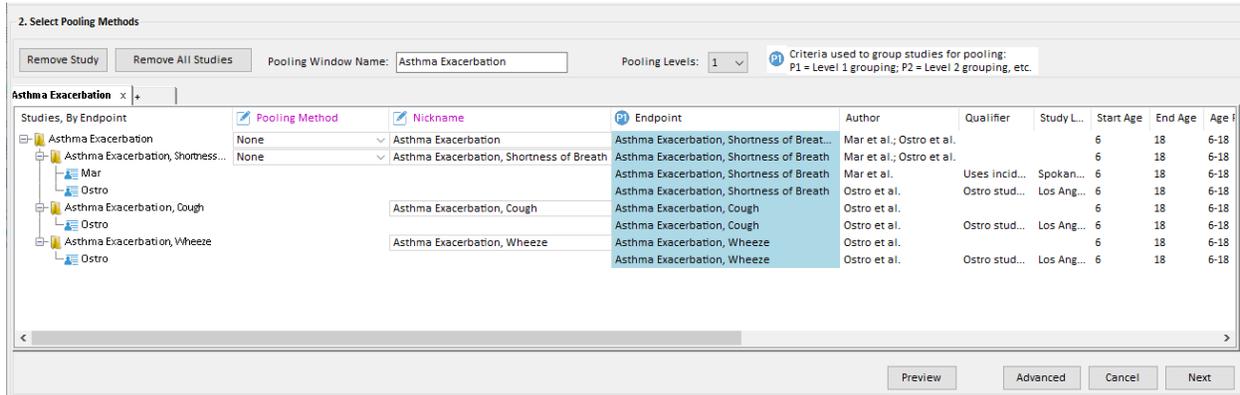
If you add a second incidence result to the window whose health impact function has the same **Endpoint Group**, but a different **Author**, you will then have a tree with two items in it. The tree branches at the point where the two health impact functions vary - at the **Author** column.



Note that a pooling method can now be selected for the two incidence results, since a branch has appeared. If we desired to pool these two incidence results, we would end

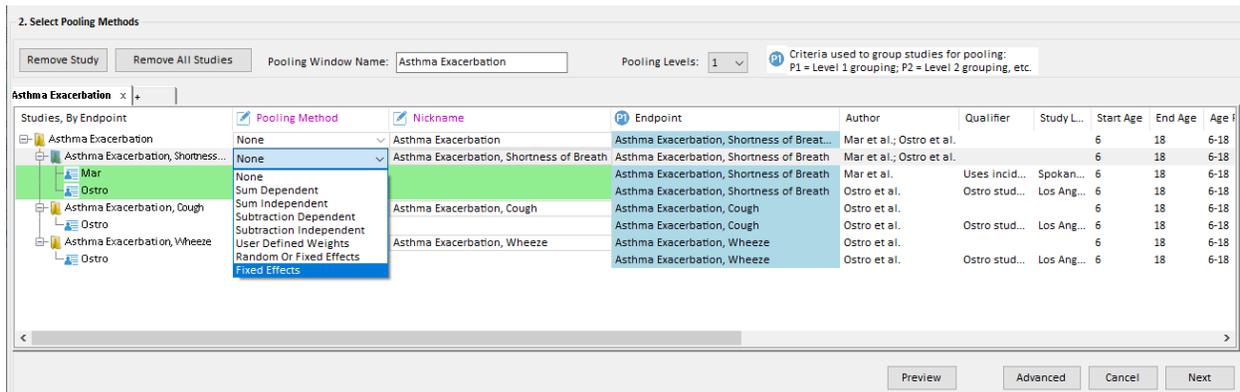
up with a pooled result representing two ‘*Asthma Exacerbation, Shortness of Breath*’ incidence results.

If you now add two more incidence results to the window whose health impact functions have the same **Endpoint Group** but different **Endpoints**, you will see the following:



Now you have many pooling options. Setting aside the issue of which pooling method to choose, there are four different pooling options at this point (including doing nothing), since we have two places where we can choose to pool or not to pool.

If you choose to pool at the Studies, By Endpoint corresponding to **Endpoint** ‘*Asthma Exacerbation, Shortness of Breath*’ you would end up with three results (one pooled and two unpooled) instead of four individual incidence results.



If you choose to pool at the Studies, By Endpoint for the **Endpoint Group** ‘*Asthma Exacerbation*’ (where the **Pooling Method** field says ‘*None*’ in the above image), you will end up with a single result representing all four of the original incidence results.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Asthma Exacerbation Pooling Levels: 1 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Asthma Exacerbation x

Studies, By Endpoint	Pooling Method	We...	Nickname	Endpoint	Author	Qualifier	Study L...	Start Age	End
Asthma Exacerbation	User Defined Weights		Asthma Exacerbation	Asthma Exacerbation, Shortness of Breat...	Mar et al.; Ostro et al.			6	18
Asthma Exacerbation, Shortness...	None		Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation, Shortness of Breath	Mar et al.; Ostro et al.			6	18
Mar		0.25		Asthma Exacerbation, Shortness of Breath	Mar et al.	Uses incid...	Spokan...	6	18
Ostro		0.25		Asthma Exacerbation, Shortness of Breath	Ostro et al.	Ostro stud...	Los Ang...	6	18
Asthma Exacerbation, Cough			Asthma Exacerbation, Cough	Asthma Exacerbation, Cough	Ostro et al.			6	18
Ostro		0.25		Asthma Exacerbation, Cough	Ostro et al.	Ostro stud...	Los Ang...	6	18
Asthma Exacerbation, Wheeze			Asthma Exacerbation, Wheeze	Asthma Exacerbation, Wheeze	Ostro et al.			6	18
Ostro		0.25		Asthma Exacerbation, Wheeze	Ostro et al.	Ostro stud...	Los Ang...	6	18

Preview Advanced Cancel Next

If you pool at both spots:

- First, the 'Asthma Exacerbation, Shortness of Breath' results are pooled to give a single 'Asthma Exacerbation, Shortness of Breath' result.
- Next, the three separate **Endpoint** results are pooled to give a single 'Asthma Exacerbation' result.

2. Select Pooling Methods

Remove Study Remove All Studies Pooling Window Name: Asthma Exacerbation Pooling Levels: 1 Criteria used to group studies for pooling: P1 = Level 1 grouping; P2 = Level 2 grouping, etc.

Asthma Exacerbation x

Studies, By Endpoint	Pooling Method	We...	Nickname	Endpoint	Author	Qualifier	Study L...	Start Age	End
Asthma Exacerbation	User Defined Weights		Asthma Exacerbation	Asthma Exacerbation, Shortness of Breat...	Mar et al.; Ostro et al.			6	18
Asthma Exacerbation, Shortness...	Fixed Effects	0.33	Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation, Shortness of Breath	Mar et al.; Ostro et al.			6	18
Mar				Asthma Exacerbation, Shortness of Breath	Mar et al.	Uses incid...	Spokan...	6	18
Ostro				Asthma Exacerbation, Shortness of Breath	Ostro et al.	Ostro stud...	Los Ang...	6	18
Asthma Exacerbation, Cough			Asthma Exacerbation, Cough	Asthma Exacerbation, Cough	Ostro et al.			6	18
Ostro		0.33		Asthma Exacerbation, Cough	Ostro et al.	Ostro stud...	Los Ang...	6	18
Asthma Exacerbation, Wheeze			Asthma Exacerbation, Wheeze	Asthma Exacerbation, Wheeze	Ostro et al.			6	18
Ostro		0.33		Asthma Exacerbation, Wheeze	Ostro et al.	Ostro stud...	Los Ang...	6	18

Preview Advanced Cancel Next

These same principles apply no matter how many incidence results are being pooled, and regardless of which pooling methods are selected.

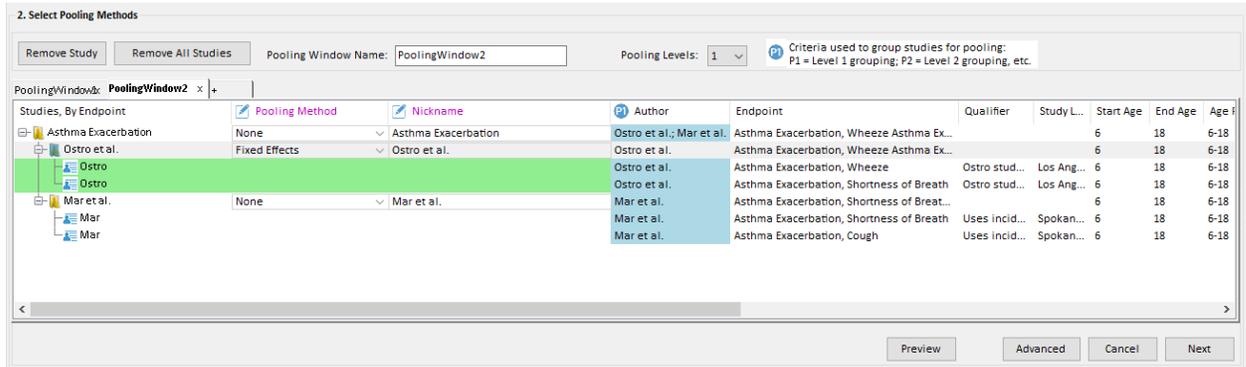
Example: Multiple Pooling Windows

There are many different ways to pool your incidence results. Sometimes you may want to look at the same results in different ways, or you may just have many results that need to be sorted by different variables. In these cases, you can open up multiple pooling windows by clicking on the rightmost + tab.

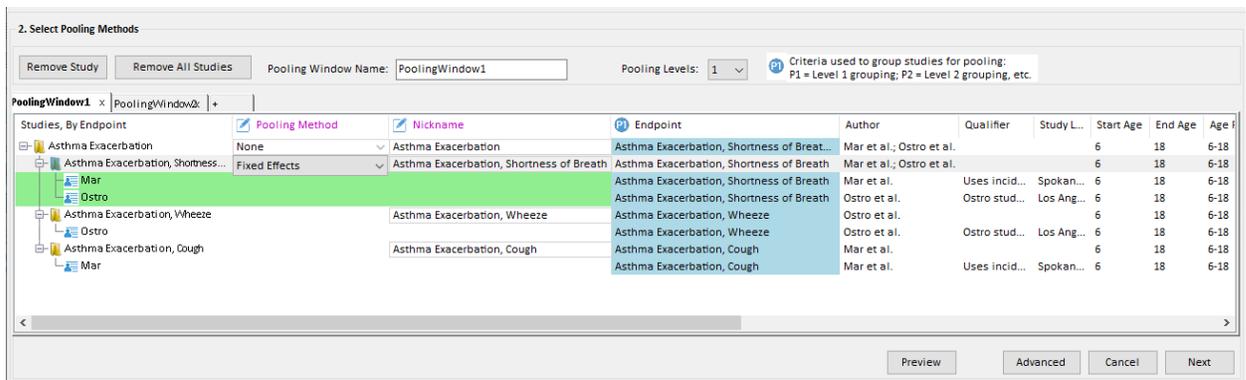
For example, you might want to pool all results of health impact functions by a particular **Author**, rather than pooling all results of health impact functions of a particular **Endpoint**. The examples below show the same set of incidence results, first sorted by **Author**, then sorted by **Endpoint**. In order to change the sorting levels of the

health impact functions, move the first category on which you wish to sort to the first position after the **Pooling Method** column. In this example, you would drag the **Author** column ahead of **Endpoint** column. As you can see, after this change the pooling options are very different.

Sorted by Author:



Sorted by Endpoint:



If you use two different pooling windows, each sorted as shown above, you can create results pooled by **Author**, and results pooled by **Endpoint**.

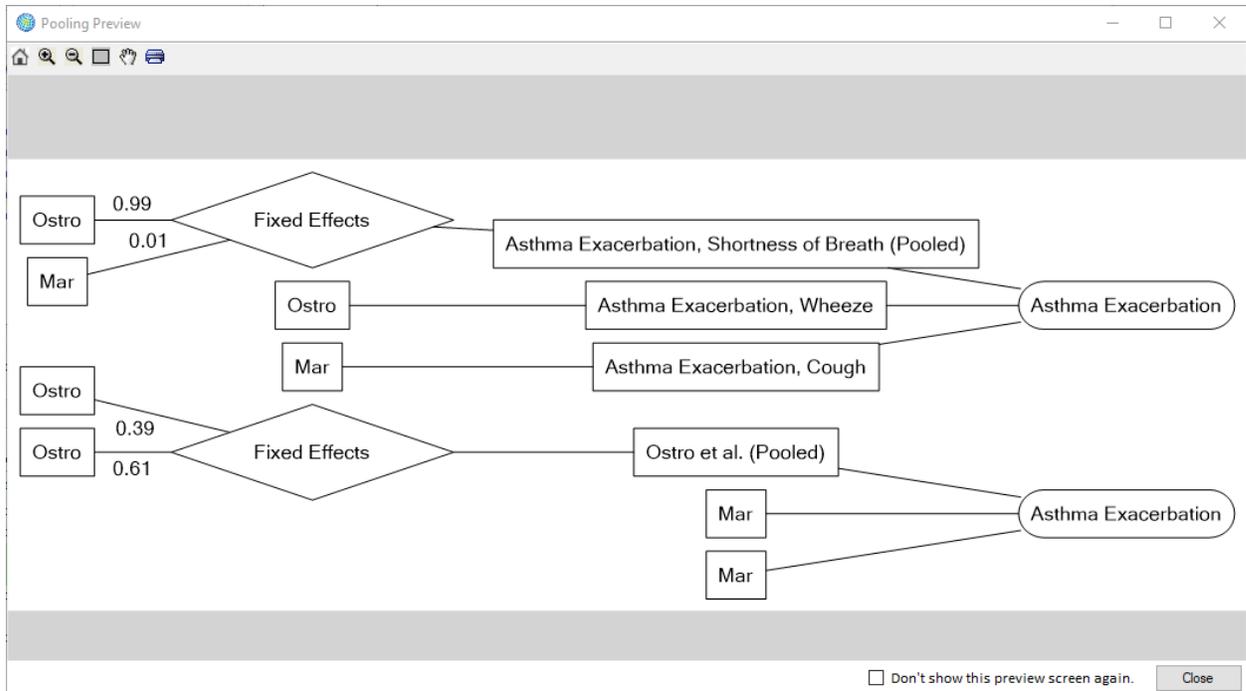
7.2.3 Valuing Pooled Incidence Results

After you have specified your incidence pooling options, click on the **Next** button and BenMAP-CE will show a **Pooling Preview**. Review and **Close** the **Pooling Preview** in order to select valuations and valuation pooling options from the **Select Valuation Methods, Pooling and Aggregation** window.

BenMAP-CE Decision Point

When might you want to pool your valuation estimates?

- You have multiple valuation functions (likely derived from different studies) for the same health endpoint that you would like to average.
- For a given endpoint, there are age-specific valuation functions for non-overlapping age groups that you would like to sum to obtain a total valuation estimate.



The **Select Valuation Methods, Pooling and Aggregation** window should look quite similar to the **Select and Pool Incidence Estimates** window, with tree views on the top half representing the valuation methods available, and various pooling windows on the lower half representing the selected valuations and pooling options.

BenMAP-CE Use Example

Example scenarios for pooling valuation estimates:

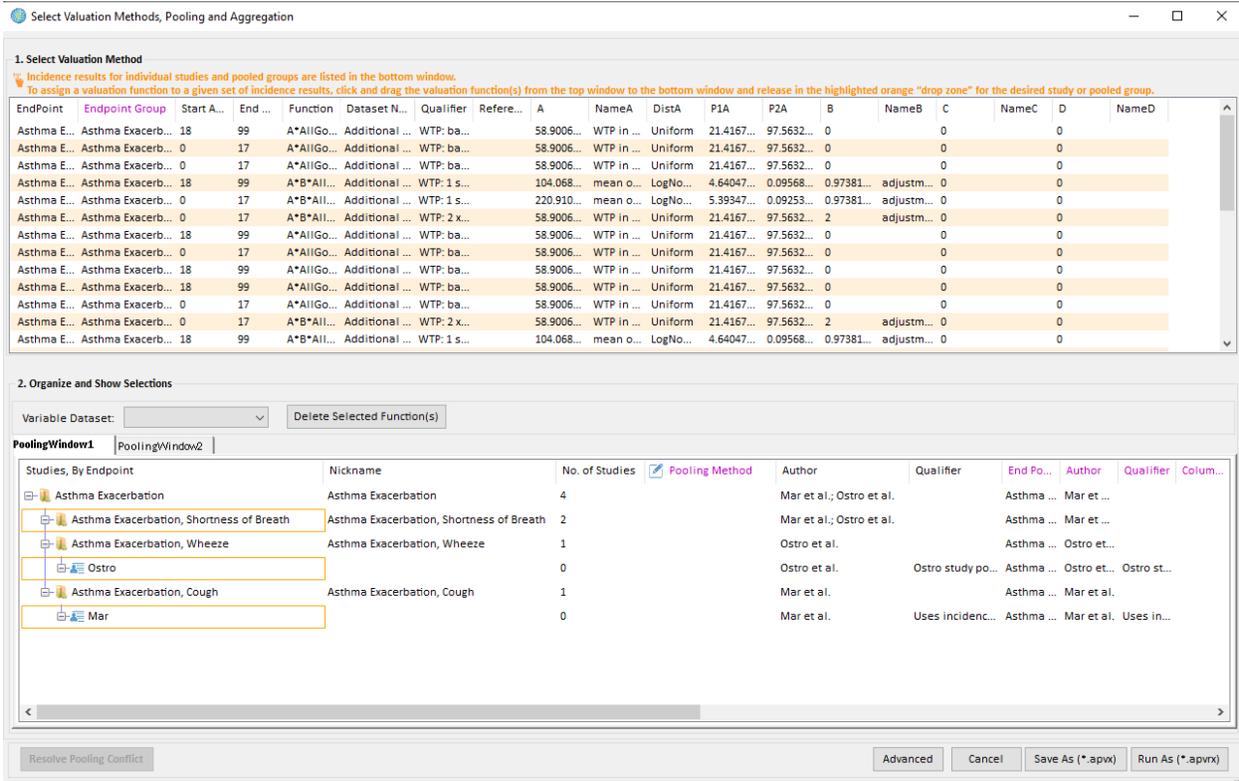
- You have two valuation functions derived from different studies for *Emergency Room Visits, Asthma* that you would like to average using equal or user-specified weights.
- You have multiple age-specific valuation functions for *Acute Myocardial Infarction* (for age groups 0-24, 25-44, 45-54, 55-64, and 65-99) that you would like to combine into a single valuation estimate for ages 0-99.

For each pooling window you created on the **Select and Pool Incidence Estimates** window, there is a corresponding pooling window in the **Select Valuation Methods, Pooling, and Aggregation** window. You will notice that the number of incidence estimates available in the valuation pooling window will reflect any pooling choices you made in the **Incidence Pooling and Aggregation** window. For example, if in the **Select and Pool**

Incidence Estimates window you pooled 5 incidence estimates into a single incidence estimate, you will see a single incidence estimate in the **Valuation Methods Pooling and Aggregation** window.

The columns present in the **Select Valuation Methods, Pooling, and Aggregation** window are determined by the incidence results left after all incidence pooling has occurred. There will be exactly enough columns in each pooling window to represent the “least” pooled incidence result. That is, the columns will be in the same order they were in the **Select and Pool Incidence Estimates** window, but the only columns

present will be those up to the level of the pooled incidence result with the most columns left over after all pooling has occurred. Here is an example:



There are several steps to take in the **Select Valuation Methods, Pooling, and Aggregation** window:

Step 1. Select your valuation methods

Valuation Methods are specific to **Endpoint Groups**, and sometimes to **Endpoints** as well. The only **Valuation Methods** which appear on the top half of the window are those which have the same **Endpoint Group** values as the pooled incidence results which are available to be valued. To select a **Valuation Method**, select it from the table and drag-and-drop it onto the appropriate incidence result in the pooling window below. Note that BenMAP-CE will only allow you to drop **Valuation Methods** onto incidence results which have the same **Endpoint Group** value. For example, BenMAP-CE will not allow you to drop a ‘*Mortality*’ valuation on a ‘*Hospital Admissions*’ incidence result. Note also that you can only drag-and-drop individual **Valuation Methods**, not entire groups of them. For explanations of the various valuation methods, see Appendix G.

BenMAP-CE Guidance and 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 endpoint 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.

If you have added any of your own valuation methods, as described in the Valuation Data section of Chapter 4: Loading Data, you can drag-and-drop them in the same way as the *EPA Standard Valuation Functions* shown in these examples.

1. Select Valuation Method

Incidence results for individual studies and pooled groups are listed in the bottom window. To assign a valuation function to a given set of incidence results, click and drag the valuation function(s) from the top window to the bottom window and release in the highlighted orange "drop zone" for the desired study or pooled group.

EndPoint	Endpoint Group	Start Age	End Age	Function	Dataset N...	Qualifier	Refere...	A	NameA	DistA	P1A
Asthma Exacerbation, Wheeze	Asthma Exacerbation	18	99	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Wheeze	Asthma Exacerbation	0	17	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	0	17	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Wheeze	Asthma Exacerbation	18	99	A*B*AllG...	Additional ...	WTP: 1 symptom-day, Dickie and Ulery (2002)		104.068...	mean o...	LogNo...	4.64047.
Asthma Exacerbation, Wheeze	Asthma Exacerbation	0	17	A*B*AllG...	Additional ...	WTP: 1 symptom-day, Dickie and Ulery (2002)		220.910...	mean o...	LogNo...	5.39347.
Asthma Exacerbation, Wheeze	Asthma Exacerbation	0	17	A*B*AllG...	Additional ...	WTP: 2 x bad asthma day, Rowe and Chestnut (19...		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Moderate or Worse	Asthma Exacerbation	18	99	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Moderate or Worse	Asthma Exacerbation	0	17	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation	18	99	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, One or More Symptoms	Asthma Exacerbation	18	99	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, One or More Symptoms	Asthma Exacerbation	0	17	A*AllGoo...	Additional ...	WTP: bad asthma day, Rowe and Chestnut (1986)		58.9006...	WTP in ...	Uniform	21.4167.
Asthma Exacerbation, Moderate or Worse	Asthma Exacerbation	0	17	A*B*AllG...	Additional ...	WTP: 2 x bad asthma day, Rowe and Chestnut (19...		58.9006...	WTP in ...	Uniform	21.4167.

2. Organize and Show Selections

Variable Dataset: Delete Selected Function(s)

PoolingWindow1 | PoolingWindow2

Studies, By Endpoint	Nickname	No. of Studies	Pooling Method	Author	Qualifier	End Po...	Author	Qualif
Asthma Exacerbation	Asthma Exacerbation	4	<input checked="" type="checkbox"/>	Mar et al.; Ostro et al.	Asthma ...	Mar et ...		
Asthma Exacerbation, Shortness of Breath	Asthma Exacerbation, Shortness of Breath	2	<input checked="" type="checkbox"/>	Mar et al.; Ostro et al.	Asthma ...	Mar et ...		
WTP: bad asthma day, Rowe and Chestnut (1986) Uniform 0-17		0	<input type="checkbox"/>	Mar et al.; Ostro et al.	WTP: ba...	Asthma ...	Mar et ...	WTP: b
Asthma Exacerbation, Wheeze	Asthma Exacerbation, Wheeze	1	<input checked="" type="checkbox"/>	Ostro et al.	Asthma ...	Ostro et...		
Ostro		0	<input type="checkbox"/>	Ostro et al.	Ostro st...	Asthma ...	Ostro et...	Ostro s
Asthma Exacerbation, Cough	Asthma Exacerbation, Cough	1	<input checked="" type="checkbox"/>	Mar et al.	Asthma ...	Mar et al.		
Mar		0	<input type="checkbox"/>	Mar et al.	Uses inc...	Asthma ...	Mar et al.	Uses i

Resolve Pooling Conflict | Advanced | Cancel | Save As (*.apvx) | Run As (*.apvx)

When BenMAP-CE runs the APV Configuration, 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 Methods** will simply be ignored when valuation results are generated, aggregated, and pooled.

Because valuation functions include an uncertainty distribution around them, generating valuation results is fairly complicated. The procedure depends on whether the incidence results being used were generated in Point Mode or with the default Monte Carlo method. See Chapter 6: Incidence Estimation (Section 6.2.3) for details on these advanced configuration settings.

In Point Mode, BenMAP-CE simply runs the valuation functions once using the point estimate of the incidence result and the mean of the valuation function as inputs.

With the Monte Carlo feature, on the other hand, BenMAP-CE generates one hundred percentile points (from the 0.5th percentile to the 99.5th percentile) to represent the distribution of the inputs to the valuation function. To get the value of the health incidence, BenMAP-CE multiplies each combination of values from the incidence result with each of the hundred valuation points, and puts the results into a holding container. (For example, if the incidence result has 10 percentile points and there are 100

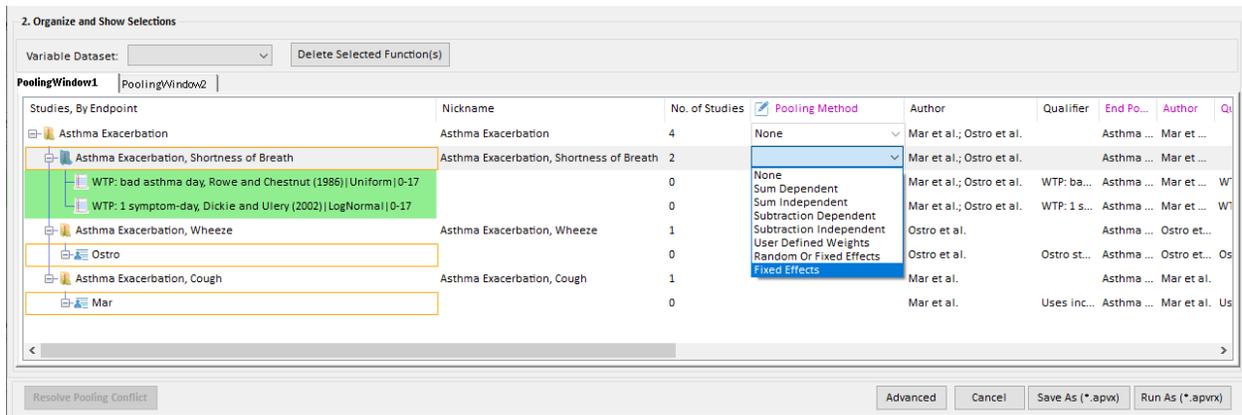
valuation points, then the holding container will have 1,000 values.) Finally, the holding container is sorted low to high and binned back down to 100 percentile points (representing the 0.5th percentile to the 99.5th percentile of the economic value of the incidence).

Step 2. Sort results

Depending on how your incidence results were pooled, the columns in the valuation pooling windows can be resorted in the same way as the **Select and Pool Incidence Estimates** window columns. This resorting will have the same sort of impact on the tree structure of valuation results that it had on the tree structure of incidence results. (See Step 2 in the section on Pooling Incidence Results.)

Step 3. Select pooling methods

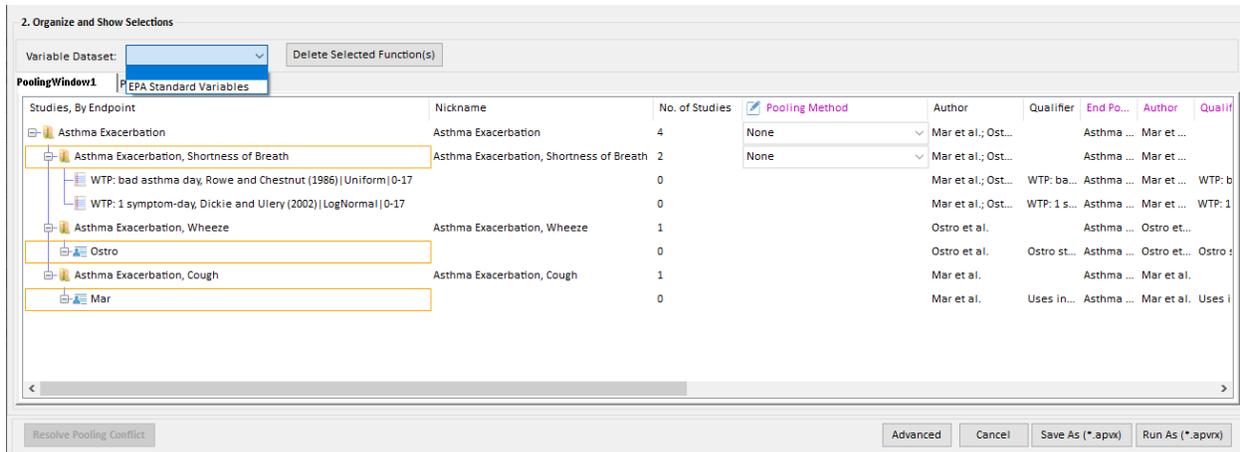
The same pooling methods are available for valuation results which were available for incidence results. (See Step 2 in the section on Pooling Incidence Results.) You should note that when more than one valuation method is selected for a particular pooled incidence result, it is possible to pool the generated valuation results.



Step 4. Select Variable Dataset

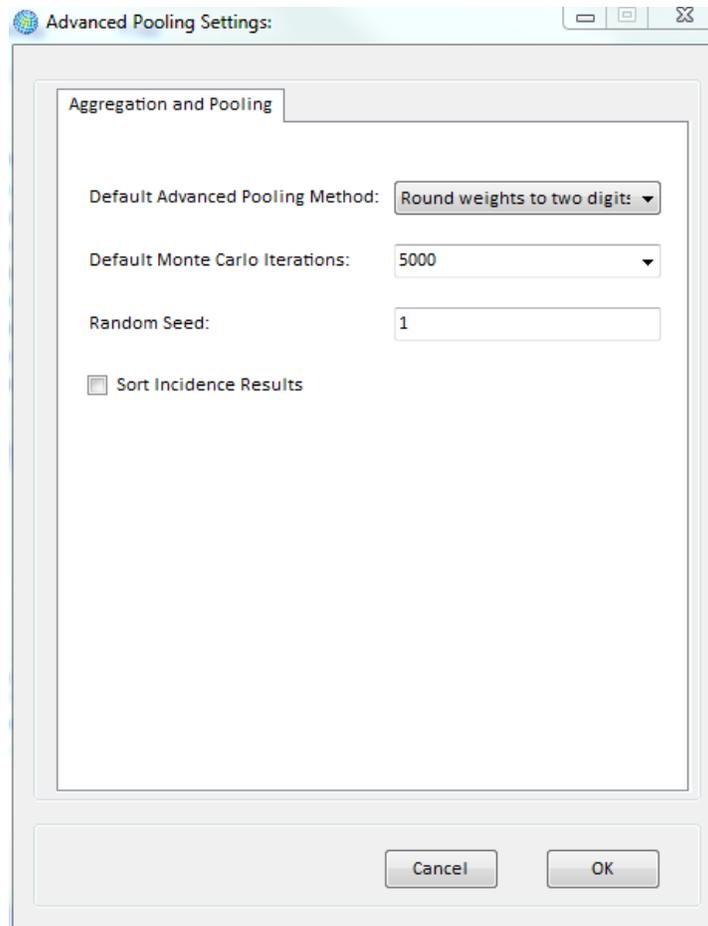
Before proceeding to the next step, you have the option to select a **Variable Dataset** from the drop-down menu beneath the pooling window (this is not required to proceed to the next step). The **Variable Dataset** can include a variety of data, such as income and poverty data that might be used in health or valuation functions. For the default EPA health and valuation functions, you just need to select the *EPA Standard Variables*.

If you have developed your own setup, then you have the option to load and use a **Variable Dataset**. This is not necessary if you do not need the extra variables that can be included in this dataset.



7.2.4 APV Configuration Advanced Settings

When specifying the incidence pooling options, you may click on the **Advanced** button on the bottom of the **Select and Pool Incidence Estimates** window. This button will open the **Advanced Pooling Settings** window. The features of this window are described in the next section.



7.2.4.1 Aggregation and Pooling

Default Advanced Pooling Method

The relative contribution of any one study in the pooling process depends on the weight assigned to that study. A key component of the pooling process, then, is the determination of the weight given to each study. BenMAP-CE lets users assign “subjective” weights and it assigns weights using a fixed effects or a random effects approach. There are three options for using weights available in the **Default Advanced Pooling Method** drop-down list:

Round weights to two digits. BenMAP-CE rounds each weight to two digits (e.g. 0.73), and then multiplies these weights by 100 to get two-digit integers. Each entire distribution (set of percentile points) is then put into a holding container an integral number of times, according to its integral weight. This holding container is then sorted low to high and binned down to the appropriate number of percentile points.

Round weights to three digits. BenMAP-CE rounds each weight to three digits (e.g. 0.732), and then multiplies these weights by 1,000 to get three-digit integers. Each distribution (set of percentile points) is then put into a holding container for an integral number of times, according to its integral weight. This holding container is then sorted low to high and binned down to the appropriate number of percentile points.

Use exact weights for Monte Carlo. BenMAP-CE uses exact weights and a Monte Carlo simulation. During each iteration of the procedure, a particular result is selected with a probability equal to its weight. Once a result is selected, one of its percentile points is chosen at random and put into a holding container. This is done some number of times (see *Monte Carlo Iterations*, below), and the holding container is then sorted low to high and binned down to the appropriate number of percentile points.

Default Monte Carlo Iterations

This drop-down list is only enabled when *Use exact weights for Monte Carlo* is selected as the **Default Advanced Pooling Method**. It specifies the number of iterations the Monte Carlo simulation should be run (see above). Its initial value is set by the **Default Monte Carlo Iterations** value from the **Advanced Pooling Settings** window (see Step 1, above).

Random Seed

The **Advanced Pooling Settings** window allows the specification of a **Random Seed**. Many of the pooling methods require the generation of sequences of random numbers, e.g. choosing a random percentile point during a Monte Carlo simulation. Providing a specific **Random Seed** value allows you to ensure that the same sequence of random

numbers is generated as in a previous analysis, thus allowing exact results to be reproduced.

If you do not set the **Random Seed** for a particular run, one will be generated automatically from the system clock (the number generated will depend on the date and time, and should change every minute). Normally, you should not set the **Random Seed** value. If you need to reproduce a specific set of results, however, the random seed used to generate previous APV Configuration Results can be determined from an APV Configuration Result file (.apvrx) Audit Trail Report.

Sort Incidence Results

The *Sort Incidence Results* should generally be always checked. This setting ensures that the incidence percentile-point results are sorted low to high.

7.2.4.2 Currency Year and Income Growth

The **Currency Year and Income Growth** window, accessed via the **Advanced** button on the **Valuation Method** window, allows you to specify an **Inflation Dataset** and a **Currency Year**, which in combination allow you to change the currency year to account for inflation. The **Income Growth Adjustment** panel allows you to adjust the valuation estimates to account for the growth in income over time for WTP functions.

Advanced Valuation Settings:

Currency Year and Income Growth

Inflation Adjustment

Inflation Dataset: EPA Standard Inflation

Currency Year: 2010

Income Growth Adjustment

Income Growth Dataset: EPA Standard Income Growth

Income Growth Year: 2010

Endpoint Groups:

- Acute Bronchitis
- Acute Respiratory Symptoms
- Asthma Exacerbation
- Chronic Asthma
- Chronic Bronchitis
- Lower Respiratory Symptoms
- Mortality
- Upper Respiratory Symptoms

Select the income growth dataset, then the year and the endpoint groups to which you want to apply the adjustment. You may select more than one endpoint group.

Cancel OK

Inflation Adjustment

The **Inflation Adjustment** needs to be carefully considered in relation to the valuation dataset that you are using. (This is discussed in detail in the section on loading inflation data in Chapter 4.) The default valuation database in the *United States* setup has a currency year of *2000*, so the inflation dataset has a value of *1* for the year *2000*.

Income Growth Adjustment

Willingness to pay (WTP) estimates are believed to be tied to the income of individuals. As income rises over time, 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.

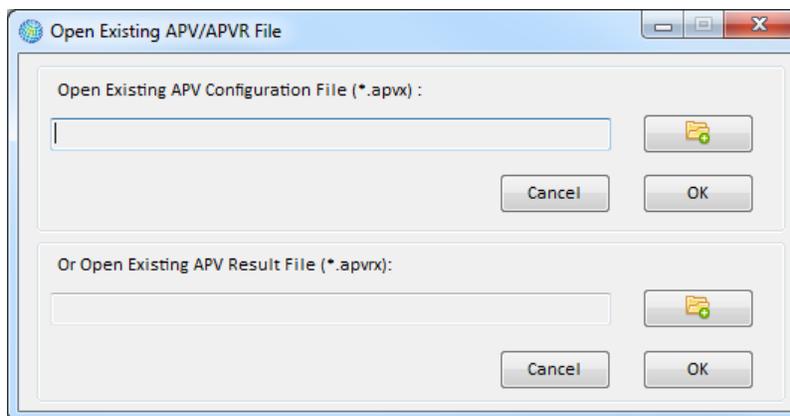
As with the **Inflation Adjustment**, the **Income Growth Adjustment** has a close connection to the valuation estimates. For example, the valuation estimates in the *United States* setup are assumed to be based on income levels from 1990, so the income

growth adjustment database has a value of 1 for the year 1990. (This is discussed in detail in the section on loading income growth data in Chapter 4.)

To use the **Income Growth Adjustment**, you need to choose a dataset and then choose the income year that you want to use. By default, BenMAP-CE sets the Income Growth Year equal to the user-specified Population Year or to the latest year for which income growth adjustment data are available if less than the population year.

7.3 Open & Modify Existing APV Configuration

If you have an existing APV configuration (*.apvx) file or APV result file (*.apvrx), you can open, and edit it. Double click **Aggregate, Pool & Value** from the main tree menu to load the APV Configuration or APV Result file.



If you have only a few changes to make to an existing configuration, it is typically much quicker to open the previous configuration, rather than entering all of your choices again. Note that the various parts of an APV Configuration are quite interdependent, so modifying part of the configuration may cause other parts to be reset. For example, modifying the tree structure for incidence pooling will cause the valuation method selection and valuation pooling tree structure to be cleared and reset. Changing the **Configuration Results Filename** in the **Select and Pool Incidence Estimates** window will not reset the incidence or valuation pooling trees as long as the new file contains incidence results generated from the same health impact functions as the old file. This can be quite helpful for generating new APV Configuration Results from several different Configuration Results files which were generated from different baseline/control scenarios, but with the same set of health impact functions.

7.4 Run APV Configuration

After having specified the various aggregation, pooling, and valuation options, you can save your APV Configuration for future use. The file that you save has an “.apvx” extension. The configuration that you have specified for APV is similar to the configuration that you developed for choosing health impact functions. (That configuration has a “.cfgx” extension.) Both files allow you to save choices that you have made, and re-run them at a later time.

To save your APV configuration with your valuation pooling choices, click the **Save As (*.apvx)** button, and name your configuration file. We suggest that you save this in the Configurations folder. When ready to generate APV Configuration results, click the **Run As (*.apvrx)** button. BenMAP-CE then requires that you specify a file in which to save the results, with an “.apvrx” extension.

7.5 View and Export Pooled Incidence and Valuation Results

Using the results from the APV Results file (“.apvrx” extension), you can create, view and export reports that reflect the choices you made about how to aggregate, pool and value your results.

7.5.1 Pooled Incidence Results

The **Pooled Incidence Results** report provides results aggregated and pooled to the level that you previously specified in the Aggregation, Pooling, and Valuation Configuration file. This report has fewer Health Impact Function fields than the **Aggregated Incidence Results Report**, and values for others will be blank. This is because after pooling, only enough fields are retained to uniquely identify individual results.

To generate pooled incidence results, click the **Pooled Incidence** tab in the upper portion of the main window. Double-click to select the study results you would like to view. The selected results should show up in the **Data** tab below, if not, click the **Show Results** button. Notice that you cannot re-aggregate the results in this stage, because you have already defined how to aggregate the results. You may also view the results on a map using the **GIS Map** tab and on a bar chart using the **Chart** tab.

7.5.2 Pooled Valuation Results

The **Pooled Valuation Results** report presents valuation results aggregated and pooled to the level you specified using the **Advanced** button when creating the APV configuration file. As with the **Pooled Incidence Results Report**, fewer Pooled Valuation Method fields are available, because only enough fields are retained to uniquely identify individual results.

Click the **Pooled Valuation Results** tab to begin viewing these results. Similar to the Pooled Incidence Results, you can double-click the study of interest and view the results table in the **Data** tab. Similarly, you may view the results on a map using the **GIS Map** tab and on a bar chart using the **Chart** tab.

7.6 Frequently Asked Questions

I am at the BenMAP-CE valuation window and cannot proceed. What should I do?

In order to proceed to the next step, you must select a Variable Dataset from the drop-down menu in the Select Valuation Methods, Pooling, and Aggregation window. The

files in the Variable Dataset can include a variety of data, such as income and poverty data that might be used in health or valuation functions. For the default EPA health and valuation functions, you just need to select the EPA Standard Variables.

If you have developed your own setup, then you need to make sure that you also load a Variable Dataset. This is necessary even if you do not need the extra variables that can be included in this dataset.

How do I edit or add other valuation functions?

To edit or add valuation functions you need to go to Modify Setup option in the Tools drop- down menu available in the upper left-hand corner of the main BenMAP-CE window. See the valuation function section in the chapter on Loading Data for details on how to do this.

How do I know what year dollars (currency year) were used?

You can find the answer in the Audit trail for the APVR file that you generated.

Do the currency year and year of the population data have to match?

No. The currency year and the year of the population data do not need to match. Currency years are always historical because we do not forecast inflation.

Chapter 8

GIS/Mapping

In this chapter...

- **Learn about BenMAP-CE's mapping functions.**
- **Map different variables and modify the map display.**

The BenMAP-CE Geographic Information System (GIS) will display maps of air quality, health and economic data. These maps can help answer a number of questions:

- *Quality assurance*: Do your air quality changes seem to be distributed correctly? Are your air quality changes and health impacts occurring in approximately the same location?
- *Presentations*: In what states or provinces are most of the benefits/disbenefits of your policy scenario concentrated?
- *Analysis*: Which air quality grid cells contain the highest ozone values?

The main **GIS Map** tool will be available once you have successfully completed the first stage of the BenMAP-CE analysis (**Air Quality Surfaces** in the main BenMAP-CE tree menu).

8.1 Overview of Mapping

The GIS will map three categories of data:

1. *Air quality (.aqgx)*. Air quality grid maps represent summary air quality metrics (e.g., daily average, daily maximum, or other metric where available) within each grid cell. Air Quality Grids can be added to the **GIS Map** by following the steps in Chapter 5: Creating Air Quality Grids.
2. *Incidence (.cfgrx)*. A configuration results file contains the results of your analysis reported at each air quality grid cell. These results have not been aggregated, pooled or valued. For more information on choosing configuration settings, see Chapter 6: Estimating Incidence.
3. *Aggregated, pooled and valued results (.apvrx)*. These are results that have been aggregated to a coarser spatial scale, see Chapter 7: Aggregation, Pooling, and Valuation.

When mapping APV Configuration results, you can generate six different types of maps: (1) *Incidence*, (2) *Aggregated Incidence*, (3) *Pooled Incidence*, (4) *Valuation*, (5) *Aggregated Valuation*, and (6) *Pooled Valuation*. For more information on how to load these files into BenMAP-CE, see Chapters 6 and 7 (Estimating Incidence, and Aggregation, Pooling, and Valuation).

8.2 Results Panel

Once you have gone through all the steps outlined in the previous chapters to import data and files, you are ready to begin exploring the visual results. Each of your selected endpoint groups will be displayed in the results panel (upper right portion of the main BenMAP-CE window).

The screenshot displays the BenMAP-CE 1.4 software interface. The top menu bar includes 'File', 'United States', 'Modify Datasets', 'Tools', and 'Help'. The main window is divided into several panels:

- Left Panel:** A tree view showing the project structure, including 'Air Quality Surfaces', 'Source of Air Quality Data (PM2.5)', 'Baseline', 'Control', 'Estimate Health Impacts', 'Aggregate, Pool & Value', and 'Valuation Method'.
- Top Panel:** Four tabs are visible: 'Health Impact Results', 'Pooled Incidence Results' (highlighted with a red box), 'Pooled Valuation Results', and 'Audit Trail Report'. The 'Pooled Incidence Results' tab contains a table with the following columns: Endpoint Group, Endpoint, Pollutant, Author, Start Age, End Age, Dataset Name, Geographic Area, Race, Ethnicity, Gender, and Incidence. The table lists several mortality studies for PM2.5.
- Bottom Panel:** A 'GIS Map' panel showing a map of the United States. The map is overlaid with a grid and color-coded according to a legend. The legend includes categories for 'QuarterlyMean' and 'D24HourMean' with various value ranges.

There are four tabs within the results panel, **Health Impact Results**, **Pooled Incidence Results**, **Pooled Valuation Results**, and **Audit Trail Report**. To create a results map layer for an endpoint group, select an entry, or entries, from the list and drag it down onto the GIS Mapping panel. BenMAP-CE will create the map and place an entry into the GIS table of contents below the newly created results group. The layer will most likely be listed under the author's name as subgroup of the main results group. This process of dragging-and-dropping can be done with as many entries as you like, on any of the first three tabs.

Audit Trail Reports facilitate transparency and reproducibility by reporting a summary of your assumptions underlying each step of the analysis. This is described in more detail below.

8.3 GIS Mapping Panel

The GIS Mapping panel is the centerpiece of this tool. Here is where you can view, edit, add, and remove layers from the table of contents and GIS map viewer.

The screenshot displays the BenMAP-CE 1.4 software interface. The main window is titled "United States" and contains several panels:

- Left Panel:** A tree view showing the project structure, including "Air Quality Surfaces", "Source of Air Quality Data (PM2.5)", "Baseline", "Control", "Air quality delta (baseline - control)", "Estimate Health Impacts", "Aggregate, Pool & Value", and "Valuation Method".
- Top Panel:** A menu bar with "File", "United States", "Modify Datasets", "Tools", and "Help". Below it are tabs for "Health Impact Results", "Pooled Incidence Results", "Pooled Valuation Results", and "Audit Trail Report".
- Table:** A table with columns: Endpoint Group, Endpoint, Pollutant, Author, Start Age, End Age, Dataset Name, Geographic Area, Race, Ethnicity, Gender, and Incidence. The table lists several mortality data points for PM2.5.
- Right Panel:** A "GIS Map" tab showing a map of the United States with a color-coded overlay. A "Map Layers" table of contents is visible on the left side of the map viewer, listing layers such as "Region Admin Layers", "Results", "Health Impacts", "Incidence", "Pollutants", and "D24HourMean".

8.3.1 GIS Map Tab

After creating an air quality surface, the **GIS Map** tab is used to view the air quality data (double-click on an available air quality surface to display it). Here you will find a GIS table of contents, toolbar, and interactive map.

8.3.1.1 GIS Table of Contents

The GIS table of contents is where you will find all your loaded map layers. The layers are sorted in groups, with subgroups below them. The general setup will include *Region Admin Layers* group, *Pollutants* group, and *Results* group. You can select or deselect as many layers or groups as you like, for viewing on the map to the right.

The screenshot displays the BenMAP-CE 1.4 software interface. At the top, there is a menu bar with 'File', 'United States', 'Modify Datasets', 'Tools', and 'Help'. Below the menu bar, there are several tabs: 'Health Impact Results', 'Pooled Incidence Results', 'Pooled Valuation Results', and 'Audit Trail Report'. The 'Health Impact Results' tab is active, showing a table with columns: Endpoint Group, Endpoint, Pollutant, Author, Start Age, End Age, Dataset Name, Geographic Area, Race, Ethnicity, Gender, and Incidence. The table contains several rows of mortality data for PM2.5, with authors like Krewski, Laden, Pope, Woodruff, and Iannelli.

Below the table, there are options to 'Create map, data and chart (double-click the selected study)', 'Create data (table) for multiple studies', and 'Aggregation for raw data'. There are also buttons for 'Select Result Fields' and 'Show Results'.

The bottom part of the interface shows a 'GIS Map' view with a legend on the left and a map of the United States on the right. The legend is highlighted with a red box and contains the following categories and values:

- Map Layers**
 - Region Admin Layers
 - Results
 - Health Impacts
 - Krewski
 - Incidence
 - <= 191,434
 - 191,434 - 474,618
 - 474,618 - 695,173
 - 695,173 - 981,972
 - 981,972 - 1,341,829
 - 1,341,829 - 2,889,078
 - Pollutants
 - PM2.5
 - Quarterly Mean
 - Delta
 - <= 1.150
 - 1.150 - 1.620
 - 1.620 - 1.820
 - 1.820 - 2.140
 - 2.140 - 2.440
 - 2.440 - 2.770
 - D24HourMean
 - Delta
 - <= 1.320
 - 1.320 - 1.620
 - 1.620 - 1.870
 - 1.870 - 2.140
 - 2.140 - 2.440
 - 2.440 - 2.840

The map shows the United States with various colored regions corresponding to the legend values. The current setup is 'United States'.

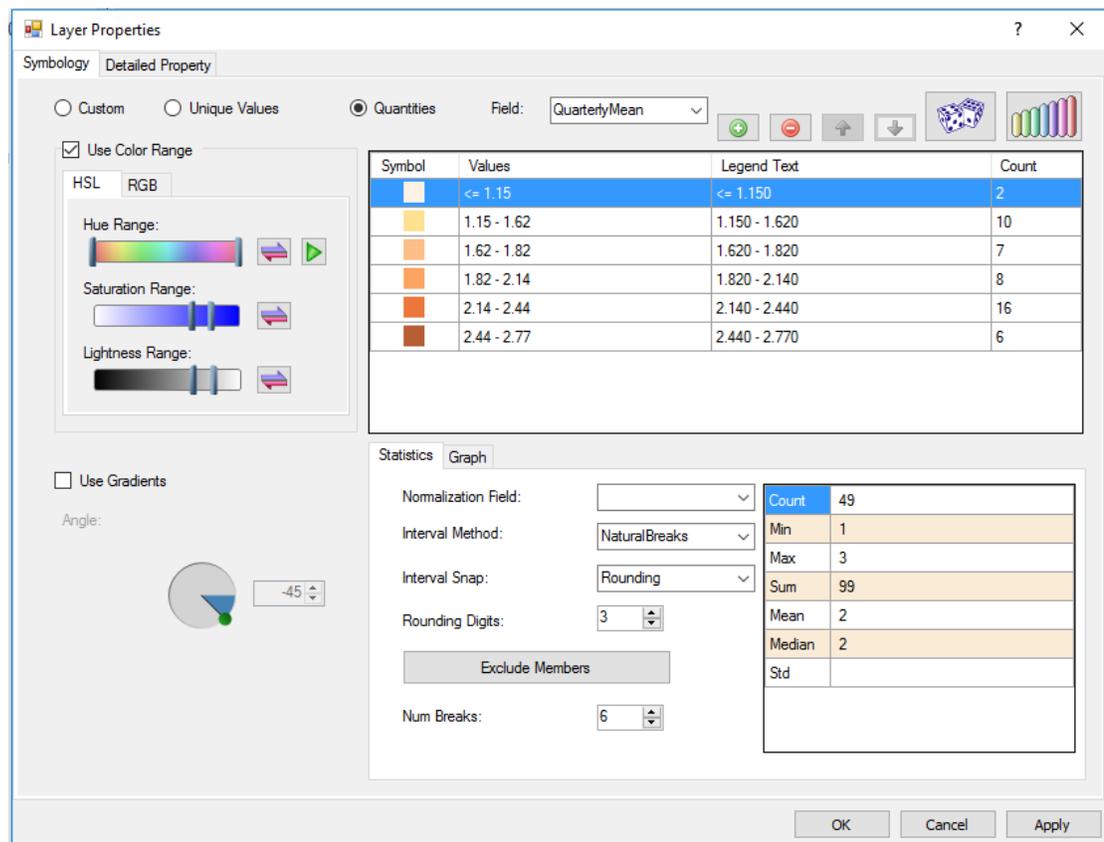
- Region Admin Layers:** This is where the administrative grids can be found for country, state, and county boundaries. For preloaded data, regional administrative layers have been defined. For example, in the *United States* setup, the *Nation*, *State*, and *County* layer will be automatically selected. As discussed in Chapter 4, you can preselect default administrative layers for new setups you create when you import air quality grids. You can turn administrative layers on or off by expanding the group.
- Pollutants:** This is where all the available pollutant data (from the tree menu) will be visible. Under this main group, there will be a group for each pollutant that was selected (e.g., *PM_{2.5}*). Below the individual pollutant, there will be metrics that were defined earlier during import and loading (e.g., *Quarterly Mean*, *D24HourMean*). Below each metric, there will be entries for each Air Quality Grid that was loaded (e.g., *Baseline*, *Control*, and *Delta*).
- Results:** This is where the layers for Health Impact Results, Pooled Incidence Results, and Pooled Validation Results will be listed. Under the results group, there will be a subgroup labeled for each set of results. Usually, these entries will be labeled by the study author's name.

Color Ramps

The color ramps are standard for the imported files. The default color ramp for the **Baseline** and **Control** Air Quality Grids goes from light green to dark blue. The default color ramp for the **Delta** Air Quality Grid goes from light yellow to dark red. Finally, the default color ramp for the **Results** group entries will be different from each other and any other color ramps that are already in the table of contents.

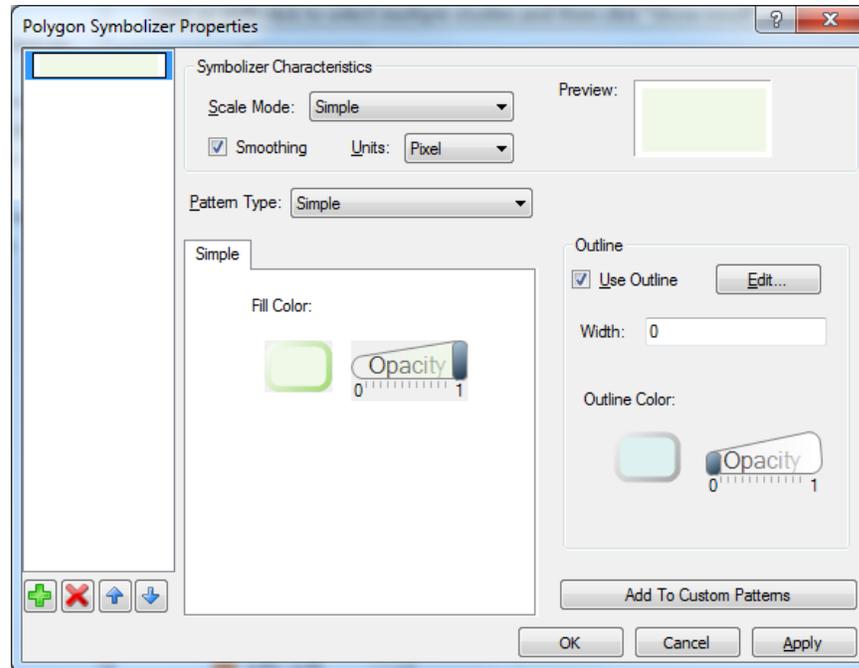
These color ramps can be changed by the user, if desired. There are two possible ways to change the colors:²⁵

- 1) To change the whole color scheme: Right click on a layer and select the **Properties** option. This will open a **Layer Properties** window, where you can change the color ramps and other properties.



- 2) To change one index within a color ramp: Click on the color box or number range that you wish to change. This opens the **Polygon Symbolizer Properties** window.

²⁵ BenMAP-CE uses DotSpatial to incorporate, analyze, and map spatial data. The editing tools for layer properties are those included in the DotSpatial libraries and have not been customized for BenMAP-CE. For more information, see: <http://dotspatial.codeplex.com/>.



Add/Remove a Group

To add a group to the GIS table of contents, right-click on a group, and click **Create New Group** from the pop-up menu. This will add the new group below the group that was right-clicked on.

To remove a group from the GIS table of contents, right-click on the group you wish to remove, and then click **Remove Group** from the pop-up menu. This will remove the entire group from the table of contents.

Remove a Layer

To remove a spatial layer from the GIS table of contents, right-click on the layer you wish to remove, and then choose **Remove Layer** from the pop-up menu. This will remove the layer from the table of contents.

Note: Adding a layer will be discussed in the GIS Toolbar section (Section 8.3.1.2).

8.3.1.2 GIS Toolbar

There are a number of standard buttons used in most map viewing programs which you can use to navigate and customize the map view. To see the name of each button in the toolbar (to the left of the GIS table of contents), simply hold the cursor over it.



Increase zoom. Allows you to zoom in.



Decrease zoom. Allows you to zoom out.



Drag mode. Allows you to manually move the map by clicking and dragging.



Zoom to full extent. Allows you to view the whole map that you are viewing.



Click to display info for the cell in popup window. Allows you to display information (all the variable values) for individual cells or points by clicking on them.



Attribute Table. Opens an Attribute Table Editor Window where the user can view, edit, or filter the data for a specific layer.



Select Features. Allows you to select features from the map. Press and hold Ctrl to select multiple features at one time.



Select By Location. Allows you to select features from a layer based on its spatial relationship with another layer.



Clear Selection. Clear selections from all layers.



Add Layer. Allows you to add a new layer to the table of contents and map. Typically the new layer is added near the top of the table of contents.



Export Map Image. Prepare print layout of GIS map and legend.



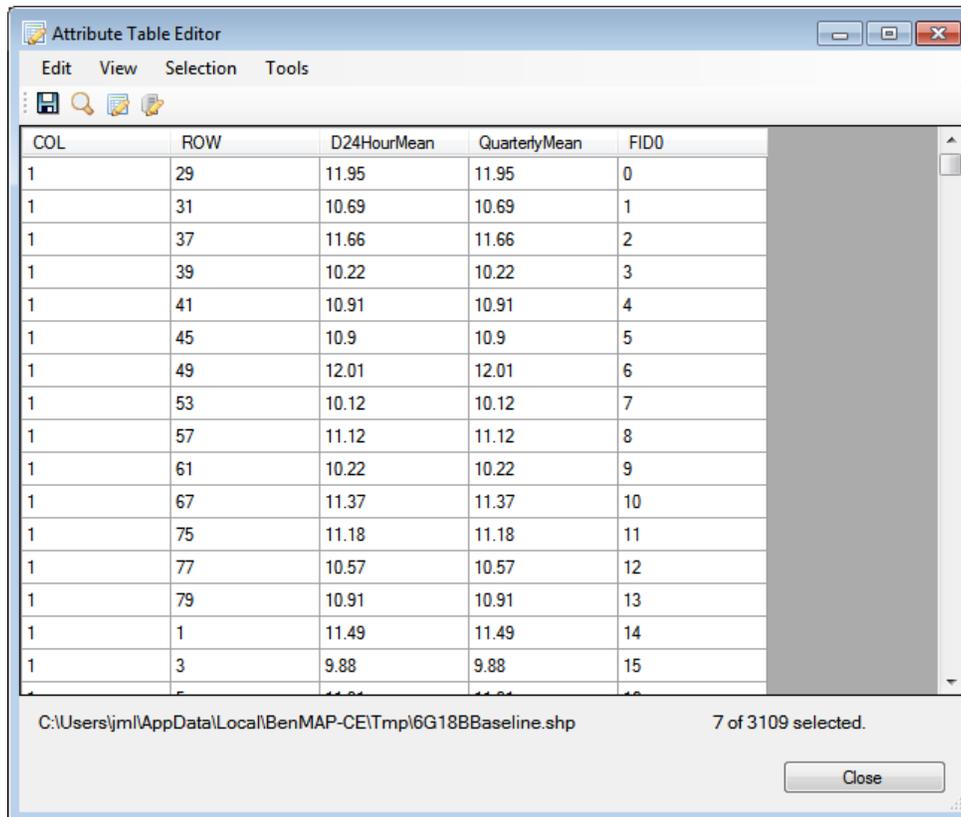
Save Shapefile. Save and export results from a GIS layer on current map.



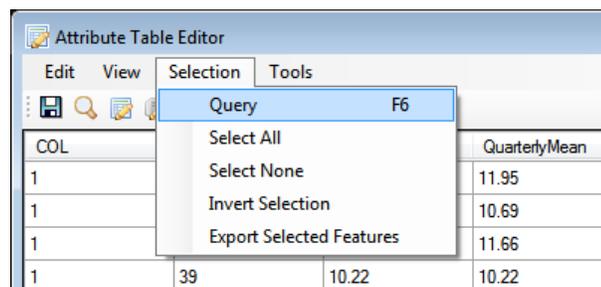
Change Projection to *projection type*. Allows you to change/toggle the type of map projections between the following types: GCS/NAD 83 (standard), Albers.

Creating a Query

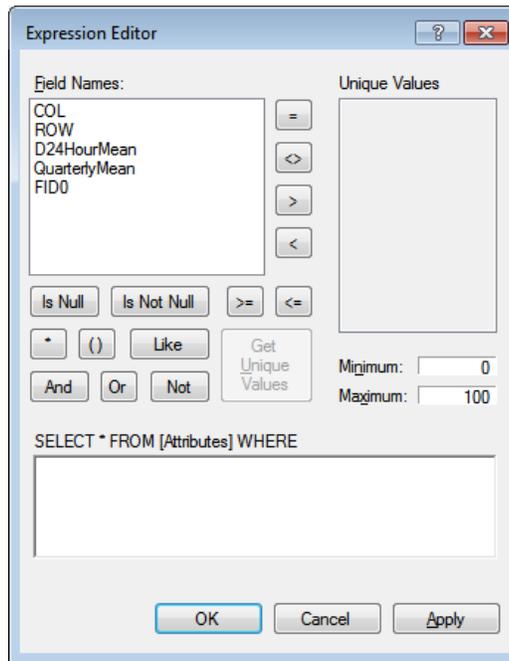
You can create a query on any map layer that shows up in the GIS table of contents. After importing a file (such as **Baseline** or **Control**) you can begin a query by selecting a layer. The selected layer will become highlighted with a light blue bubble around it. Next, click the **Attribute Table** button in the GIS toolbar. This will open the **Attribute Table Editor** window, shown below:



From the window above, click **Selection** from the top menu bar. This will open a drop-down menu where you can select the **Query** option.

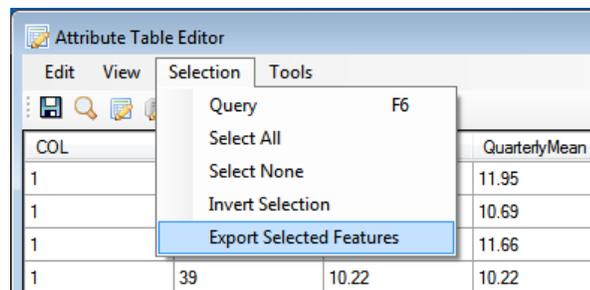


This will open **Expression Editor** window (shown below):



The **Expression Editor** is used to query your data. First, select a **Field Name** to query, by double-clicking on a desired entry. The **Field Name** should show up in brackets in the bottom text box. Next, select an operator from the panel of buttons. The selected operator symbol should show up in the bottom text box next to the field name. Finally, to complete the first query entry, click in the bottom text box (after the operator symbol) and enter a value that you would like to compare against (e.g., `[D24HourMean] >= 15`). More attributes can be added to the query by clicking the **And**, **Or**, or **Not** buttons. Once you are satisfied with the query statement, click **OK** on the **Expression Editor** window. It may take a few minutes to find all the results. The **Attributes Table Editor** should appear, with the requested selections highlighted. The map on the GIS portion of the main BenMAP-CE home screen should show the query results.

To save these results, click the **Selection** button from the top menu bar of the **Attribute Table Editor** window. From the dropdown menu, select the **Export Selected Features** entry. This will open a **Save As** window, where you can save the query into a shapefile (.shp). Click the **Close** button on the **Attribute Table Editor**.



To add the query as a new layer, click the ‘+’ button located in the GIS toolbar (located next to the GIS table of contents). An **Open** window will be displayed, where you can select your recently saved shapefile. Select the file and click **Open**. The new layer should appear near the top of the GIS table of contents. The layer can be dragged-and-dropped into any map group you would like within the GIS table of contents.

8.3.2 Data Tab

The **Data** tab allows you to view all the data that is being presented in the map.

Column	Row	Endpoint	Author	Start Age	End Age	Point Estimate	Population	Delta	Mean	Baseline	Percent of Baseline	Stdev
1	1	Mortality ...	Krewski...	30	99	699.2200	2,740,782.0...	2.2600	698.8376	48,860.0063	1.3075	101.6
4	1	Mortality ...	Krewski...	30	99	450.7856	3,699,794.0...	1.4800	450.5415	52,499.0000	0.9582	71.85
5	1	Mortality ...	Krewski...	30	99	428.5873	1,854,889.0...	2.5800	428.3016	28,945.2402	1.4797	68.09
6	1	Mortality ...	Krewski...	30	99	2,889.0779	14,319,545...	2.7700	2,887.34	180,444.0...	1.6001	458.7
8	1	Mortality ...	Krewski...	30	99	308.0581	2,934,973.0...	1.5600	307.8694	34,042.0195	0.9044	49.08
9	1	Mortality ...	Krewski...	30	99	379.2871	2,216,278.0...	2.0400	379.0562	32,098.1250	1.3810	60.35
10	1	Mortality ...	Krewski...	30	99	47.7365	288,499.0000	2.0000	47.7088	4,120.1387	1.1579	7.997
12	1	Mortality ...	Krewski...	30	99	1,231.6730	7,917,879.0...	1.6200	1,230.99	131,096.6...	0.9390	196.2
13	1	Mortality ...	Krewski...	30	99	981.9724	5,542,535.0...	2.2200	981.3879	76,403.8828	1.2645	156.1
16	1	Mortality ...	Krewski...	30	99	184.1618	877,358.0000	2.6400	184.0466	12,064.0967	1.2526	29.25
17	1	Mortality ...	Krewski...	30	99	880.6256	4,231,559.0...	2.3900	880.0905	63,676.1797	1.3821	139.9
18	1	Mortality ...	Krewski...	30	99	938.4200	3,781,983.0...	2.7100	937.8281	59,898.4375	1.3657	149.0
19	1	Mortality ...	Krewski...	30	99	377.5955	1,814,652.0...	2.2500	377.3704	29,248.4883	1.2902	60.05
20	1	Mortality ...	Krewski...	30	99	279.4019	1,818,448.0...	2.2200	279.2356	21,739.2891	1.2845	44.44
21	1	Mortality ...	Krewski...	30	99	567.1401	2,564,815.0...	2.2900	566.7996	42,787.0313	1.3247	90.18
22	1	Mortality ...	Krewski...	30	99	474.6178	2,408,922.0...	1.9900	474.3432	41,168.9375	1.1522	75.54
23	1	Mortality ...	Krewski...	30	99	85.9573	573,478.0000	1.6200	85.9099	9,149.1172	0.9390	13.69
24	1	Mortality ...	Krewski...	30	99	414.9869	2,350,041.0...	2.0800	414.7440	34,447.9414	1.2040	66.03
25	1	Mortality ...	Krewski...	30	99	346.7676	2,344,120.0...	1.7300	346.5735	34,573.4453	1.0024	55.23
26	1	Mortality ...	Krewski...	30	99	877.7975	4,882,136.0...	1.3500	877.4289	75,388.0859	0.9988	108.0
27	1	Mortality ...	Krewski...	30	99	274.8764	3,116,816.0...	1.1500	274.7341	41,158.2500	0.6679	43.83
28	1	Mortality ...	Krewski...	30	99	415.5616	1,680,361.0...	2.4000	415.3088	29,924.0605	1.3879	66.06
29	1	Mortality ...	Krewski...	30	99	793.7341	3,536,052.0...	2.1300	793.2552	58,860.9219	1.3477	126.2
30	1	Mortality ...	Krewski...	30	99	55.8210	607,318.0000	1.0200	55.7927	9,419.9932	0.5923	8.909
31	1	Mortality ...	Krewski...	30	99	168.5165	1,055,517.0...	1.8000	168.4213	16,151.3418	1.0428	26.83
32	1	Mortality ...	Krewski...	30	99	319.0060	1,590,070.0...	2.4900	318.8098	22,146.7148	1.4395	50.69
33	1	Mortality ...	Krewski...	30	99	92.7804	893,001.0000	1.9300	92.7308	11,501.7021	0.8062	14.79
34	1	Mortality ...	Krewski...	30	99	862.3339	4,660,517.0...	2.2900	861.8162	65,057.5156	1.3247	137.1
36	1	Mortality ...	Krewski...	30	99	171.4481	1,187,169.0...	1.7600	171.4481	17,168.4481	1.1817	17.81

At the bottom of this tab, there are a few options:

- The left-most set of buttons allows you to toggle between pages of data.
- The middle option allows you to change the number of digits that appear after the decimal point.
- The far right entry allows you to export the data table. Clicking the **Export** button opens a **Save As** window, allowing you to save the data as a .csv file.

8.3.3 Chart Tab

The **Chart** tab allows you to select certain regions of data within the main layer to compare localized results more easily.

The screenshot shows the BenMAP-CE 1.4 software interface. The top panel displays a table with columns: Endpoint Group, Endpoint, Pollutant, Author, Start Age, End Age, Dataset Name, Geographic Area, Race, Ethnicity, Gender, and Incidence. The table contains several rows of mortality data for PM2.5. Below the table, the 'Chart' tab is active, showing a 'Bar Graph' configuration. On the left, a list of US states is shown with checkboxes for selection. The 'X Axis' is set to 'STATE_NAME'. On the right, a bar chart titled 'Incidence' displays the health impact for five selected states: Maine, Massachusetts, Michigan, Montana, and Nevada. The y-axis is labeled 'Health Impact' and ranges from 0 to 600. The x-axis is labeled 'Region'.

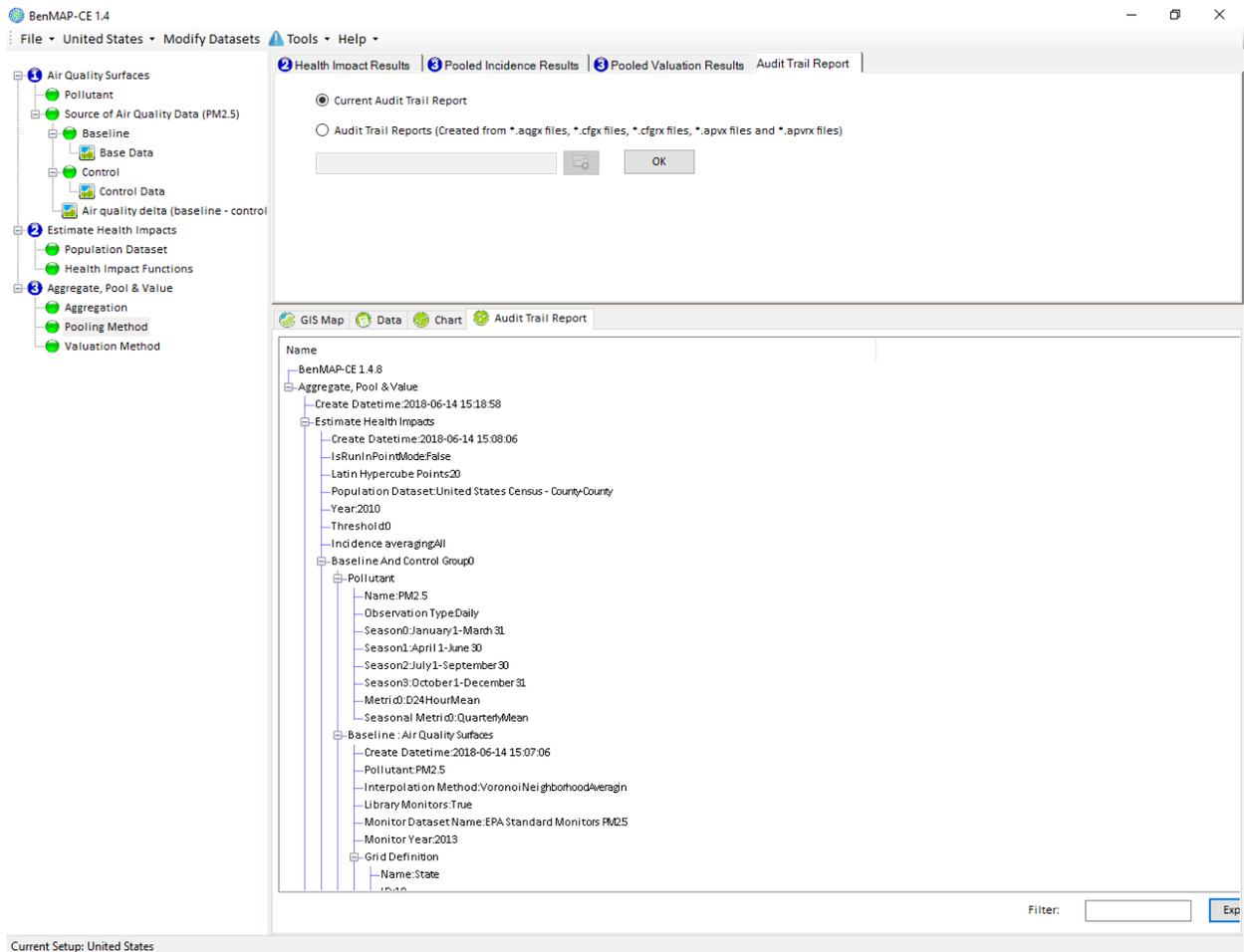
Region Name	Col	Row
<input checked="" type="checkbox"/> Maine	23	1
<input checked="" type="checkbox"/> Massach...	25	1
<input checked="" type="checkbox"/> Michigan	26	1
<input checked="" type="checkbox"/> Montana	30	1
<input checked="" type="checkbox"/> Nevada	32	1
<input type="checkbox"/> New Jers...	34	1
<input type="checkbox"/> New York	36	1
<input type="checkbox"/> North Ca...	37	1
<input type="checkbox"/> Ohio	39	1
<input type="checkbox"/> Pennsylv...	42	1
<input type="checkbox"/> Rhode Is...	44	1
<input type="checkbox"/> Tennes...	47	1
<input type="checkbox"/> Texas	48	1
<input type="checkbox"/> Utah	49	1
<input type="checkbox"/> Washing...	53	1
<input type="checkbox"/> Wisconsin	55	1
<input type="checkbox"/> Maryland	24	1
<input type="checkbox"/> Alabama	1	1
<input type="checkbox"/> Arizona	4	1
<input type="checkbox"/> Arkansas	5	1
<input type="checkbox"/> California	6	1
<input type="checkbox"/> Colorado	8	1
<input type="checkbox"/> Connecti...	9	1

You have the option of selecting the regions that you would like to compare using the list to the left of the chart by checking and unchecking certain regions. The chart automatically updates with each new selection.²⁶

8.3.4 Audit Trail Report

Audit Trail Reports facilitate transparency and reproducibility by reporting a summary of your assumptions underlying each of five types of files generated by BenMAP-CE: Air Quality Grids (with the *.aggx* extension), Incidence Configurations (with the *.cfgx* extension), Configuration Results (with the *.cfrx* extension), Aggregation, Pooling, and Valuation Configurations (with the *.apvx* extension), and Aggregation, Pooling, and Valuation Results (with the *.apvrx* extension).

²⁶ The data charts were originally developed using ZedGraph software, which is no longer supported. The BenMAP-CE development team is considering options to update and improve this feature.



Note that each successive step in an analysis contains a summary of its inputs and attributes, and those of each previous step in the analysis. For example, in the above report the attributes of the *Health Impact Function* file used to generate the APV Results are present in the *Estimate Health Impacts* node. Similarly, the metadata for both the baseline and control air quality grids are present under the *Estimate Health Impacts* node.

The process of creating an Audit Trail is described below:

- Click the **Audit Trail Report** tab in the results window. Select *Current Audit Trail Report* (this is the default setting). Click **OK**.
- Carefully review the report, ensuring that the air quality grids, population data, health incidence data, health impact functions and economic value estimates appear as you expected.

- Click the **Export** button to save the audit trail report. The default location for saving audit trail export (.txt, .ctlx, or .xml) files will be the location you chose earlier for saving your shape files.

Audit Trail Reports have three export options: .txt, .ctlx, and .xml. These file types can all be viewed using a standard text editor. The .txt and .xml files will contain the same information displayed in the **Audit Trail Report** window

BenMAP-CE Guidance and Best Practices

It is *always* recommended that you save your Audit Trail Report at the end of each analysis.

(however, the .xml file contains tags to retain the tree structure). If you are familiar with the command line feature, you may use the audit trail report to produce a control (.ctlx) file using an existing analysis, rather than creating one from scratch. The control file documents variables and configurations (file paths) associated with an analysis. For more information about the command line tool see Appendix L.

8.4 Frequently Asked Questions

Can I reorder the data layers?

Yes, data layers may be dragged within the GIS table of contents to reorder them within a group.

How do I export shapefiles?

When viewing any of the displayed maps in the **GIS Map** tab (lower right frame of the main window), click on the GIS toolbar icon for *Save Shapefile* (looks like a 3.5-inch diskette). Follow the prompts to provide a name and location for the file. BenMAP-CE will export a set of files (.dbf, .prj, .shp, .shx) associated with the shapefile that you can use with any GIS viewer.

How do I save a GIS map as an image?

To export the map as an image, click the *Export map* image icon (immediately below the *Save Shapefile* icon). This will use built-in DotSpatial GIS tools to allow you to save the map as a formatted image (.png) file. Alternately, use the *Print Screen* (PrtScn) button on your keyboard to create an image (saved in memory) which you can then paste into a graphics editor or document.

Can I display my map using a projection other than GCS-NAD 83 or Albers?

Yes, click the GIS toolbar icon for “**change projection to...**”; this feature will allow you to alternate between GCS-NAD83 and Albers projections. For more options, right-click on the **Map Layers** feature in the GIS table of contents and select **Projection** from the pop-up menu. This will display information about the current projection. Click the **Change Projection** button to view and apply other available projections.

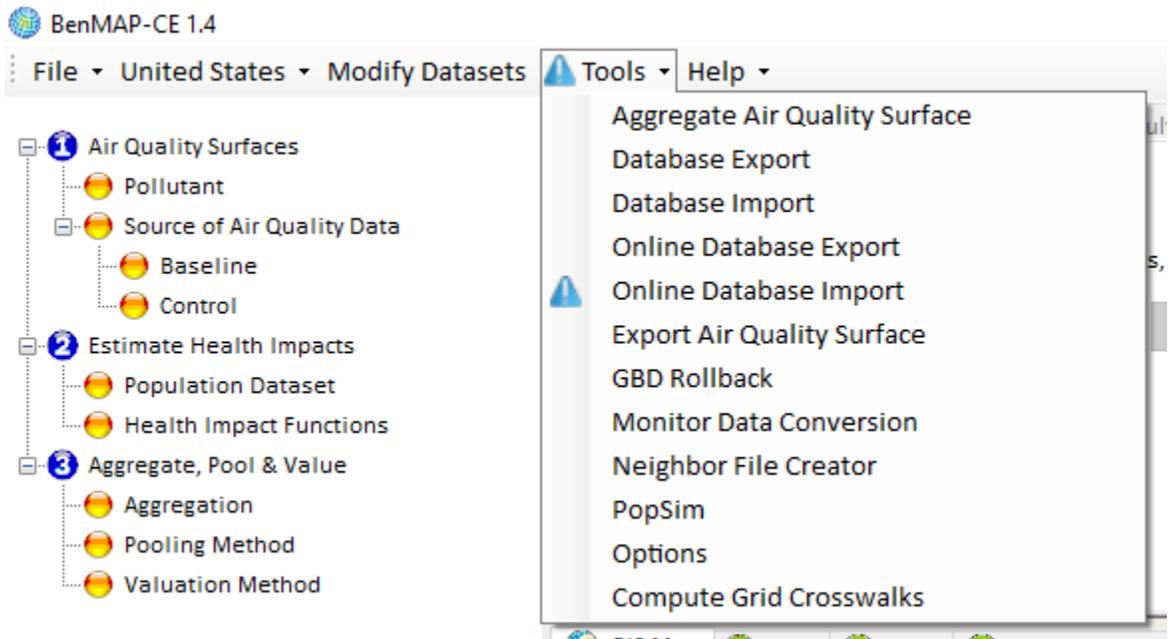
Chapter 9

Tools Menu

In this chapter...

- **Learn about the options in the Tools menu.**

The **Tools** menu, available on the main BenMAP-CE screen, provides access to six special add-on tools and an **Options** menu. Below we summarize the purpose of each tool. Note that other sections of the manual have already covered several of these items, so we merely list them here and point you to the appropriate section.



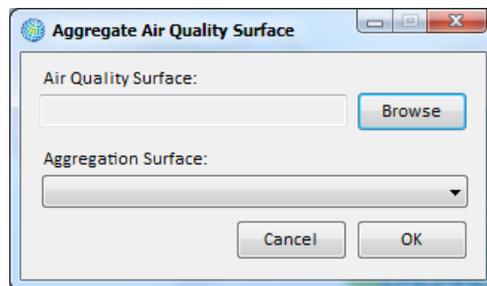
- **Aggregate Air Quality Surface.** Use this tool to change an air quality grid based on one grid definition to a coarser grid definition, using a simple spatially weighted average approach. For example—you could aggregate your air quality surface from 12km × 12km grids to U.S. Counties.
- **Database Export.** Export your entire database (every item in your setup) or parts of your setup (e.g. all GridDefinitions, or individual Health Impact Function Datasets) to a specified file location.
- **Database Import.** Import an entire setup or parts of an individual setup from a specified file location.
- **Online Database Export.** Export your BenMAP-CE dataset(s) and upload to a cloud-based data archive to share with the BenMAP-CE community.
- **Online Database Import.** Import dataset(s) provided by the BenMAP-CE community from a shared cloud-based data archive.
- **Export Air Quality Surface.** Generate a data file that contains all of the air quality values saved within an air quality surface (.aqgx) file.
- **GBD Rollback.** This application estimates the air pollution-attributable health burden, and the benefits of improved air quality, in each country using data from the Global Burden of Disease (GBD) study.

- **Monitor Data Conversion.** Convert monitor data files into BenMAP-CE format.
- **Neighbor File Creator.** This tool creates a text file (.txt) identifying "neighbor" monitors and associated interpolation weights for each grid cell in an air quality grid. Identifying the major contributors to air quality in a given cell can be helpful for understanding and troubleshooting BenMAP results, and for understanding how different geographic patterns of air quality changes can affect health benefits.
- **PopSim.** Simulate the cumulative effects of air pollution on different age groups over time (U.S. data only).
- **Options.** View and edit the general options for BenMAP-CE.
- **Compute Grid Crosswalks.** Remove or re-create crosswalks between grid definitions.

9.1 Air Quality Surface Aggregation

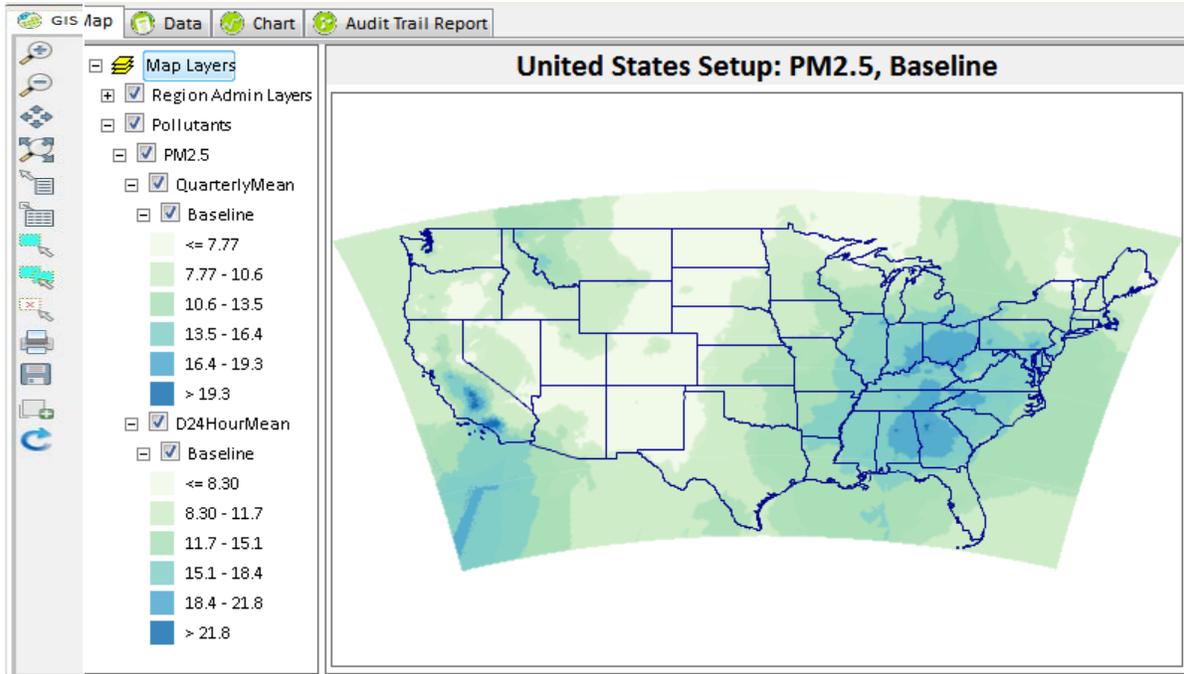
Using the **Aggregate Air Quality Surface** tool, you can change an air quality grid based on one grid definition to another (coarser) grid definition, using a simple spatially weighted average approach.

To start, choose *Aggregate Air Quality Surface* from the **Tools** drop-down menu. This will bring up the **Aggregate Air Quality Surface** window. Click the **Browse** button to find the air quality grid (.aqgx file) that you want to change and then use the **Aggregation Surface** drop-down list to select the new grid definition that you want to use. For example, you might want to aggregate a 12km model grid to the county-level. Click **OK** when done.

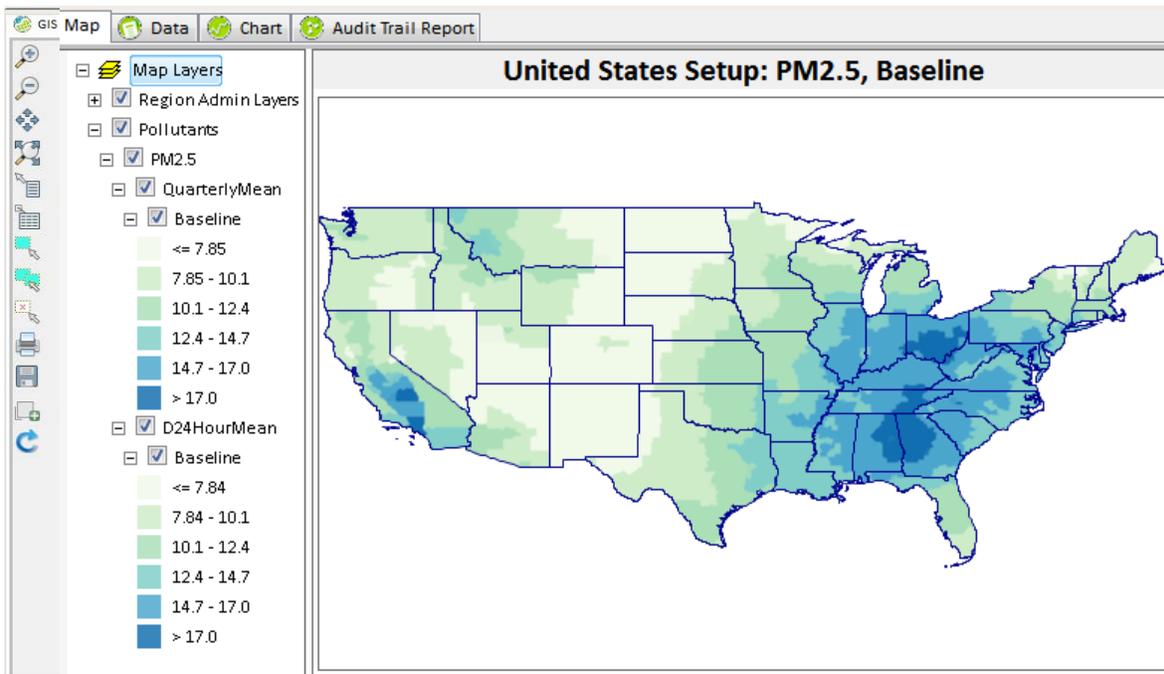


This will bring up the **Save Aggregated Air Quality Grid** window, where you specify the name of the newly aggregated air quality surface (.aqgx file) you are creating and its location. After the file is created, BenMAP-CE will return you to the main BenMAP-CE screen. You can then use the new file just as you would any other air quality surface.

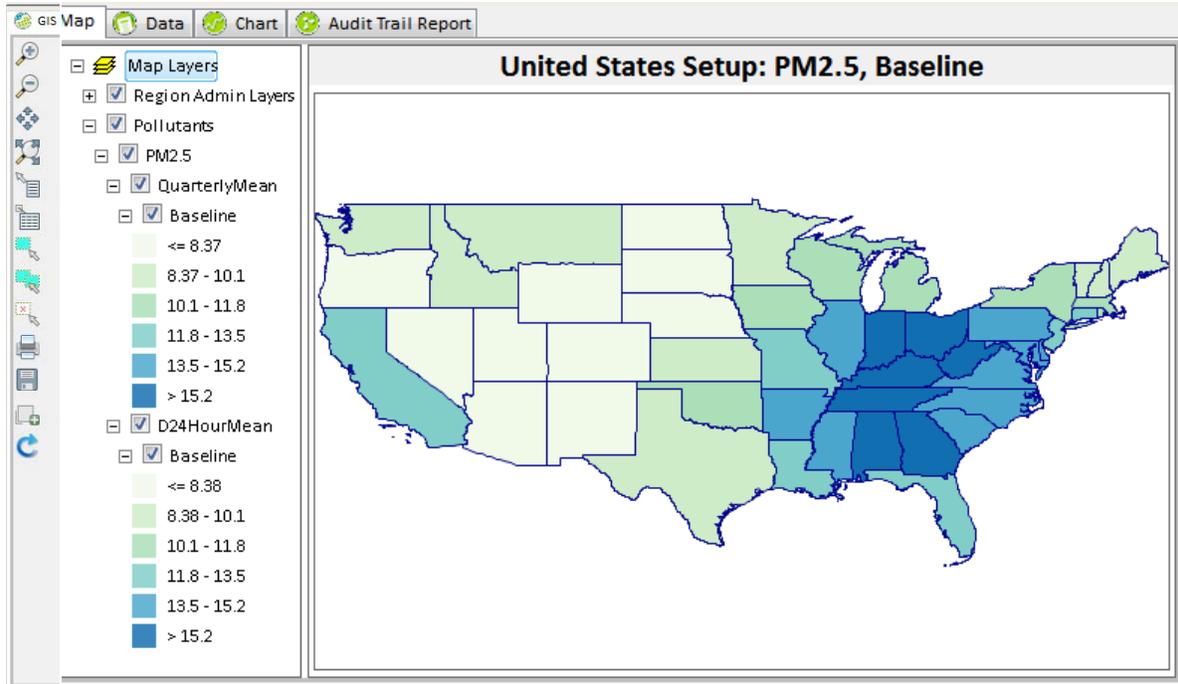
Below is an example of an air quality surface for PM_{2.5} created by the CMAQ model using a 12km grid. (The following images are cropped from the main BenMAP-CE window to focus on the GIS panel.)



Here is an example of the above 12km air quality surface aggregated to the county level.



And, here is an example of the 12km air quality surface aggregated to the state level.



Observe that there is more variation in the county-level file than the state-level file. This is expected, because BenMAP-CE is just using a simple spatially weighted average of the data.

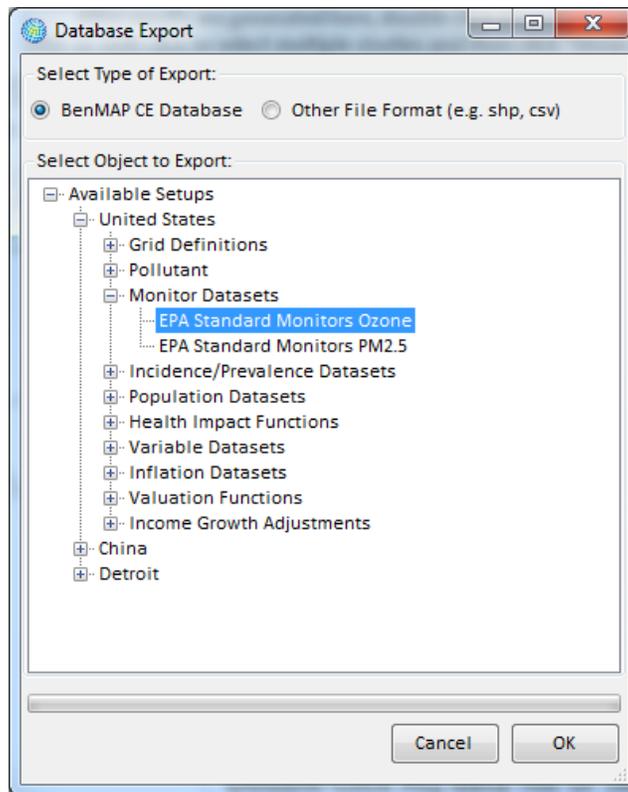
9.2 Database Export

BenMAP-CE allows you to export and import entire databases (all **Available Setups**), individual setups (e.g., *United States, China*), and parts of individual setups (e.g. all **Grid Definitions**, or individual **Health Impact Function** datasets). This functionality can be used to archive data, share data with other BenMAP-CE users, move databases between computers, as well as to view data in other applications like Excel or ArcMap. In particular, all of the steps involved in creating a setup can be done just once, after which the data can be exported and then imported on other computers. Version 1.5 also allows users to import databases exported from previous versions. This can be useful for transferring existing setups to the new version of BenMAP-CE without needing to re-recreate them.

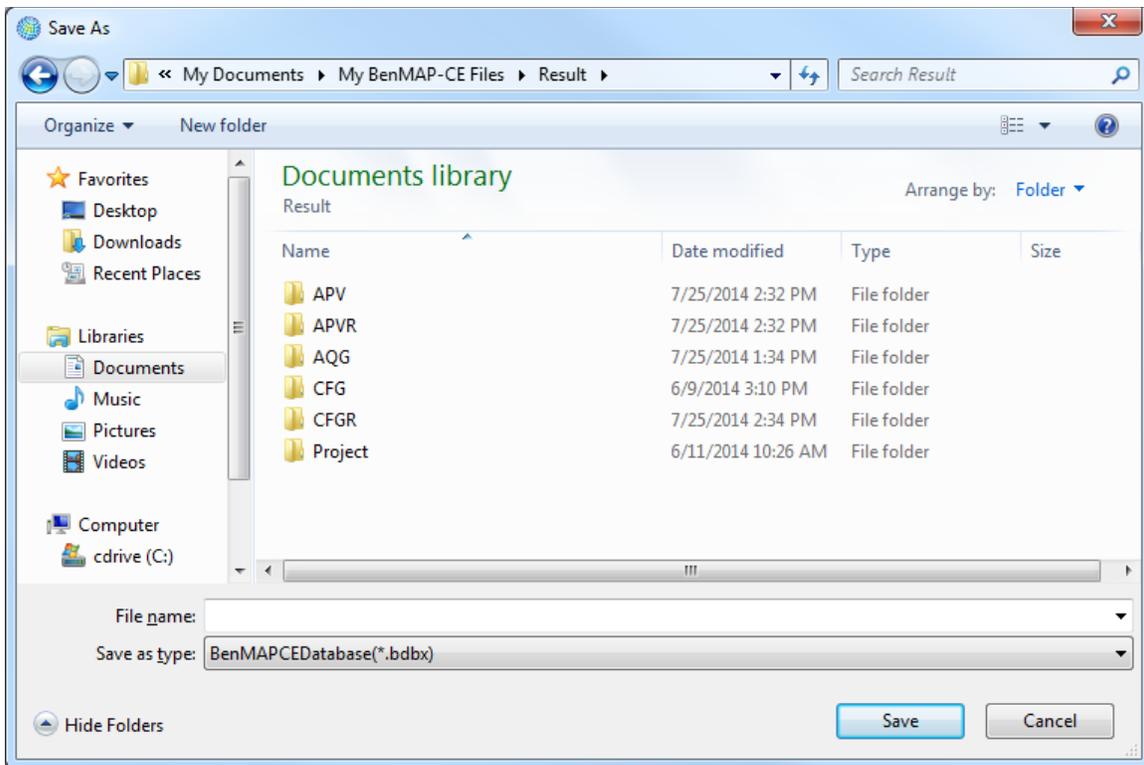
To export part or all of an existing setup, go to the **Tools** menu, and choose the *Database Export* option.

This will bring up the **Database Export** window. Initially, all of the setups are listed in a tree menu, which is initially in a collapsed view. To expand any of the menu items, click on the '+' sign to the left of menu item. This will expand the tree menu to show additional listings for the expanded item. To collapse the tree menu, simply click on the '-' sign.

Choose a dataset to export by selecting it from the tree menu. The default Type of Export is BenMAP CE database. Press **OK**. In the screenshot below, we have chosen to export *EPA Standard Monitors O3* to a BenMap-CE database file.

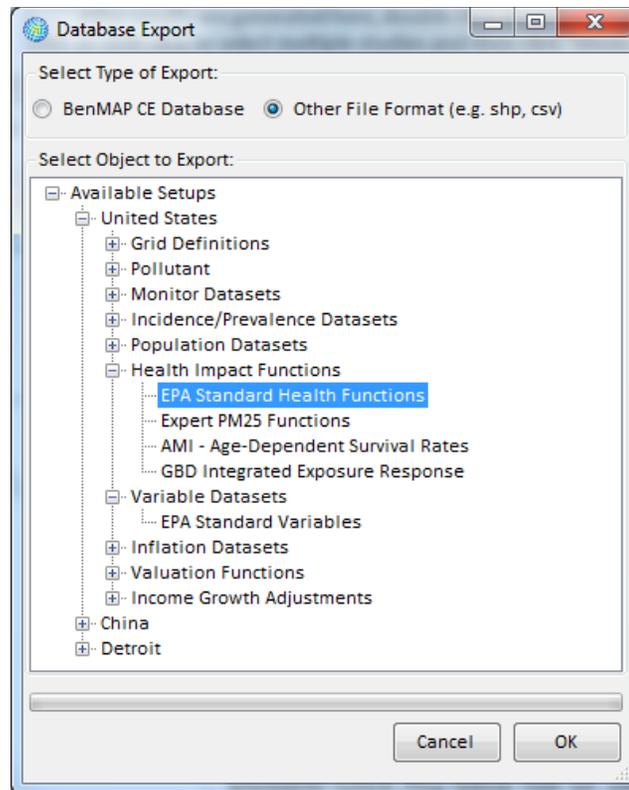


This will bring up the **Save As** window. From here you may name the export file, select the export file type and choose the save location.

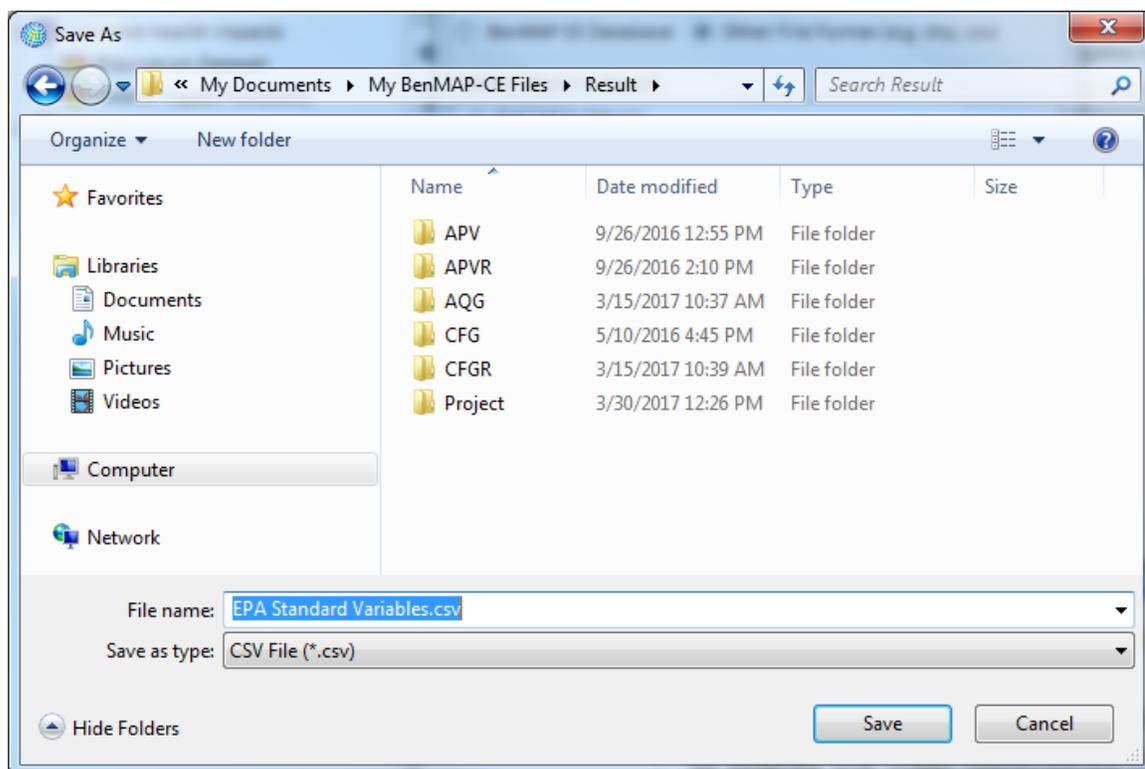


NOTE: Exported BenMAP-CE database files have a *.bdbx* extension, and are a binary format not suitable for viewing in external applications.

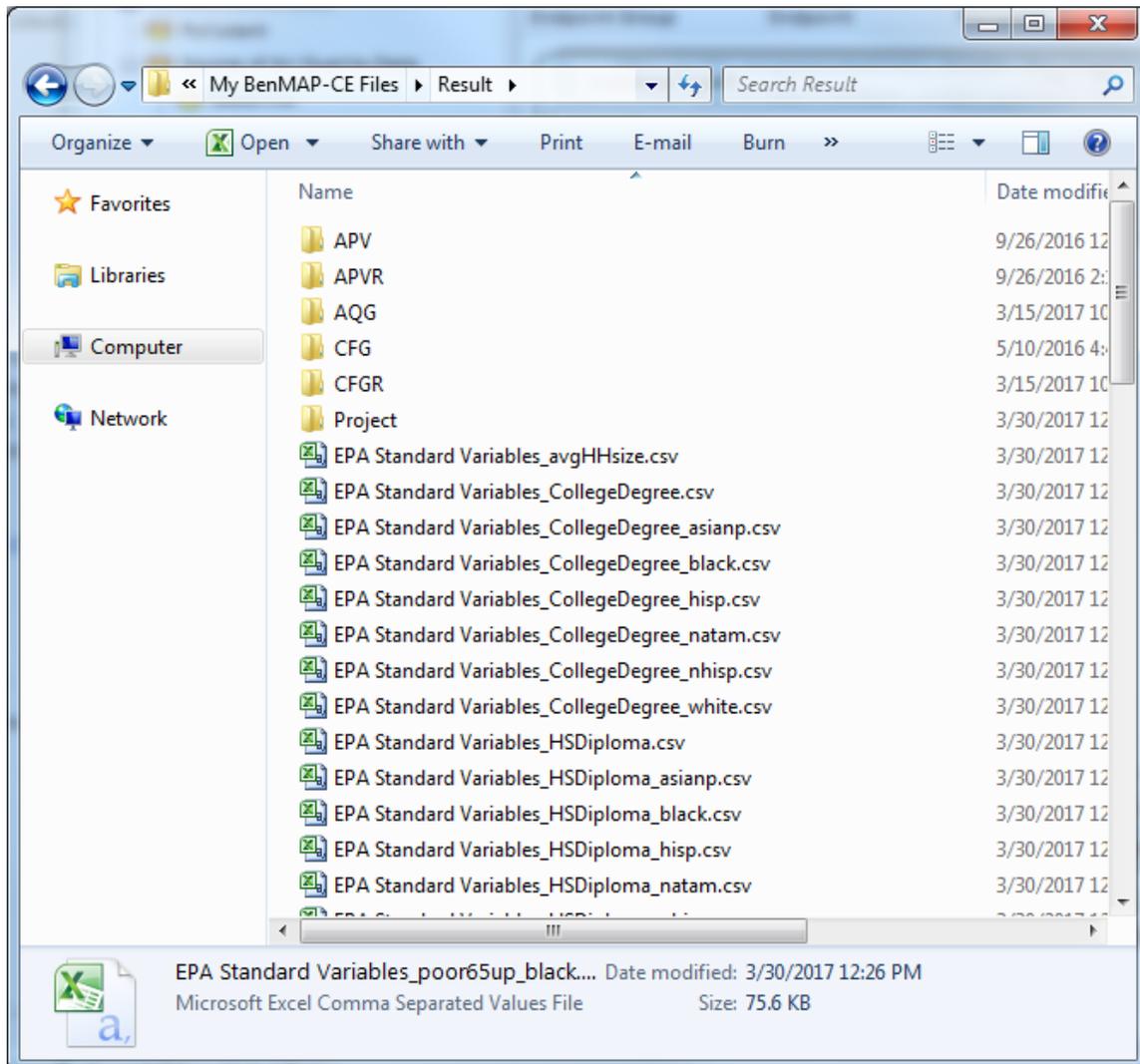
To view exported data in external application, choose “Other file Format”. The tool will export Grid definitions in shapefile (**.shp*) format and the rest of the data in CSV (**.csv*) format. If the dataset contains multiple tables, it will be exported into multiple CSV files. In the screen below, we have chosen to export *EPA Standard Variables* to a CSV file.



In the **Save As** window, the File name has been pre-populated with dataset name. You may leave it as is or change to something you want.



In this case, each variable of *EPA Standard Variables* dataset is saved as one CSV file with its variable name appended to the end of the dataset name.

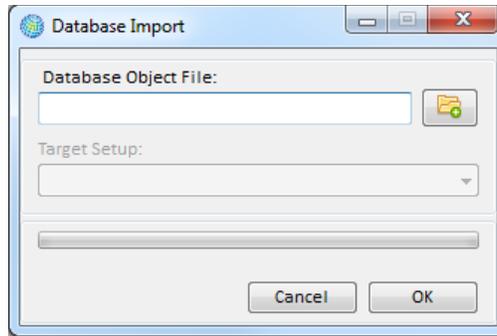


NOTE: When exporting datasets in Other File Format, only one dataset can be selected at one time.

9.3 Database Import

Import entire setups or parts of individual setups. This option is described in the Import Setups section of Chapter 4: Loading Data.

To import part or all of an existing setup, go to the **Tools** menu, and choose the *Database Import* option. This will bring up the **Database Import** window.



The **Database Object File** identifies the file that you want to import. Click on the **Browse** icon to locate the file. This will display the **Open** window.

Find and select the *.bdbx* file that you want to import, and then click **Open**. This will return you to the **Database Import** window. Click **OK** to finish the import process.

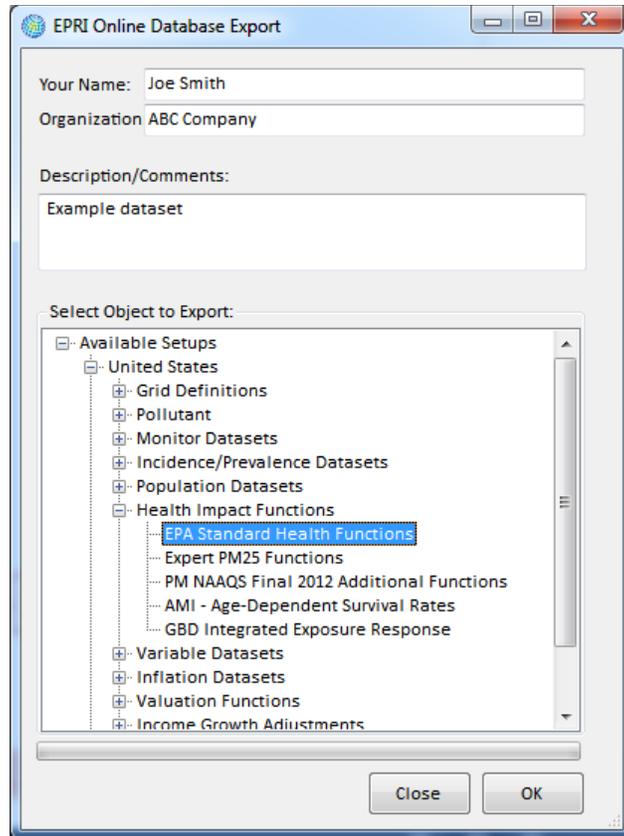
If the import file contains a subset of a setup, such as a collection (e.g., a set of Grid Definitions) or an individual dataset (e.g., a single grid definition from among many available), select the **Setup** into which it should be imported from the **Target Setup** drop-down list. Click **OK** to finish.

NOTE: Duplicates of datasets (typically identified by their names, e.g., ‘*Detroit Population*’) will default to the existing dataset in the Setup. New datasets (i.e., non-duplicated) will be added to the setup.

9.4 Online Database Export

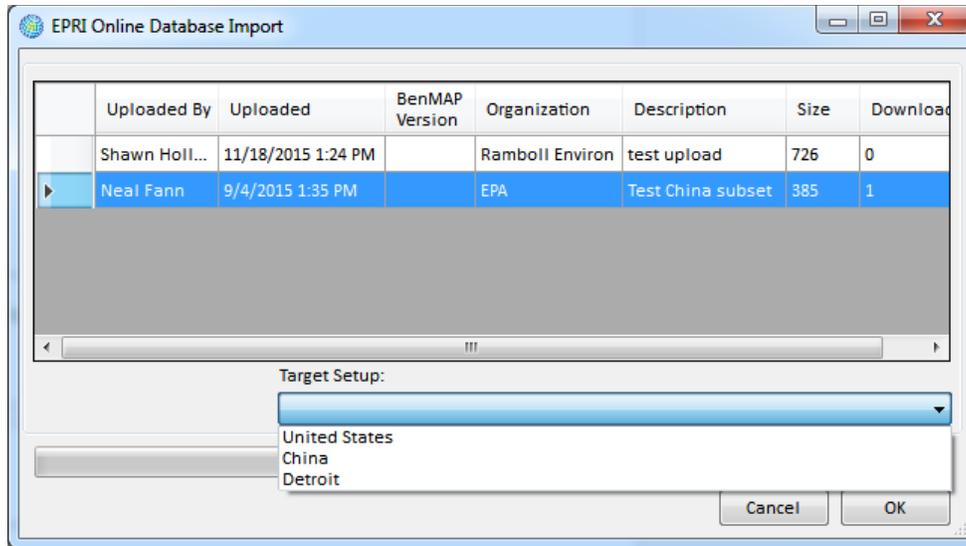
The **Online Database Export** feature was designed to facilitate sharing of BenMAP-CE datasets among the user community. The data is stored in an online database. Sharing and use of this data is at the discretion of the user community.

To share data in the online repository, select *Online Database Export* from the BenMAP-CE **Tools** menu. In the **EPRI Online Database Export** form, provide your name (required field), organization, and a description of the data you are sharing. Then select the object you wish to export from the tree menu. Similar to the database export feature (see Section 9.2), you can select an entire setup, dataset type, or specific data element. Once you make your selection and click “**OK**”, you will be prompted to confirm the upload.



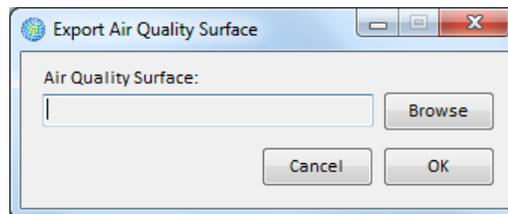
9.5 Online Database Import

The **Online Database Import** feature was designed to provide user access to an online repository of BenMAP-CE datasets shared by the user community. To import data from the online repository, select *Online Database Import* from the BenMAP-CE **Tools** menu. Select the data you wish to import by clicking on the record selector on the left side of the data grid. The record will be highlighted. Next choose your **Target Setup** from the dropdown list. Then, click the “**OK**” button. The progress bar near the bottom of the screen will update to show progress of the import. When complete, the system will notify you that the “*The database file was imported successfully.*”



9.6 Export Air Quality Surface

The **Export Air Quality Surface** tool generates a data file (.csv) reporting all of the data contained in the air quality grid. After choosing *Export Air Quality Surface* from the **Tools** menu, the **Export Air Quality Surface** window will appear. Click the **Browse** button to choose the air quality grid that you want to examine.



Click **OK** after you have selected your file. Use the **Save As** window to choose the directory where you want to save your file. And in the **File name** box, type in the name of the file.

To help keep track of what you are doing, you might want to use the same file name as your air quality grid, or something very similar. (If you use the same name, you can always distinguish the two files by the extension. An air quality grid has an .*aqg*x extension and the file you are generating here has a .*csv* extension.)

When done, click the **Save** button. You can view the files you have created with any database viewer. For each **Metric** and **Seasonal Metric**, you can see the actual values. In addition, you can see the **Statistics** calculated for each. In the example below for a 12km PM_{2.5} air quality surface, you can see in the first grid cell (Column = 1, Row = 246) that the mean of the *D24HourMean* values is 9.15 and the mean of the *QuarterlyMean* values is 9.2.

Col	Row	D24HourMean	QuarterlyMean
1	246	9.15	9.2
2	246	9.16	9.2
3	246	9.16	9.21
4	246	9.17	9.21
5	246	9.17	9.22
6	246	9.54	9.65

Note that the exported files may be very large (tens to hundreds of megabytes in file size and with row counts exceeding typical spreadsheet applications). With large files, you might need to use a database program to work with the files. Alternatively, these files can also be read by simple text editors.

9.7 GBD Rollback

The World Health Organization global burden of disease (GBD) study measures burden of disease using the disability-adjusted-life-year (DALY). This time-based measure combines years of life lost due to premature mortality and years of life lost due to time lived in states of less than full health. The DALY metric was developed in the original GBD 1990 study to assess the burden of disease consistently across diseases, risk factors and regions.²⁷

The **GBD Rollback** tool uses data from the 2013 GBD study to allow users to estimate the human health burden of PM_{2.5} levels in each country as well as the benefits of reducing these air pollution levels. Users can “roll back,” or adjust ambient PM_{2.5} levels in one or more countries or regions and calculate the total burden, or avoided deaths, in that region. The tool also estimates avoided life years lost, changes in life expectancy, and the economic benefits associated with avoided deaths.

This feature is analogous to the monitor roll-back tool already available in core BenMAP-CE (discussed in Chapter 5); that tool allows users to adjust downward (or upward) air quality monitoring data in specified locations according to various algorithms (including proportional, quadratic and incremental rollbacks). The tool uses a grid with 0.1 degree resolution (approximately 10km) grid cells and includes:

- 2015 PM_{2.5} pollution concentrations²⁸. Negative concentrations were adjusted to zero.

²⁷ For more information on the GBD, see: http://www.who.int/topics/global_burden_of_disease.

²⁸ Cohen, A. J.; Brauer, M.; Burnett, R. T. (2017). Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *Lancet*, 389(10082), 1907-18.

- 2015 global population²⁹ data stratified by age and gender. Elder populations were combined into an "80UP" age group to align with the incidence dataset.
- 2013 mortality incidence³⁰ for six health endpoints (COPD, cerebrovascular disease, ischemic heart disease, lung cancer, acute lower respiratory infection, and non-accidental) stratified by age and gender. Neonatal ("0 to 0") and "1 to 4" age groups were combined into a "0 to 4" age group to align with population data.
- The 2013 Integrated Exposure Response (IER) function³¹ employed by the 2013 GBD study to estimate premature mortality associated with ambient air pollution. The 2013 IER function estimates premature mortality from COPD, cerebrovascular disease, ischemic heart disease, and lung cancer.
- The Shape-Constrained Health Impact Function (SCHIF) developed by Burnett et al. (in preparation)³². Like the IER, the SCHIF is a meta-analytic concentration-response function developed using data from many PM_{2.5}-related epidemiological studies. Depending on the country being evaluated, the SCHIF estimates draw from mortality incidence from either (a) all non-accidental causes, or (b) a "re-attributed" incidence rate representing deaths due to COPD, cerebrovascular disease, ischemic heart disease, lung cancer, and acute lower respiratory infection).

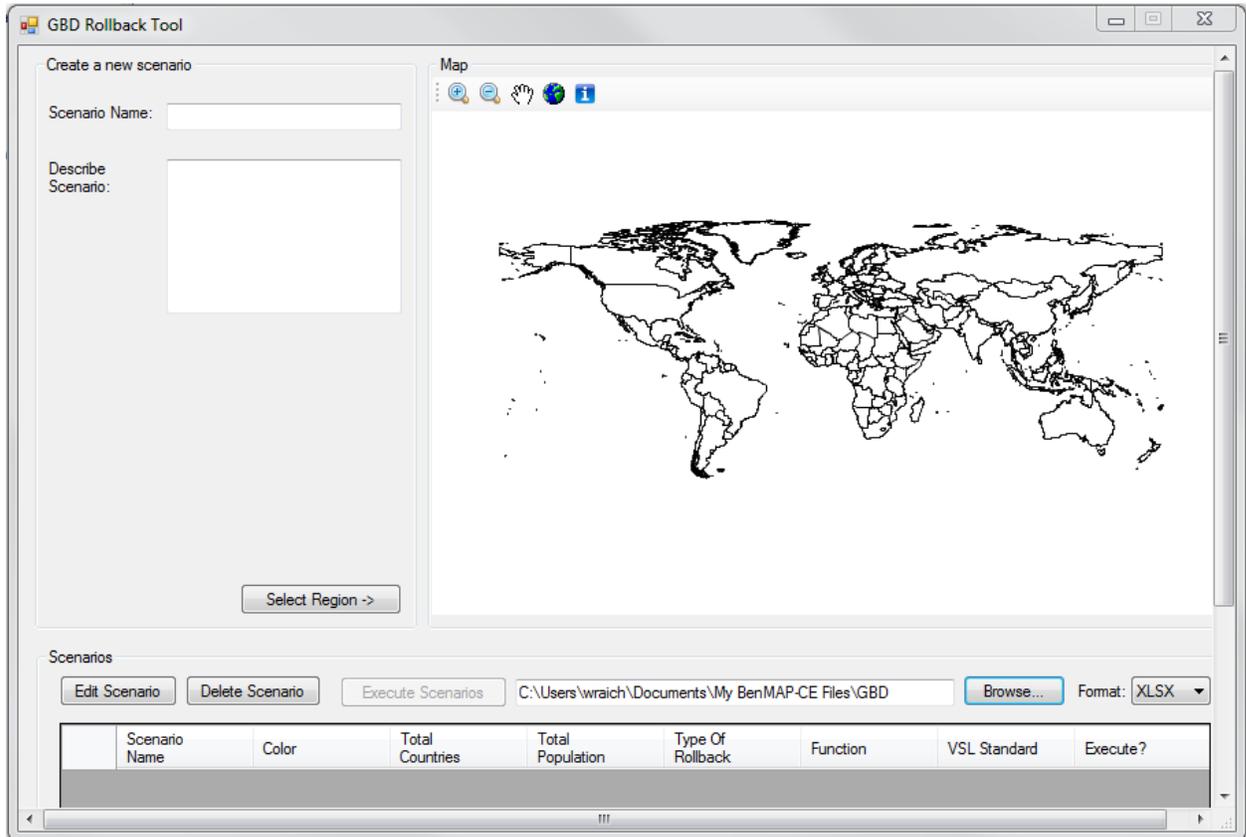
²⁹ Center for International Earth Science Information Network - CIESIN - Columbia University. 2016. Gridded Population of the World, Version 4 (GPWv4): Administrative Unit Center Points with Population Estimates. Palisades, NY: NASA Socioeconomic Data and Applications Center (SEDAC). <http://dx.doi.org/10.7927/H4F47M2C>.

³⁰ Global Burden of Disease Study 2013. Global Burden of Disease Study 2013 (GBD 2013) Incidence, Prevalence, and Years Lived with Disability 1990-2013. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2015. <http://ghdx.healthdata.org/record/global-burden-disease-study-2013-gbd-2013-incidence-prevalence-and-years-lived-disability>

³¹ Burnett RT, Pope CA, Ezzati M, Olives C, Lim SS, Mehta S, Shin HH, Singh G, Hubbell B, Brauer M, Anderson HR, Smith KR, Balmes JR, Bruce NG, Kan H, Laden F, Pruss-Ustun A, Turner MC, Gapstur SM, Diver WR, Cohen A. (2014). An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environ Health Perspect* 122:397-403; <http://dx.doi.org/10.1289/ehp.1307049>

³² Burnett RT, Chen H, Szyszkowicz M, Fann N, Hubbell B, Pope CA, Apte JS, Brauer M, Cohen A, Weichenthal S, Coggins J, Di Q, Brunekreef B, Frostad J, Lim SS, Kan H, Pruss-Ustun AM, AARP collaborators, ACS collaborators, CTS collaborators, Canadian Breast Screening collaborators, CanCHEC/CCHS collaborators, Chinese Male Cohort Collaborators, CUELS collaborators, English Cohort collaborators, Hong Kong collaborators, NHIS collaborators, NHS collaborators, Rome Census Cohort collaborators, VHM&PP collaborators. 2017. A new approach to estimating global mortality burden from outdoor fine particle exposure. Manuscript in preparation.

To begin, select *GBD Rollback* from the **Tools** menu. The **GBD Rollback Tool** window will be displayed.



9.7.1 Create New Scenario

To create a new scenario, first provide a **Scenario Name** (required) and description for the scenario (optional). Then, click the **Select Region** button.

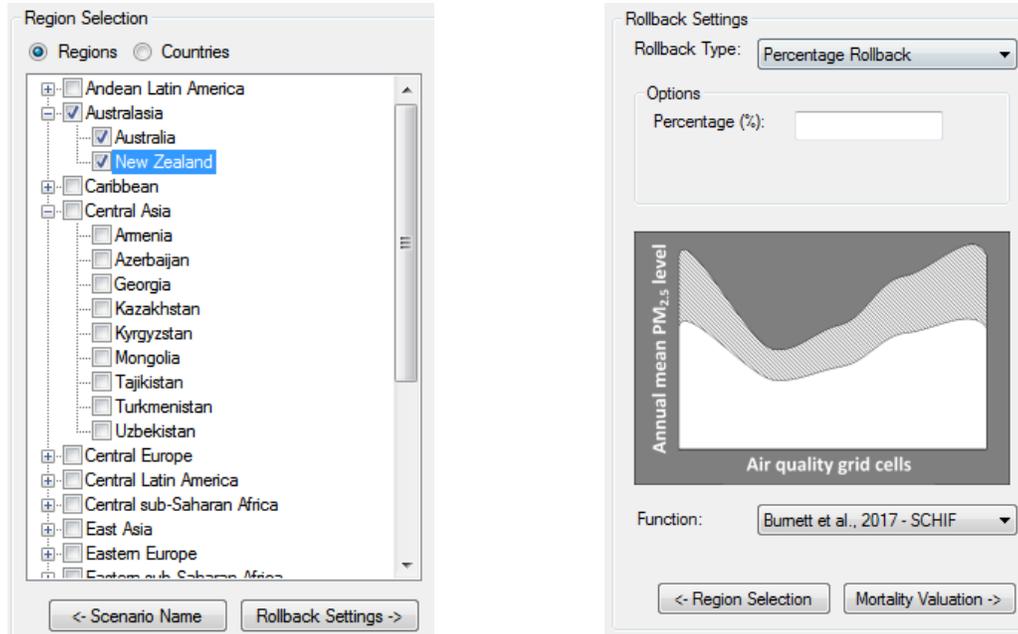
9.7.2 Select Regions or Countries

The **Region Selection** box provides options to view *Regions* (as defined in the GBD study) with the associated countries listed beneath, or to view *Countries*. Multiple selections are possible with either selection method and you can toggle between the two.

If you view *Regions*, you can select an entire region (all countries within this region) by checking the box next to the desired region. To view the individual countries for a region, click on the '+' sign to the left of the region. This will expand the tree menu. To collapse the tree menu, simply click on the '-' sign.

If you do not know the name of the region which contains the country you want to select, click the option to view *Countries*. You can search the alphabetically-sorted list to find countries.

As you make selections, the map will highlight them in a bright blue color. Note that the map is not interactive (i.e., you cannot click on the map to make selections). The toolbar located above the map allows you to zoom in and out, pan, view full extent, and identify countries.



9.7.3 Choose Rollback Settings

After selecting the countries or regions to analyze, click the **Rollback Settings** button. The **Rollback Settings** pane allows you to choose the **Rollback Type** (*Percentage Rollback*, *Incremental Rollback*, or *Rollback to a Standard*) and configuration options.³³ (An illustrative example for the selected **Rollback Type** will be displayed below the *Options* box.) The tool allows for a negative rollback, indicating an increase in pollution concentration.

For *Percentage* and *Incremental Rollback* types, you must enter a percent value or whole number by which to reduce the pollutant concentration in all grid cells in the

Fundamental Concept - Rollback

A **rollback** is a simplified type of BenMAP analysis in which all monitor data are reduced under the Control scenario using a uniform rule. BenMAP-CE includes three rollback options for monitor data: **Percentage rollback** reduces all monitor observations by the same percentage. **Incremental rollback** reduces all observations by the same increment. **Rollback to a standard** reduces all monitor observations as necessary so that they all meet a specified standard.

³³ The tool uses a background PM_{2.5} concentration = 5.8 µg/m³ (lowest measured level in epidemiological literature).

selected countries. If you select *Rollback to a Standard*, you may select from a list of national pollutant standards enforced by various countries in different years³⁴.

Standard Group	Concentration Limit ($\mu\text{g}/\text{m}^3$)	Exposure Duration
WHO	10	Annual
US Primary	12	Annual
US Secondary	15	Annual
EU	25	Annual
Japan	15	Annual
China Class I	15	Annual
China Class II	35	Annual
Hong Kong	35	Annual
India	40	Annual
Singapore	12	Annual
WHO	25	24-hour
US Primary	35	24-hour
Japan	35	24-hour
China Class I	35	24-hour
China Class II	75	24-hour
Hong Kong	75	24-hour
India	60	24-hour
Singapore	37.5	24-hour

The **Function** dropdown allows users to select either the SCHIF (Burnett et al., 2017) or the IER function used in the 2013 GBD studies (Burnett et al., 2014).

9.7.4 Choose Mortality Valuation Settings

Once you have completed the Rollback Settings window, click the **Mortality Valuation** button. The **Mortality Valuation Settings** pane allows you to specify how you wish to calculate the economic benefits associated with avoided premature deaths.

Estimated reductions in premature mortality are valued using country-specific estimates of the value per statistical life (VSL). This value is not the monetary value of individual lives. Rather, it reflects the amount individuals are willing to pay to incrementally reduce their risks of death from adverse health conditions that may be caused by environmental pollution. For example, if each individual in a population

Fundamental Concept - VSL

The **Value of Statistical Life (VSL)** is the monetary value derived from economic studies of the amount of money individuals are willing to exchange for small reductions in their annual mortality risk, integrated over a whole population. It does NOT reflect the value of the life of any specific individual.

³⁴ Air Quality Standards / Guidelines / Objectives For Different Countries (updated April 2014). Table developed by Scott Voorhees, US EPA.

is willing to pay \$20 to reduce his or her risk of death by 10 in 100,000, the VSL for that population would be \$200,000 ($= \$20 \div (10/100,000)$). For 500 avoided deaths in that population, the economic benefit would be \$100,000,000 ($= 500 * \$200,000$).

The **VSL Standard** dropdown menu allows users to select the VSL estimate applied to mortality risk reductions. Because VSL estimates from primary research are not available in most countries, it is necessary to transfer international estimates across countries, adjusting for differences in income levels.³⁵ Users may select one of two base VSL estimates generously provided by the authors of World Bank (2016).³⁶ VSL estimates were derived by the authors using a benefit-transfer approach, with a base VSL of \$3.83 million (2011 USD) as recommended by a meta-analysis of willingness-to-pay (WTP) studies by the Organisation for Economic Co-operation and Development (OECD). The base VSL is transferred to other countries and years using the following formula:

$$VSL_{c,n} = VSL_{OECD} * \left(\frac{Y_{c,n}}{Y_{OECD}} \right)^e$$

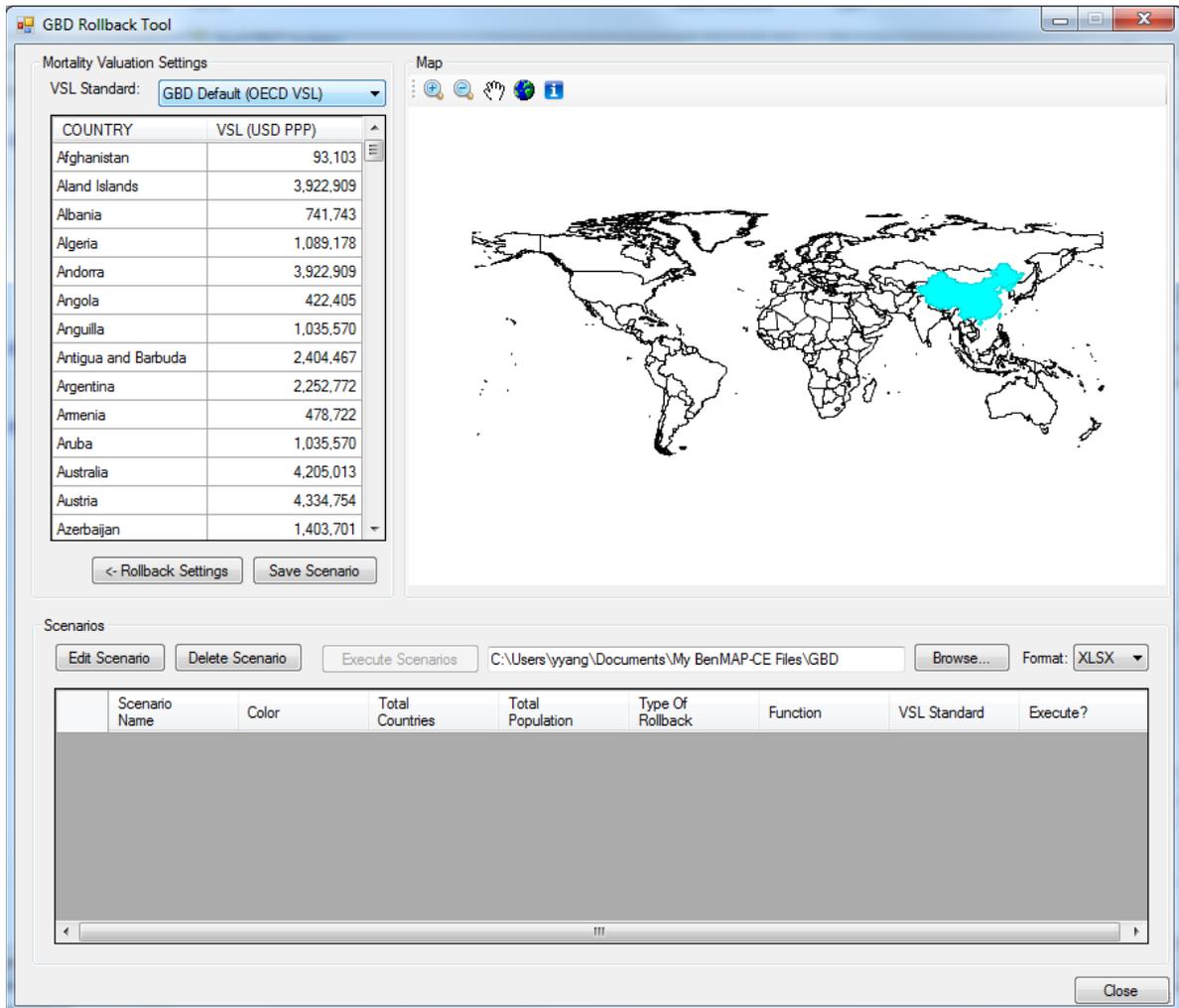
where $VSL_{c,n}$ is the VSL for country c in year n ; VSL_{OECD} is the base OECD VSL; $Y_{c,n}$ is the GDP per capita for country c in year n ; Y_{OECD} is the average GDP per capita for the sample of OECD countries (roughly \$37,000); and e is the income elasticity of the VSL. In all instances, monetary values are adjusted for price inflation and converted to 2011 U.S. dollars at purchasing power parity (PPP) rates. While the authors provide VSL estimates for a range of income elasticity (e) values, we present their central results, which assume an elasticity of 1.2 for low- and middle-income countries, and 0.8 for high-income countries. This benefits transfer approach is also used with a base VSL estimate from USEPA of \$8.7 million (2011 USD).

Additionally, we use country-level VSL estimates to derive population-weighted average VSLs by GBD regions. These VSLs allow GBD Tool users to value reduced mortality at the regional level. The regional VSLs are also used to impute missing country-level VSLs for 29 of the 210 countries in the GBD tool. The “Metadata” tab in the GBD Tool Output spreadsheet displays VSL estimates by country and region.

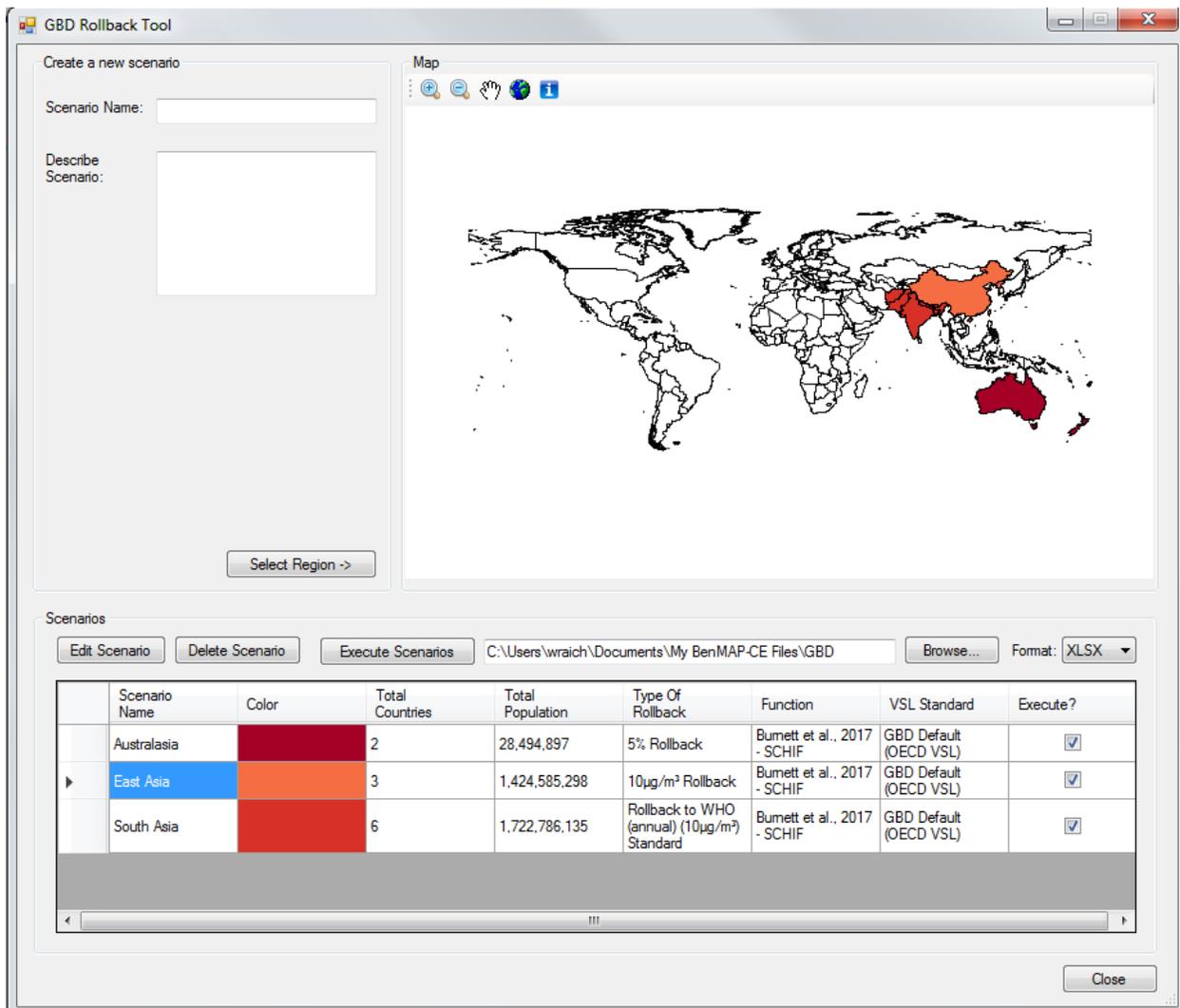
The left pane of the **Mortality Valuation Settings** window provides a list of countries with their respective VSL estimates. Once you have selected the base VSL estimate (OECD or USEPA), click the **Save Scenario** button to save your rollback configuration. The map will update the color of the selected countries so that each saved scenario is uniquely different.

³⁵ Mortality risk reductions have been shown to be sensitive to income; individuals are willing to pay more for risk reductions as their income increases. Thus, the VSL is commonly adjusted to reflect differences in the average income levels both across countries and within a country over time.

³⁶ World Bank and IHME (2016). *The Cost of Air Pollution: Strengthening the Economic Case for Action*. Washington, DC: 2016 International Bank for Reconstruction and Development/The World Bank.



The table at the bottom of the window will update to display the attributes for the saved scenario (*Scenario Name, Color, Total Countries, Total Population, Type of Rollback, etc.*), along with an *Execute?* option for you to indicate whether the tool should execute or ignore the scenario. If you double-click in the *Total Countries* or *Total Population* fields, the program will display a table of the individual countries and populations included in the scenario.



9.7.5 Execute Scenarios and Save Results

Click the **Execute Scenarios** button to generate results for saved scenarios. If a selected country does not have sufficient data (i.e., population, pollution concentration, or incidence rates) to perform the analysis, it will be bypassed and a message (including the country name) will be provided.

If you are adding to a list of saved scenarios and do not want to re-run the previously executed ones, uncheck *Execute?* in the summary table so that only the new scenario is checked.

Upon execution, the **GBD Rollback** tool will export results in (.xlsx format) to a default file location.³⁷ You can change the file location by clicking the **Browse** button. You can

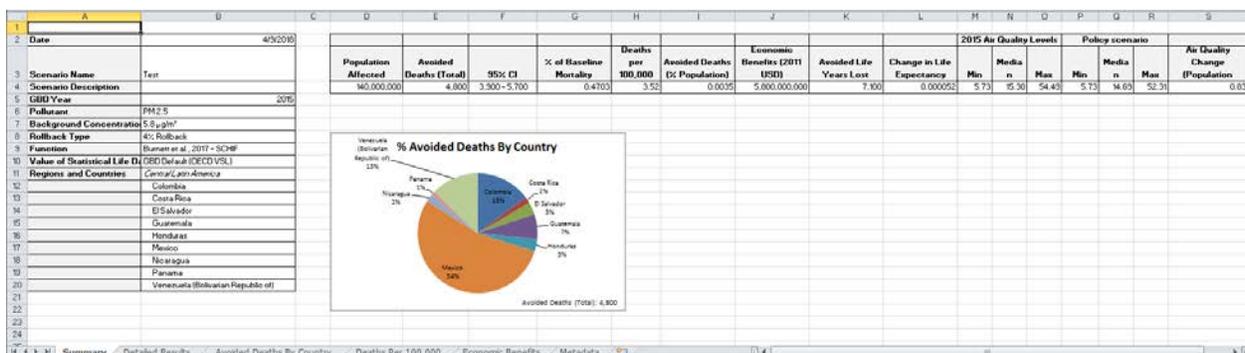
³⁷ The files are stored by default at the following location: C:\Users\\Documents\My BenMAP-CE Files\GBD.

also change the export file format to *CSV* by clicking on the **Format** drop-down list. The results file is named using the *Scenario Name* followed by the time stamp of scenario execution.

- The *XLSX* option provides a formatted summary table, detailed results, charts, and metadata about the supporting datasets used in this analysis.
- The *CSV* option will only create two files: one containing the summary data and one with detailed results.

If you want to edit or delete saved scenarios, select one record at a time from the scenario table (use the record selector on the left side of the grid) and click the **Edit Scenario** or **Delete Scenario** button. If a scenario is edited and re-executed, the timestamp in the filename will help the user identify the new version.

Once you close the **GBD Rollback** tool, the scenarios are cleared from memory; scenarios are not saved in the BenMAP-CE database. Information about the scenario configurations are saved in the results file to help you document the analysis and re-create it if necessary.



The GBD results file (.xlsx format) contains six worksheet tabs, described as follows:

- **Summary:** Gives a basic background on the scenario chosen, including name, description, pollutants, rollback type, health impact function, VSL selection, and countries. It also provides a quick overview of total and affected population results.
- **Detailed Results:** Gives in-depth breakdown of population and results for each selected country. Important fields on this tab include *Population Affected*, *Avoided Deaths (Total)*, *Economic Benefits*, *Avoided Life Years Lost*, and *Change in Life Expectancy*.
- **Avoided Deaths by Country:** Displays the number of avoided deaths for the region and by country in a bar graph. This graph was generated from the data on the **Detailed Results** tab.

- **Deaths Per 100,000:** Displays the number of deaths for each country (with a population multiplier of 100,000) in a bar graph. This graph was generated from the data on the **Detailed Results** tab.
- **Economic Benefits:** Displays the economic benefits associated with avoided premature mortality for the region and each country in a bar graph. This graph was generated from the data on the **Detailed Results** tab.
- **Metadata:** Provides supplementary information about the underlying data and functions used by the **GBD Rollback** tool.

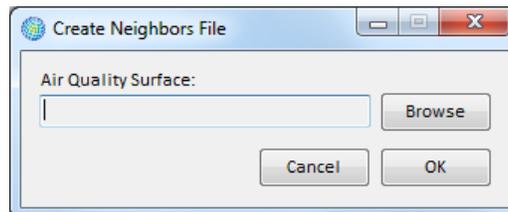
9.8 Monitor Data Conversion

The **Monitor Data Conversion Tool** provides the user with a mechanism to transform their data from commonly available formats (e.g., one monitoring result per row) to BenMAP-ready format. The tool is currently under development for inclusion in a future version of BenMAP-CE.

9.9 Neighbor File Creator

The **Neighbor File Creator** tool generates a file containing gridded monitor data created by BenMAP-CE for *Monitor Data* or *Monitor Rollback* air quality surfaces.

To start, choose *Neighbor File Creator* from the **Tools** drop-down menu. This will bring up the **Create Neighbors File** window. Click on the **Browse** button, and find the air quality surface you want to analyze.



After locating the file, click the **OK** button. The file path for the selected file will be displayed in the **Air Quality Surface** box. Click **OK**. A **Save As** window will open. Provide a file name for the **Neighbors file (.csv)** you want to create and click **Save**. An example Neighbors File is shown below.

Col	Row	MonitorName	Weight	Distance
1	246	410030013881011	0.215167	627.5258
1	246	530410006881011	0.253047	533.5881
1	246	530090009881011	0.286508	471.2713
2	246	410030013881011	0.213318	620.813
2	246	530410006881011	0.252881	523.688
2	246	530090009881011	0.287978	459.8632
3	246	410030013881011	0.211387	614.2613
3	246	530410006881011	0.252682	513.8765
3	246	530090009881011	0.289525	448.4836

The first two columns specify the *Column* and *Row* variables for each grid cell. In the example above, you will see that Column = 1 and Row = 246 are repeated three times, indicating that three different monitors were used to estimate air quality at this grid cell. The *MonitorName* column provides the monitor identifier. The *Weight* column specifies the weight used in the air quality calculation (e.g., *Voronoi Neighbor Averaging*). And the *Distance* column gives the distance (in kilometers) from the monitor location to the center of the grid cell.

Note that if an air quality grid was created using the *Closest Monitor* option (see Chapter 5 under the Monitor Data section), then only a single monitor is used for any given grid cell. As a result, the neighbor file will contain the same 5 fields, but the *Weight* column will contain a value of “1” all the way down. In addition, there will only be one entry for each grid cell.

Col	Row	MonitorName	Weight	Distance
1	246	410030013881011	1	627.5258
2	246	410030013881011	1	620.813
3	246	410030013881011	1	614.2613
4	246	410030013881011	1	607.8755
5	246	410030013881011	1	601.661
6	246	410671003881011	1	548.5376
7	246	410671003881011	1	541.038
8	246	410671003881011	1	533.702

9.10 PopSim

The Population Simulation (PopSim)³⁸ model was designed to estimate two outputs related to premature mortality attributed to the CAAA: life-years lost, and changes in life expectancy. The population simulation approach provides some advantages over the BenMAP-CE model in terms of simulation of the dynamic effects of mortality across a population through time, but also has several significant disadvantages relative to BenMAP-CE in terms of the spatial resolution of pollutant exposure estimates. As a result, the population simulation approach operates as a supplement to the BenMAP-based primary estimates for selected measures of the impact of reducing risks of premature mortality.

The core BenMAP-CE 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. Conversely, the PopSim tool estimates the change in population mortality risk over a multi-year period, but it is not yet possible to estimate the economic value of these impacts in BenMAP-CE.

PopSim is designed to track the effect of alternative assumptions about the mortality effects of fine particulate matter (PM_{2.5}) air pollution in the U.S. population over time. The tool incorporates detailed life table data for historical years, by age, gender, and cause of death, obtained from the Census Bureau and the Centers for Disease Control (CDC). It also incorporates Census mortality and population projections for future years, again by age and gender, using the projected death and birth rates that underlie the Census Bureau's published population projections.

The PopSim model allows users to:

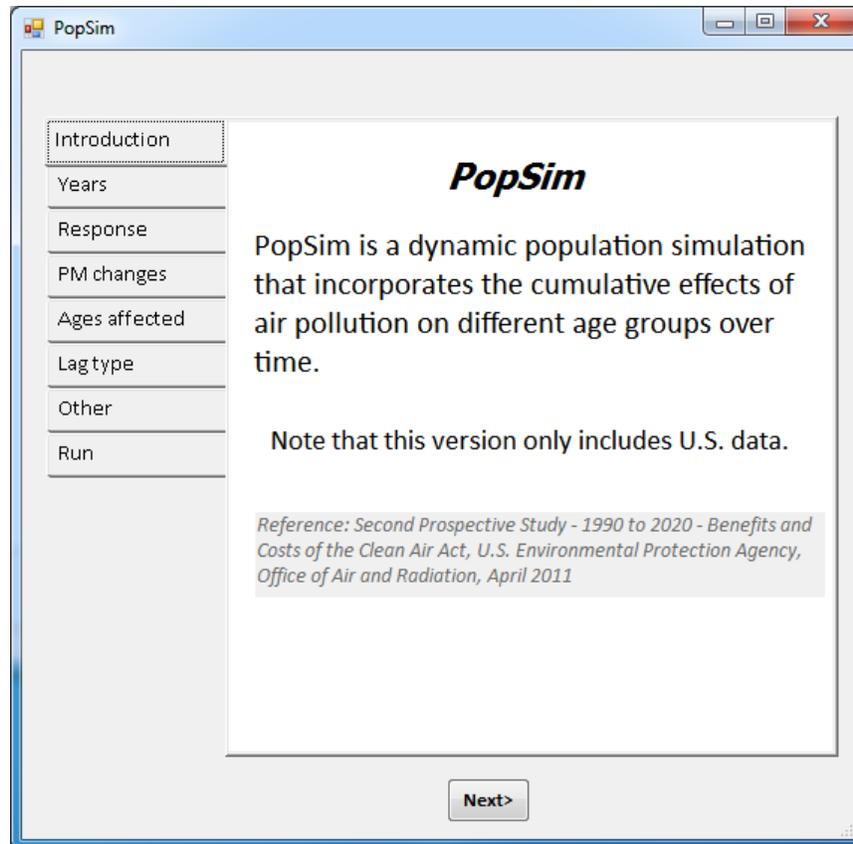
- Simulate population in the U.S. by single year cohorts of age and gender for years between 1980 and 2050 under alternative assumptions about the degree of hazard posed by air pollution relative to baseline historical and projected Census mortality rates;
- Estimate changes in life years relative to baseline Census mortality rates;
- Apply air pollution hazards differentially by cause of death; and
- Analyze the effect of alternative cessation lag structures on the timing of total mortality and on total life years in the U.S. population, based on differential application by cause of death or other specifications of cessation lag.

The dynamic life-table approach used in this model can theoretically provide improved estimates of the mortality impacts of air pollution in future years over the more common

³⁸ The PopSim tool in BenMAP-CE is based upon an Access-based model originally developed for EPA by Industrial Economics, Inc. This Population Simulation model is described in Chapter 5 of EPA's "Second Prospective Study – 1990 to 2020 – Benefits and Costs of the Clean Air Act. U.S. Environmental Protection Agency, Office of Air and Radiation, April 2011."

static approach because it explicitly accounts for the year-to-year cascade of impacts on mortality and population following an air pollution change.

To use the PopSim tool, first select **PopSim** from the BenMAP-CE **Tools** menu.

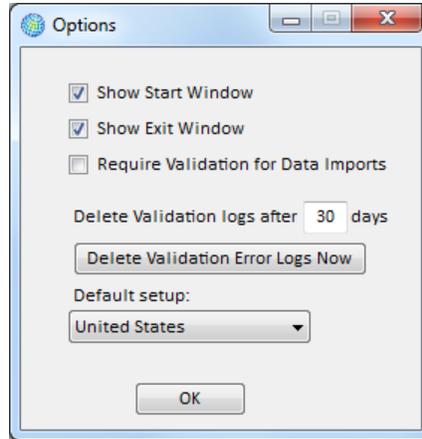


Click through each of the menu items (e.g., **Years**, **Response**, **PM changes**, **Ages affected**, **Lag type**, and **Other**) and review the default settings, or adjust the values as needed. For more information on the underlying datasets or model specifications, please refer to the “*Population Simulation Model for Air Pollution Hazards. Version 3.0. User Manual and Documentation*” (IEC, September 2015).

When you are finished with the model settings, click the **Run model** button on the **Run** screen. A progress bar will be displayed as the calculations are performed. When the model run has completed, a dialog will be displayed. Click **OK** to acknowledge the message. Then click the **Output** button. Specify a location to save the model results. By default, the tool will save to the “*My Documents\My BenMAP-CE Files\PopSim*” folder. The progress bar will once again display as the files are saved to your destination. When complete, a dialog will indicate “*File Saved*”. Click **OK** to acknowledge the message. You may close the PopSim tool, or return to the beginning to initiate a new model run. The tool will remember the settings from the previous run only.

9.11 Options

You can customize some of the generic options for BenMAP-CE using the *Options* window available from the **Tools** menu.



- **Show Start Window:** If unchecked, the **Welcome** window will not appear during subsequent start-ups. You can update your choice at any time.
- **Show Exit Window:** If unchecked, the window confirming you would like to exit will not appear during subsequent shut-downs. You can update your choice at any time.
- **Require Validation for Data Imports:** If checked, you will be required to validate their input files prior to importing datasets. If unchecked, validation will be an available option but it will not be required.
- **Delete Validation logs after __ days:** You can specify the number of days BenMAP-CE will retain the validation logs (the default value is 30 days). If the number of days is left blank, BenMAP-CE will not automatically delete any validation logs.³⁹
- **Delete Validation Error Logs Now:** Select this button if you want to immediately clear all validation logs. *Note: There is no confirmation option here – once the button is clicked, all logs will be deleted.*
- **Default setup:** Select the preferred setup to appear by default in the main BenMAP-CE window.

9.12 Compute Grid Crosswalk

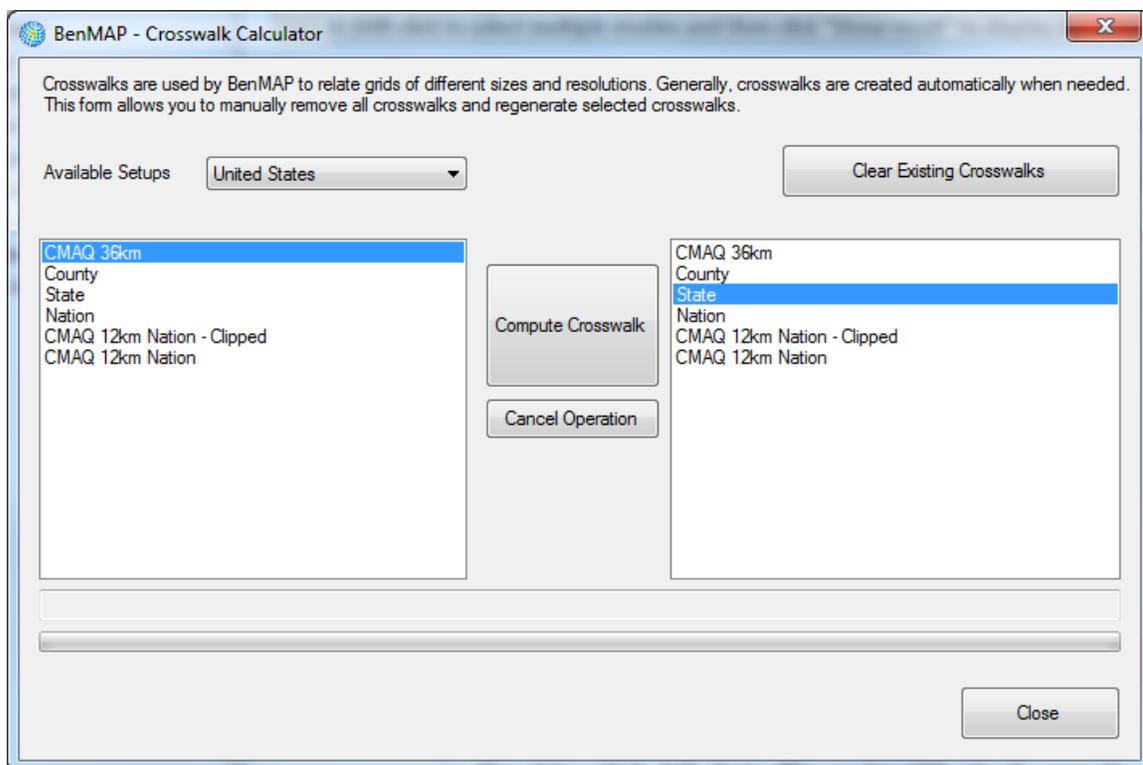
The Compute Grid Crosswalk tool allows users to manually remove crosswalks and regenerate selected crosswalks. A crosswalk is a file used to relate data, such as air quality, population and demographic data, at one spatial scale to another. Crosswalks

³⁹ Validation logs are saved at C:\Users\\Documents\My BenMAP-CE Files\ValidationResults.

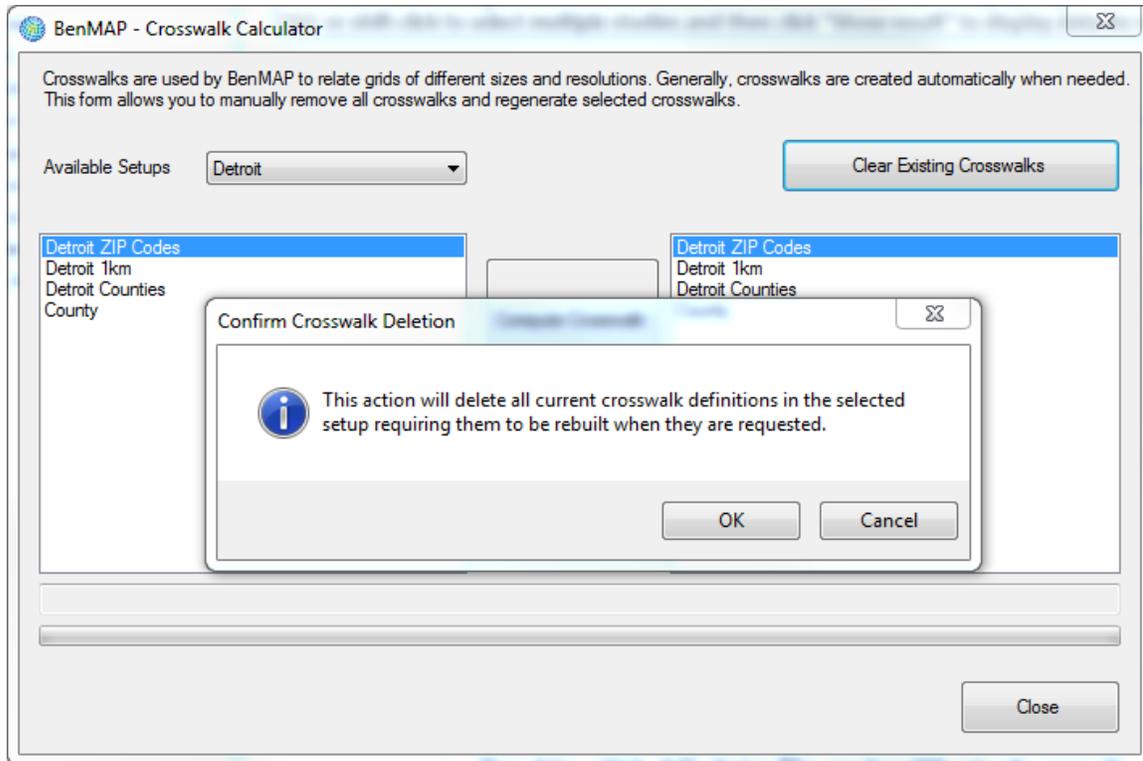
are created during when importing a grid definition or calculating health impacts and economic benefits. BenMap-CE does not remove or overwrite a crosswalk automatically if it already exists. In cases where a crosswalk was broken due to database error or force quitting of the application, users can use this tool to remove the crosswalk from the database. Users may then let BenMap-CE automatically re-generate the crosswalk during processing or re-create it in the tool to save time later.

To re-create crosswalks, go to the **Tools** menu, and choose the **Compute Grid Crosswalks**.

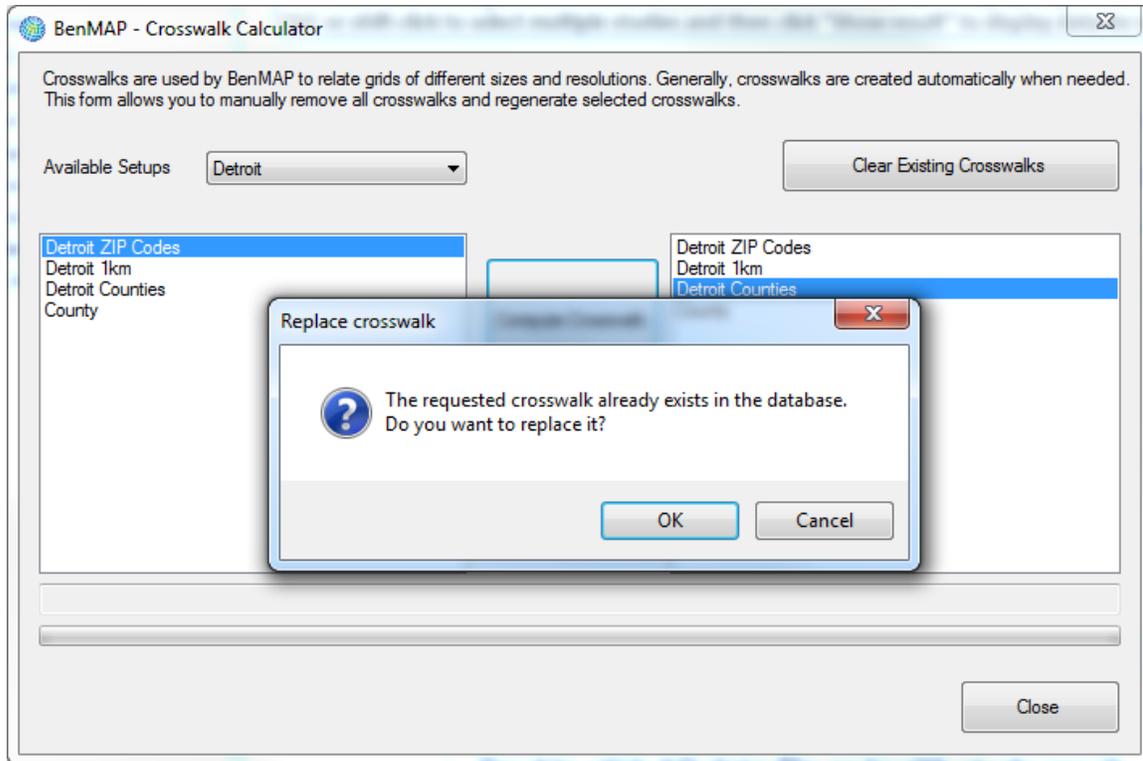
This will bring up the Crosswalk Calculator window. You will see all grid definitions associated to the active setup are listed in both windows. To switch to a different setup, choose another setup from **Available Setups**.



To remove all crosswalks among the grid definitions in the windows, click **Clear Existing Crosswalks**. A message box will pop up asking you to confirm this operation. Click **OK** to confirm. In the screenshot below, we are trying to remove all existing crosswalks for *Detroit* setup from the database. NOTE: crosswalks for default setups (e.g. *China* and *United States*) are locked and cannot be removed.



To create or overwrite a crosswalk between two grid definitions, highlight one grid definition from the left side, highlight the other grid definition from the right side, and click Compute Crosswalk button. If the crosswalk already exists, you will be asked to confirm replacement by clicking **OK** in the message box. In the screenshot below, we selected *Detroit ZIP Codes* from the left window and *Detroit Counties* from the right window to create crosswalks between the grids for both directions.



The calculation may process slowly depending on the size and resolution of your grid cells. If you have to interrupt the process, simply click Cancel Operation. It is recommended to highlight the grid definition with higher resolution from the left window and the one with lower resolution from the right window. This will speed up the calculation.



Environmental Benefits Mapping and Analysis Program – Community Edition

User's Manual

Appendices

March 2023

Appendix A. Monitor Rollback Algorithms

This Appendix details the algorithms BenMAP-CE calculates when you perform a “monitor rollback.” The monitor rollback adjusts the air quality monitoring data to reflect hypothetical changes in air pollution in a given location. The program allows you to roll back the monitoring data using three approaches: Percentage, Increment, and Rollback to Standard.

Once a set of monitors has been selected, the user may define one or more non-overlapping rollback **regions**. A region is simply an area in which you perform a specified rollback. Three rollback **types** are available:

- Percentage Rollback. Monitor values are reduced the same percentage.
- Incremental Rollback. Monitor values are changed by the same fixed increment.
- Rollback to a Standard. Monitor values are reduced so that attainment of a specified standard is reached.

Each of these rollback types has different rollback parameters associated with it.

A.1 Percentage Rollback

Percentage Rollback involves setting only two parameters - a **percentage** and a **background level**. The rollback procedure is similarly straightforward - each observation at each monitor in the region has the portion of its value which is above **background level** reduced by **percentage**.

Example: Background Level: 35; Percentage: 25

Initial Observations at a monitor in rollback region:

20 20 25 59 35 51 83 35 30 67 87 79 63 35 35

If we select the background level of 35, we first calculate the portion of each observation that is above background level, that is, we subtract the background level from the initial observation level. Observations below background level are given a value of 0.

Observation portions above **background level**:

0 0 0 24 0 16 48 0 0 32 52 44 28 0 0

When we apply the rollback percentage, each observation portion gets reduced by 25%. Reduced portions above background level:

0 0 0 18 0 12 36 0 0 24 39 33 21 0 0

Then, each reduced portion is added to the background level of 35. Zero values are replaced by the initial observations. Reduced Observations:

20 20 25 53 35 47 71 35 30 59 74 68 56 35 35

A.2 Incremental Rollback

Incremental Rollback similarly involves setting only two parameters - an **increment** and a **background level**. The rollback procedure is quite similar to the percentage rollback procedure - each observation at each monitor in the region has the portion of its value which is above **background level** reduced by **increment**. The reduced values are not allowed to become negative, however. That is, the values are truncated at zero.

Example: Background Level: 35; Increment: 25 Initial Observations:

20 20 25 59 35 51 83 35 30 67 87 79 63 35 35

Observation portions above background level:

0 0 0 24 0 16 48 0 0 32 52 44 28 0 0

Reduced portions above background level:

0 0 0 0 0 0 23 0 0 7 27 19 3 0 0

Reduced Observations:

20 20 25 35 35 35 58 35 30 42 62 54 38 35 35

A.3 Rollback to a Standard

Rollback to a Standard has two groups of parameters - those associated with the **Attainment Test**, which determines whether a monitor is in attainment (meets the standard), and those associated with the **Rollback Methods**, which are used to bring out of attainment monitors into attainment.

The **Attainment Test** parameters are **Metric**, **Ordinality**, and **Standard**. A monitor is considered in attainment if the nth highest value of the metric specified by **Metric** is at or below the value specified by **Standard**, where n is the value specified by **Ordinality**. For example, if **Metric** is "TwentyFourHourDailyAverage," **Ordinality** is two, and **Standard** is eighty-five, a monitor will be considered in attainment if the second highest value of TwentyFourHourDailyAverage is at or below eighty-five.

Supported metrics for pollutants with hourly observations (Ozone) include FiveHourDailyAverage, EightHourDailyAverage, TwelveHourDailyAverage, TwentyFourHourDailyAverage, OneHourDailyMax, and EightHourDailyMax. Supported metrics for pollutants with daily observations (PM_{2.5}) include TwentyFourHourDailyAverage and AnnualAverage. For Annual Average, **Ordinality** does not apply, since there is only a single metric value to work with.

The **Rollback Method** parameters are **Interday Rollback Method**, **Interday Background Level**, **Intraday Rollback Method**, and **Intraday Background Level**. These four parameters determine the rollback procedures used to bring out of attainment monitors into attainment. The **Interday Rollback Method** and **Background Level** are used to generate target values for the metric specified by the **Attainment Test**. The **Intraday Rollback Method** and **Background Level** are used to adjust hourly observations to meet the target metric values generated in the previous step. As such, the **Intraday Rollback Method** and **Background Level** are used only for pollutants with hourly observations (ozone).

A.3.1 Interday Rollback – Generating Target Metric Values

Because standards are defined on metrics, not directly on observations, the first step in rolling back out-of-attainment monitors is generating target metric values. There are three supported rollback methods for Interday Rollbacks: Percentage, Incremental, and Peak Shaving. Each of these rollback methods requires some preprocessing of the initial monitor metric values. We will discuss this preprocessing first, and then go through Percentage, Incremental, and Peak Shaving rollbacks in turn.

The Interday Background Level specifies the portion of each metric value which cannot be affected by human intervention - we call this portion the non-anthropogenic portion. Whatever portion is left over after subtracting out the background level is referred to as the anthropogenic portion. The anthropogenic portion of the initial monitor metric values is the only part which will be affected by the Interday Rollback Method.

BenMAP calculates an out of attainment value by determining the particular monitor metric value which caused the monitor to be out of attainment - this value is the n th highest value of the metric specified by the Attainment Test metric, where n is the Attainment Test ordinality. BenMAP then calculates an anthropogenic out of attainment value by subtracting the Interday Background Level from the out of attainment value. BenMAP also calculates an anthropogenic standard by subtracting the Interday Background Level from the Attainment Test standard. Finally, BenMAP calculates a set of anthropogenic metric values and a set of non-anthropogenic metric values using the following procedure on each initial monitor metric value:

IF the metric value is less than or equal to the Interday Background Level,

non-anthropogenic metric value = metric value

anthropogenic metric value = 0

ELSE

non-anthropogenic metric value = Interday Background Level

anthropogenic metric value = metric value - Interday Background Level

A.3.1.1 Interday Rollback – Percentage

To generate target metric values using Percentage rollback, BenMAP calculates the percentage required to reduce the anthropogenic out of attainment value to exactly meet the anthropogenic standard. This percentage reduction is then applied to all of the anthropogenic metric values. Finally, these reduced anthropogenic metric values are added to the non-anthropogenic metric values to give the final target metric values.

Example:

Initial Metric Values:

30 35 50 100 80 44 67 88 90 70 50 30 55 90 80 85

Attainment Test: Highest value of metric ≤ 70

Interday Background Level: 40

Out of Attainment Value: 100

Anthropogenic Out of Attainment Value: 60 (= 100 - 40)

Anthropogenic Standard: 30 (= 70 - 40)

Percentage Reduction Required: 50% $(=(60-30)/60)$

Non-Anthropogenic Metric Values:

30 35 40 40 40 40 40 40 40 40 40 30 40 40 40 40

Anthropogenic Metric Values:

0 0 10 60 40 4 27 48 50 30 10 0 15 50 40 45

Reduced Anthropogenic Metric Values:

0 0 5 30 20 2 14 24 25 15 5 0 8 25 20 23

Target Metric Values:

30 35 45 70 60 42 54 64 65 55 45 30 48 65 60 63

A.3.1.2 Interday Rollback – Incremental

To generate target metric values using Incremental Rollback, BenMAP calculates the increment required to reduce the anthropogenic out of attainment value to exactly the anthropogenic standard. This incremental reduction is then applied to all of the anthropogenic metric values (but they are not allowed to fall below zero). Finally, these

reduced anthropogenic metric values are added to the non-anthropogenic metric values to give the final target metric values.

Example:

Initial Metric Values:

30 35 50 100 80 44 67 88 90 70 50 30 55 90 80 85

Attainment Test: Highest value of metric ≤ 70

Interday Background Level: 40

Interday Rollback Method: Incremental

Out of Attainment Value: 100

Anthropogenic Out of Attainment Value: 60

Anthropogenic Standard: 30 (=70 - 40)

Incremental Reduction Required: 30

Non-Anthropogenic Metric Values:

30 35 40 40 40 40 40 40 40 40 40 30 40 40 40 40

Anthropogenic Metric Values:

0 0 10 60 40 4 27 48 50 30 10 0 15 50 40 45

Reduced Anthropogenic Metric Values:

0 0 0 30 10 0 0 18 20 0 0 0 0 20 10 15

Target Metric Values:

30 35 40 70 50 40 4 58 60 40 40 30 40 60 50 55

A.3.1.3 Interday Rollback - Peak Shaving

To generate target metric values using Peak Shaving rollback, BenMAP simply truncates all anthropogenic metric values at the anthropogenic standard. These reduced anthropogenic metric values are added to the non-anthropogenic metric values to give the final target metric values. **Example:** Initial Metric Values:

30 35 50 100 80 44 67 88 90 70 50 30 55 90 80 85

Attainment Test: Highest value of metric ≤ 70

Interday Background Level: 40

Interday Rollback Method: Peak Shaving

Anthropogenic Standard: 30

Non-Anthropogenic Metric Values:

30 35 40 40 40 40 40 40 40 40 40 30 40 40 40 40

Anthropogenic Metric Values:

0 0 10 60 40 4 27 48 50 30 10 0 15 50 40 45

Reduced Anthropogenic Metric Values:

0 0 10 30 30 4 27 30 30 30 10 0 15 30 30 30

Target Metric Values:

30 35 50 70 70 44 67 70 70 70 50 30 55 70 70 70

A.3.2 Intraday Rollback - Adjusting Hourly Observations

Once a set of target metric values has been calculated for a pollutant with hourly observations (e.g., Ozone), BenMAP must adjust the hourly observations so that they produce the target metric values. There are two supported rollback methods for Intraday Rollback – Percentage and Incremental. Each of these rollback methods requires some preprocessing of the initial monitor observations, and each can require multiple iterations to hit the target metric values.

We will discuss this preprocessing and iteration first, and then go through Percentage and Incremental rollbacks in turn.

For various reasons, each of the Intraday Rollback methods can fail to hit the target metric values during a single pass through the rollback procedure (these will be discussed in detail below). As such, each of the rollback methods uses an iterative approach to get within a threshold of each of the target metric values - currently this threshold is 0.05. The iterative approach works as follows:

For each target metric value, BenMAP calculates the current value of the Attainment Test metric. For the first iteration, the metric value will be calculated using unadjusted hourly observations. For subsequent iterations, the metric value will be calculated using the current values of the adjusted hourly observations.

If the difference between the metric value and the target metric value is less than or equal to 0.05, the rollback procedure is finished. Otherwise, another iteration is required.

The Intraday Background Level specifies the portion of each observation which cannot be affected by human intervention - we call this portion the non-anthropogenic portion. Whatever portion is left over after subtracting out the background level is referred to as the anthropogenic portion. The anthropogenic portion of the initial monitor observations is the only part which will be affected by the Intraday Rollback Method.

In a way analogous to the Interday Rollback procedure, BenMAP calculates the twenty-four hourly anthropogenic observations and the twenty-four hourly non-anthropogenic observations using the following procedure for each hourly observation:

IF the current value of the observation is less than or equal to the Intraday Background Level,

non-anthropogenic observation = observation

anthropogenic observation = 0

ELSE

non-anthropogenic observation = Intraday Background Level

anthropogenic observation = observation - Intraday Background Level

Given (i) an Attainment Test Metric (e.g., EightHourDailyMax), (ii) an Intraday Background Level, and (iii) a target metric value for the day, BenMAP proceeds to adjust hourly observations in the following steps:

1. Calculate the Attainment Test metric (e.g., the 8-hour daily maximum);
2. Identify the “window” - i.e., the set of hours used to calculate the metric (e.g., if the 8-hour daily maximum is achieved in the first 8 hours, then the window is comprised of the first 8 hours);
3. Calculate the non-anthropogenic hourly observations (=min(hourly observation, Intraday Background Level));
4. Calculate the anthropogenic hourly observations (=hourly observation - Intraday Background Level);
5. Calculate the non-anthropogenic metric value (= the metric using the non-anthropogenic hourly observations in the “window”);
6. Calculate the anthropogenic metric value (= the metric using the anthropogenic hourly observations in the “window”);

7. Calculate the anthropogenic target metric value (= the target metric value minus the non- anthropogenic metric value);
8. Calculate the reduction required to get the anthropogenic metric value down to the anthropogenic target metric value;
9. Adjust all anthropogenic hourly observations by the reduction calculated on the previous step;
10. Calculate the adjusted hourly observations (= the adjusted anthropogenic hourly observation + the non-anthropogenic hourly observation).

A.3.2.1 Intraday Rollback - Percentage

Below, we present two examples of a percentage-based Intraday Rollback. In one example, a single iteration is needed, and in the second example, two iterations are required because a number of the monitor values fall below the assumed background level.

A.3.2.1.1 Example: All Hourly Observations Exceed the Intraday Background (Single Iteration)

If all of the hourly observations in a day are greater than the Intraday Background Level, then the above procedure is straightforward and can be accomplished in a single iteration. We illustrate with the following example. Suppose that:

Metric = EightHourDailyMax,

Target metric value for a given day = 85

Intraday Background Level = 40.

And that the hourly observations on that day are:

530 45 50 60 45 45 45 60 70 100 100 100 100
 100 100 100 100 60 45 50 45 45 47 47

Based on these observations, we see that the 8-hour daily maximum = 110.

Assuming a background level of 40, then the Anthropogenic hourly observations are:

490 5 10 20 5 5 5 20 30 60 60 60 60 60
 60 60 60 20 5 10 5 5 7 7

Then, we know:

Anthropogenic metric value = 70.

Non-anthropogenic metric value = 40.

Anthropogenic target metric value = 45.

Percentage reduction required = $((70-45)/70) = 35.7\%$

All of the hourly anthropogenic observations are reduced by 35.7%. The average of the first 8 values (the window on which the Test metric is based) will be exactly 45, the anthropogenic target metric value. Finally, the adjusted hourly observations are calculated by adding the non- anthropogenic hourly observation to the adjusted hourly anthropogenic observations.

A.3.2.1.2 Example: Some Hourly Observations are Below the Intraday Background (Multiple Iterations Required)

In the above example, the anthropogenic target metric value was met on a single iteration because all of the hourly observations were greater than the Intraday Background Level. In this case, a simple percent reduction of all hourly values will produce an average in the window that is equal to the anthropogenic target metric value. If some of the hourly observations in a day are less than or equal to the Intraday Background Level, however, then BenMAP uses an iterative procedure.

On each iteration, it adjusts hourly observations using the 10-step method given above. It then compares the new metric value to the target metric value. If the difference is less than or equal to 0.05 ppb, the rollback procedure is finished. Otherwise, another iteration is required. The iterative procedure is illustrated in the following example.

Suppose that:

Metric = EightHourDailyMax,

Target metric value for a given day = 85

Intraday Background Level = 40.

Suppose also that the hourly observations on that day are:

530	20	25	60	35	35	40	60	70	100	100	100	100
100	100	100	100	60	33	40	30	30	25	20		

Then, we know that the 8-hour daily maximum = 100.6.

Non-Anthropogenic Hourly Observations, Iteration One:

40	20	25	40	35	35	40	40	40	40	40	40	40
40	40	40	40	40	40	33	40	30	30	25	20	

Anthropogenic Hourly Observations, Iteration One:

490	0	0	20	0	0	0	20	30	60	60	60	60	60
60	60	60	20	0	0	0	0	0	0				

Non-Anthropogenic Metric Value: 34.4 (EightHourDailyMax - calculated over the same eight hour window as the initial metric value was calculated over)

Anthropogenic Metric Value: 66.3

Anthropogenic Target Metric Value: 50.6

Percentage Reduction Required: 23.6%

Reduced Anthropogenic Hourly Observations, Iteration One:

374	0	0	15	0	0	0	15	23	46	46	46		
46	46	46	46	46	15	0	0	0	0	0	0	0	0

Reduced Hourly Observations, Iteration One:

414	20	25	55	35	35	40	55	63	86	86	86	86	
86	86	86	86	55	33	40	30	30	25	20			

Reduced Metric Value (EightHourDailyMax): 85.8

Target Metric Value (EightHourDailyMax): 85

Non-Anthropogenic Hourly Observations, Iteration Two:

40	20	25	40	35	35	40	40	40	40	40	40	40	40
40	40	40	40	40	33	40	30	30	25	20			

Anthropogenic Hourly Observations, Iteration Two:

374	0	0	15	0	0	0	15	23	46	46	46	46	46
46	46	46	46	15	0	0	0	0	0	0	0		

Non-Anthropogenic Metric Value: 40 (EightHourDailyMax - calculated over the same eight hour window the initial metric value was calculated over)

Anthropogenic Metric Value: 45.8

Anthropogenic Target Metric Value: 45

Percentage Reduction Required: 1.9%

Reduced Anthropogenic Hourly Observations, Iteration Two:

368	0	0	15	0	0	0	15	23	45	45	45	45	45
45	45	45	15	0	0	0	0	0	0				

Reduced Hourly Observations, Iteration Two:

408	20	25	55	35	35	40	55	63	85	85	85	85	85
85	85	85	55	33	40	30	30	25	20				

Reduced Metric Value (EightHourDailyMax): 85

The above example, in addition to illustrating the Intraday Percentage Rollback, also illustrates one reason why the iterative procedure can be necessary. When using the EightHourDailyMax metric in the Attainment Test, it is possible for the window over which the maximum eight hour average occurs to move after a single pass through the rollback procedure. When this happens, it becomes necessary to go through additional iterations to hit the target metric value.

A.3.3 Intraday Rollback - Incremental

To adjust hourly observations using Incremental rollback, BenMAP calculates the increment required to reduce the anthropogenic metric value to exactly the anthropogenic target metric value. This incremental reduction is then applied to all of the anthropogenic observations (but - they are not allowed to fall below zero). Finally, these reduced anthropogenic observations are added to the non-anthropogenic observations to give the final reduced observations.

Example:

Initial Hourly Observations:

20	20	25	60	35	35	40	70	35	30	65	90	76
65	35	35	54	60	33	40	30	30	25	20		

Initial Metric Value (EightHourDailyMax): 60

Target Metric Value (EightHourDailyMax): 55

Intraday Background Level: 40

Intraday Rollback Method: Incremental

Non-Anthropogenic Hourly Observations, Iteration One:

20	20	25	40	35	35	40	40	35	30	40	40	40
40	35	35	40	40	33	40	30	30	25	20		

Anthropogenic Hourly Observations, Iteration One:

0	0	0	20	0	0	0	30	0	0	25	50	36
25	0	0	14	20	0	0	0	0	0	0		

Non-Anthropogenic Metric Value (EightHourDailyMax): 38.8

Anthropogenic Metric Value (EightHourDailyMax): 21.3

Anthropogenic Target Metric Value (EightHourDailyMax): 16.3

Incremental Reduction Required: 5.0

Reduced Anthropogenic Hourly Observations, Iteration One:

0	0	0	15	0	0	0	25	0	0	20	45	31
20	0	0	9	15	0	0	0	0	0	0		

Reduced Hourly Observations, Iteration One:

20	20	25	55	35	35	40	65	35	30	60	85	71
60	35	35	49	55	33	40	30	30	25	20		

Reduced Metric Value (EightHourDailyMax): 56.25

Target Metric Value (EightHourDailyMax): 55

Non-Anthropogenic Hourly Observations, Iteration Two:

20	20	25	40	35	35	40	40	35	30	40	40	40
40	35	35	40	40	33	40	30	30	25	20		

Anthropogenic Hourly Observations, Iteration Two:

0	0	0	15	0	0	0	25	0	0	20	45	31
20	0	0	9	15	0	0	0	0	0	0		

Non-Anthropogenic Metric Value (EightHourDailyMax): 38.8

Anthropogenic Metric Value (EightHourDailyMax): 17.5

Anthropogenic Target Metric Value (EightHourDailyMax): 16.3

Incremental Reduction Required: 1.25

Reduced Anthropogenic Hourly Observations, Iteration Two:

0	0	0	14	0	0	0	24	0	0	19	44	30
19	0	0	8	14	0	0	0	0	0	0		

Reduced Hourly Observations, Iteration Two:

20	20	25	54	35	35	40	64	35	30	59	84	70
59	35	35	48	54	33	40	30	30	25	20		

Reduced Metric Value (EightHourDailyMax): 55.3

Target Metric Value (EightHourDailyMax): 55

This example should actually continue for one further iteration, with a new Incremental Reduction of 0.3. This illustrates another reason why the iterative procedure can be necessary - for incremental reductions, the prohibition against values becoming negative can cause target metric values to not be met. Incremental reductions thus very often require multiple iterations.

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 some type of grid used in an air quality model, such as the CMAQ model, or to a grid defined by political boundaries such as the counties of the United States. In the United States setup, the program includes population counts aggregated to each 12km by 12km grid cell. In the next step, BenMAP estimates the air pollution exposure for each grid-cell, thus assuming that people living within a particular grid-cell experience the same air pollution levels.

You have a variety of approaches to estimate the exposure to air pollution for the people living within a given population grid-cell. Perhaps the simplest approach is to use model data directly, and to assume that the people living within a particular model grid-cell experience the level estimated by the model. 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 take an average of nearby monitors.

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. Table B-1 lists the types of metrics commonly used in concentration-response functions. In the case of air pollution metrics calculated on a daily basis, such as the one-hour maximum and the 24-hour average, it is often the case that there are missing days of data. Air quality modeling is often conducted on a subset of the days in the year, and air quality monitors often miss a number of observations throughout the year. BenMAP accounts for missing days from different data sources as described below.

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

Measurement Frequency	Metric Name	Metric Description
Daily (e.g., PM _{2.5})	Daily Average	Daily average
	Annual Average	Average of four quarterly averages. The four quarters are defined as: Jan-Mar, April-June, Jul-Sep, Oct-Dec.
	Annual Median	Median of values throughout the year.
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.
	12-hour Daily Average	Average of hourly values from 8:00 A.M. through 7:59 P.M.
	24-hour Daily Average	Average of hours from 12:00 A.M. through 11:59 P.M.

B.1 Direct Modeling

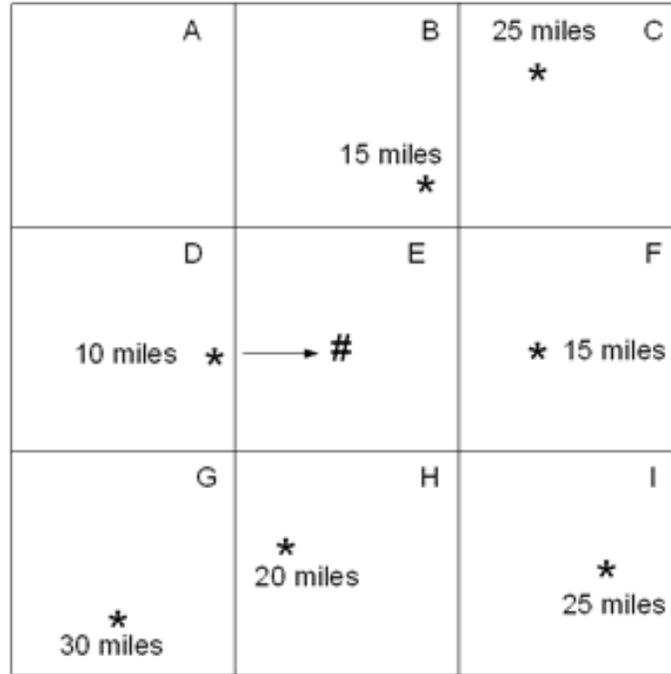
When using direct modeling data to estimate exposure, BenMAP assumes that the people living within a particular air pollution model grid-cell experience the same air pollution levels. BenMAP then estimates the air pollution metrics of interest, as defined for each pollutant. (See the section on defining pollutants in the Loading Data chapter.)

Generally, modeling data providing hourly observations are complete for any given day. However, it is common to have missing days of modeling data during the course of a year. Given the estimated metrics from the available data, BenMAP assumes that the missing days have the same values as the seasonal average of available data.

B.2 Closest Monitor

When using the closest monitor to represent air pollution levels at a population grid-cell, BenMAP identifies the center of the population grid-cell, and then chooses the monitor that is closest to the center. In the simplest case, BenMAP assigns the closest monitor to a population grid-cell, uses the monitoring data to calculate the annual and daily air pollution metrics. The annual metrics and daily metrics are then used to calculate health effects.

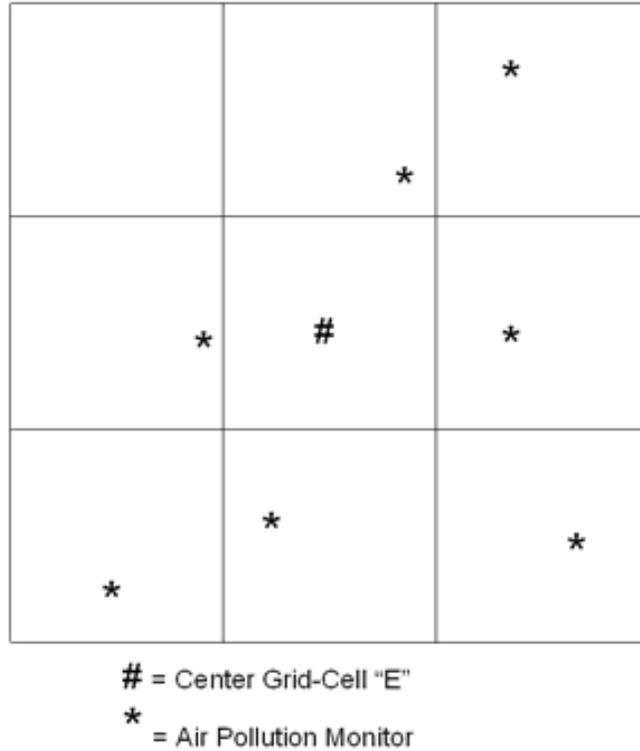
The figure below presents nine population grid-cells and three monitors, with the focus on identifying the monitor closest to grid-cell "E." In this example, the closest monitor happens to be 10 miles away from the center of grid-cell E, and the data from this monitor would be used to estimate air pollution levels for the population in this grid-cell. An analogous procedure would be used to estimate air pollution levels in the other grid-cells (A, B, C, D, F, G, H, and I).



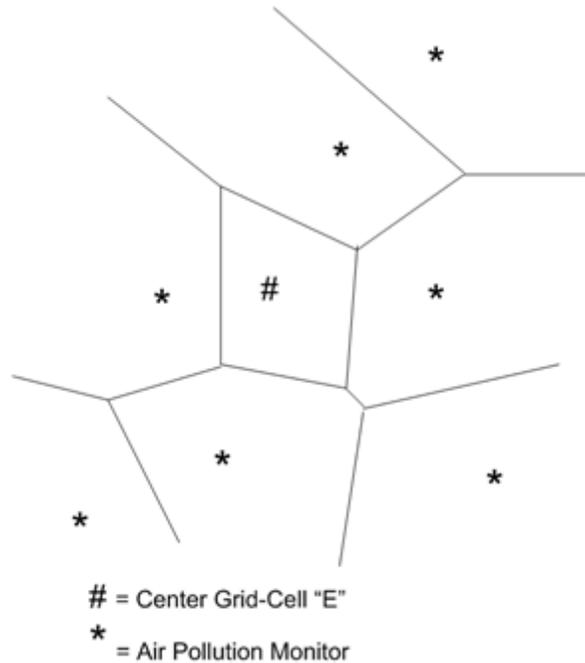
= Center Grid-Cell "E"
 * = Air Pollution Monitor

B.3 Voronoi Neighbor Averaging (VNA)

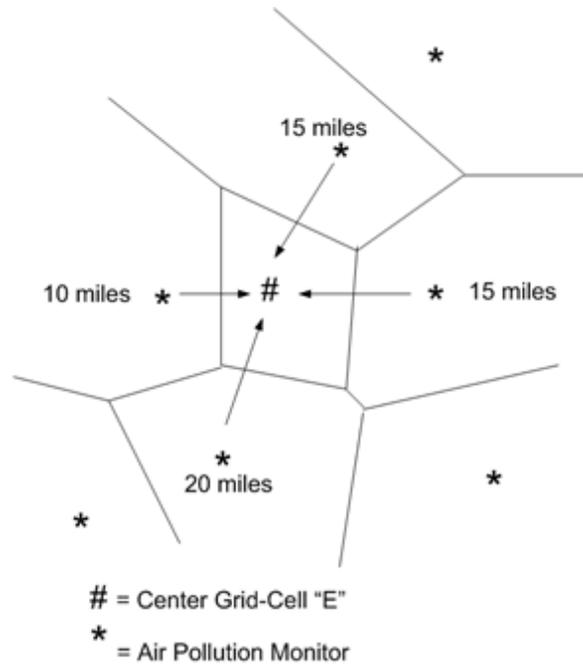
Instead of using the single closest monitor to estimate exposure at a population grid-cell, the VNA algorithm interpolates air quality at every population grid cell by first identifying the set of monitors that best “surround” the center of the population grid-cell.



In particular, BenMAP identifies the nearest monitors, or “neighbors,” by drawing a polygon, or “Voronoi” cell, around the center of each BenMAP grid cell. The polygons have the special property that the boundaries are the same distance from the two closest points.



BenMAP then chooses those monitors that share a boundary with the center of grid-cell “E.” These are the nearest neighbors, BenMAP uses these monitors to estimate the air pollution level for this grid-cell.



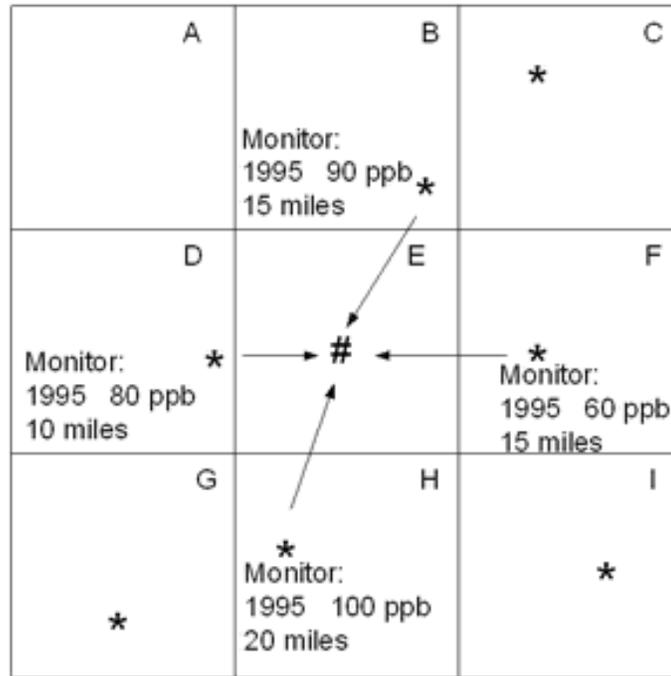
To estimate the air pollution level in each grid-cell, BenMAP calculates the metrics for each of the neighboring monitors, and then calculates an inverse-distance weighted average of the metrics. The further the monitor is from the BenMAP grid-cell, the smaller the weight.

In the figure below, the weight for the monitor 10 miles from the center of grid-cell E is calculated as follows:

$$weight_{10miles} = \frac{\frac{1}{10}}{\left(\frac{1}{10} + \frac{1}{15} + \frac{1}{15} + \frac{1}{20}\right)} = 0.35$$

The weights for the other monitors would be calculated in a similar fashion. BenMAP would then calculate an inverse-distance weighted average for 1995 air pollution levels in grid-cell E as follows:

Forecast 1995 = $0.35 \times 80 \text{ ppb} + 0.24 \times 90 \text{ ppb} + 0.24 \times 60 \text{ ppb} + 0.18 \times 100 \text{ ppb} = 81.2 \text{ ppb}$.



= Center Grid-Cell "E"
* = Air Pollution Monitor

B.4 Fixed Radius

When using the fixed radius option to represent air pollution levels at a population grid-cell, BenMAP identifies all monitors within a specified distance of the center of the population grid cell, calculates the metrics at each monitor, and then calculates a weighted average of the metrics using the algorithms described for VNA. When no monitors are within the specified distance, BenMAP assigns the closest monitor to a population grid-cell, and calculates the metrics using the algorithms described for the closest monitor approach.

B.5 Monitor or Model Data with Missing Days

When estimating air pollution exposure, it will often happen that metrics are missing for one or more days in the season or year. To remedy this, BenMAP calculates seasonal average values to substitute for missing daily values within each season. When combining air pollution metrics from multiple monitors, BenMAP first calculates the seasonal average values for the daily metrics, substitutes these for missing values, and then performs the user-specified interpolation method, such as VNA.

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, Δx , and the change in population health response (usually an incidence rate), Δy 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, $\Delta x = \Delta \text{PM}$ - to illustrate how the relationship between Δx and Δy is derived from each of several different C-R functions.

Estimating the relationship between ΔPM and Δy 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 ΔPM and Δy (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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$, 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-1. 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_c , of being adversely affected. The relative risk (RR) is simply:

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

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

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

The odds ratio is then:

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

This can be rearranged as follows:

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

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:

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

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:

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

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:

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

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

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:

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

Or, equivalently,

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

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 $\alpha = \ln(B)$. 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,

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

where B_0 is the incidence of y when all covariates in the model are zero, and x_1, \dots, x_n are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in y are calculated, and is therefore not important.

The relationship between ΔPM and Δy is:

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

This may be rewritten as:

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

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, Δy , 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 (β) and its standard error (σ_β) 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:

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

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

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, β (e.g., Schwartz et al., 1995, Table 4). Recall that the relative risk (RR) is simply the ratio of two risks:

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

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

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

The coefficients associated with the lower and upper bounds (e.g., the 2.5th and 97.5th 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 (σ_β) can be approximated by:

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

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

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

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:

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

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:

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

The odds of an occurrence is:

$$\begin{aligned} odds &= \frac{y}{1-y} = \frac{\left(\frac{1}{1 + e^{-X \cdot \beta}} \right)}{1 - \frac{1}{1 + e^{-X \cdot \beta}}} \\ \Rightarrow odds &= \frac{\left(\frac{1}{1 + e^{-X \cdot \beta}} \right)}{1 - \frac{1}{1 + e^{-X \cdot \beta}}} = \frac{\left(\frac{1}{1 + e^{-X \cdot \beta}} \right)}{\left(\frac{e^{-X \cdot \beta}}{1 + e^{-X \cdot \beta}} \right)} = \frac{1}{e^{-X \cdot \beta}} = e^{X \cdot \beta} \\ &\Rightarrow \ln(odds) = X \times \beta \end{aligned}$$

The odds ratio for the control scenario ($odds_c$) versus the baseline ($odds_0$) is then:

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

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:

$$\begin{aligned} odds\ ratio &= \frac{\frac{y_0}{1-y_0}}{\frac{y_c}{1-y_c}} = \frac{e^{X_0 \cdot \beta}}{e^{X_c \cdot \beta}} = e^{\beta \cdot \Delta X} = e^{\beta \cdot \Delta PM} \\ \frac{y_c}{1-y_c} &= \frac{y_0}{1-y_0} \times e^{-\beta \cdot \Delta PM} \end{aligned}$$

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

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

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

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

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

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

When the coefficient (β) and its standard error (σ_β) 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:

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

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

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:

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

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

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

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

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

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

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:

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

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):

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

The odds ratio, may then be calculated:

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

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

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

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 $\mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ exposure.

Assuming that the baseline rate for chronic bronchitis (y_0) should be 7.17 percent, the question becomes whether the “increment” of 45 $\mu\text{g}/\text{m}^3$ should be added to or subtracted from the existing $\text{PM}_{2.5}$ 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:

$$\text{odds ratio}_1 = \frac{\left(\frac{y_0}{1-y_0}\right)}{\left(\frac{y_c}{1-y_c}\right)} = \frac{\left(\frac{1.81 \times 0.0717}{1-(1.81 \times 0.0717)}\right)}{\left(\frac{0.0717}{1-0.0717}\right)} = 1.931$$

$$\text{odds ratio}_2 = \frac{\left(\frac{y_0}{1-y_0}\right)}{\left(\frac{y_c}{1-y_c}\right)} = \frac{\left(\frac{0.0717}{1-0.0717}\right)}{\left(\frac{0.0717}{1-\frac{181}{1.81}}\right)} = 1.873$$

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

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

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:

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

Assuming that:

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

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

It is then possible to calculate the underlying coefficient:

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

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

Since this coefficient estimate is based on the assumption that

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

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:

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

Using the formula for the change in the incidence rate and assuming a 10 $\mu\text{g}/\text{m}^3$ decline in $\text{PM}_{2.5}$, it is shown that this results in changes within the bounds suggested by the two estimates based on using the estimated odds ratios:

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

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

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

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 (β_1 or β_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:

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

The proportional hazards model takes the form:

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

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:

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

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. Health Incidence & Prevalence Data in U.S. Setup

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:

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

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;

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

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-1. Regional Definitions from U.S. Census

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

If the sum of deaths calculated in the previous (regional) step was still less than 20, the counties in the “regional collection of counties” were not assigned the single rate from the above step. Instead, we proceeded to the national level, identifying all counties in the nation whose death counts were less than 20 (excluding any such counties that were assigned a rate in the previous steps). We summed the death counts in those counties and divided by the sum of the populations in those counties to derive a single rate for the “national collection of counties.” This rate was then applied to each of those counties in the “national collection of counties.” 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-2. National Mortality Rates (per 100 people per year)
by Health Endpoint 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	0.59396	0.01951	0.07804	0.10665	0.17264	0.40542	0.86162	1.79670	4.62837	13.58034
Mortality, Non-Accidental	A00-R99	0.55495	0.00949	0.01874	0.04112	0.10876	0.33084	0.79395	1.73208	4.49595	13.20867
Mortality, Respiratory	J00-J98	0.01297	0.00102	0.00127	0.00253	0.00570	0.02013	0.06560	0.20585	0.57827	1.42362
Mortality, Chronic Lung	J40-J47, J67	0.00053	0.00032	0.00040	0.00074	0.00186	0.01033	0.04045	0.13873	0.36008	0.68593
Mortality, Lung Cancer	C34	0.00002	0.00001	0.00007	0.00033	0.00282	0.02378	0.07992	0.19701	0.32952	0.31820
Mortality, Ischemic Heart Disease	I20-I25	0.00033	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	0.00539	0.00069	0.00099	0.00214	0.00502	0.01794	0.05877	0.18453	0.51055	1.26213
Mortality, NCD + LRI	**	0.18459	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	0.00269	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	0.00116	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-3. All-Cause Mortality Rate (per 100 people per year), by Source, Year, and Age Group

Source & Year	Infant	1-17	18-24	25-34	35-44	45-54	55-64	65-74	75-84	85+
Calculated CDC 2012-2014	0.193*	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-4. 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 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, 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.

D.1.4 Ethnicity-Stratified Mortality Projections (2020-2060)

Baseline ethnicity-stratified mortality rates were derived from county-level CDC WONDER data for 2007-2016 (see section D.1.3). In order to project these rates into future years, we procured national-level mortality projections from the U.S. Census Bureau. These projections cover the years 2017 to 2060. Because county-level mortality projections are not readily available, we apply the national rates of change to the baseline county values. In addition, we bridge the temporal gap between the 2007-2016 period—treated as a 2011 average—and the 2017-onwards projection using historical CDC WONDER data for 2011 to 2017.

Table D-5. Datasets Used in Developing Ethnicity-Stratified Incidence Rates (2020-2060)

Initial Baseline Dataset	CDC WONDER Cause of Death Dataset - for adjustment to 2017	Census Bureau Population Projection	Census Bureau Deaths Projections
Years: 2007-2016 Ages: < 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 Spatial scale: County-level	Years: 2011, 2012, 2017 Ages: < 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 Spatial scale: Census regions	Years: 2017-2060 Ages: 0, 1, 2,...99, 100 Spatial scale: National	Years: 2017-2060 Ages: 0, 1, 2, ...84, 85+ Spatial scale: National

D.1.4.1 Adjusting Baseline Dataset to 2017 Using CDC WONDER Historical Data

First, the baseline dataset was adjusted to 2017 values using historical data from CDC WONDER's Underlying Cause of Death dataset (<https://wonder.cdc.gov/ucd-icd10.html>). All-cause mortality datasets were pulled for years 2011, 2012, and 2017, for Hispanic and Non-Hispanic groups. Data was aggregated from the Census Region to a national level. Values for years 2011 and 2012 were averaged together to represent the midpoint of the 2007 to 2016 baseline rates. A scaling factor was calculated between the 2011-2012 average and 2017 values. These scaling factors (Table D-6) were applied to the original, county-level baseline mortality rates to adjust the dataset to represent 2017 mortality incidence.

Table D-6. Ratio of 2017 to 2011-2012 All-Cause Mortality Rate

Start Age	End Age	Hispanic	Non-Hispanic
0	0	1.003709	0.933217
1	4	0.857753	0.941917
5	14	1.006885	1.067448
15	24	1.141657	1.101791
25	34	1.282166	1.261146
35	44	1.162141	1.147084
45	54	0.988912	0.998058
55	64	0.987971	1.049693
65	74	0.970447	0.983404
75	84	0.947003	0.951082
85	99	0.939672	0.996925

D.1.4.2 Projecting Dataset into 2060 Using Census Bureau Projections

To project this adjusted 2017 baseline dataset into the future, we used a similar process to the methods described in Section D.1.2. We obtained Census Bureau projected deaths and population for 2017-2060 at the national level and adjusted the age groups to match between datasets. The Census Bureau datasets were stratified by ethnicity (Hispanic and Non-Hispanic). We calculated all-cause mortality rates by dividing projected deaths by projected population.

We then calculated, separately for each age group and ethnicity group combination, the ratio of the Census Bureau national mortality rate in year Y (Y= 2017, 2018, ...2060) to the 2017 rate. These ratios are shown for a selection of years and age groups in Table D-7.

Table D-7. Ratio of Future All-Cause Mortality Rate to 2017 Estimated All-Cause Mortality Rate

Ethnicity	Year	Age Group					
		0-0	5-14	25-34	45-54	65-74	85-99
Hispanic	2020	0.98	0.97	0.94	0.94	0.97	0.99
	2030	0.93	0.89	0.76	0.82	0.88	0.92
	2040	0.87	0.79	0.61	0.70	0.80	0.87
	2050	0.82	0.71	0.48	0.59	0.73	0.86
	2060	0.77	0.63	0.38	0.49	0.66	0.86
Non-Hispanic	2020	0.97	0.96	0.93	0.93	0.97	1.00
	2030	0.87	0.82	0.69	0.71	0.86	0.91
	2040	0.79	0.72	0.52	0.57	0.75	0.86
	2050	0.72	0.62	0.40	0.45	0.63	0.86
	2060	0.65	0.54	0.30	0.36	0.56	0.87

Finally, the ratios displayed in Table D-7 were used to develop projected mortality datasets on the county level stratified by ethnicity (Hispanic and Non-Hispanic) for years 2020, 2025, ... 2055, 2060. We multiplied each county's incidence value with the scaling ratio corresponding to its specific age group and ethnicity for years 2020-2060.

D.1.5 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.⁴⁰ To address the frequent county-level data suppression for race-specific

⁴⁰ Infant mortality dates for race- and ethnicity-stratified datasets do not currently exclude neonatal deaths.

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 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.

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-CE.

D.1.6 Race-Stratified Mortality Projections (2020-2060)

The methods for projecting race-stratified mortality incidence rates are comparable to those described in section D.1.4 for ethnicity-stratified rates. The only difference includes mapping different definitions of race across datasets, as summarized in Table D-8. Due to existing population configurations in BenMAP-CE, the mortality projections were adjusted to match the limited set of racial groups currently in the tool.

Table D-8. Census Bureau Projections Racial Groups

Census Bureau Population Projections	Census Bureau Deaths Projections	Existing Definitions in BenMAP-CE
<p>Definition 1</p> <ol style="list-style-type: none"> White alone Black alone American Indian and Alaska Native alone Asian alone Native Hawaiian and Other Pacific Islander alone Two or more races <p>Definition 2</p> <ol style="list-style-type: none"> White alone or in combination Black alone or in combination American Indian and Alaska Native alone or in combination Asian alone or in combination Native Hawaiian and Other Pacific Islander alone or in combination 	<p>Definition 1</p> <ol style="list-style-type: none"> White alone Black alone American Indian and Alaska Native alone Asian alone Native Hawaiian and Other Pacific Islander alone Two or more races 	<ol style="list-style-type: none"> White Black Native American Asian

As can be seen in Table D-8, the racial categories available in the Census Bureau projections data do not match those already in BenMAP-CE. Most notably, (1) the *Native Hawaiian and Other Pacific Islander alone* population is not aggregated with *Asian alone* as is done in BenMAP-CE; and (2) *Two or more races* is separated into its own racial group. To map the Census Bureau data into BenMAP-CE definitions, the following steps were taken.

First, the population dataset was used to find proportions to re-assign death and population counts in the *Two or more races* group to the five other racial groups:

- The *White alone* count was subtracted from the *White alone or in combination* count to find the aggregate number of those that were considered multiracial, who included “White” in their racial identity. This is referred to as the “combination count.” This was completed for each racial group.
- The “combination counts” for each racial group were added together, to give the total counts by those in the *Two or more races* group. This total is larger than the size of the *Two or more races* group itself, since individuals selected multiple races.

3. Each “combination count” per racial group was divided by the total combination count to create a proportion. This proportion represents the aggregate proportion of combination counts out of all combination counts for a racial group.
4. These proportions were used to assign deaths and population counts in the Two or more races group to the other five groups.

Example:

$(\text{Two or more races total} * \text{Proportion for White group}) + \text{White alone} = \text{White population}$

Finally, the death and population counts for the Native Hawaiian and Other Pacific Islander group was added to the Asian group.

The resulting mortality scaling ratios are shown for select years and age groups in Table D-9.

Table D-9. Ratio of Future All-Cause Mortality Rate to 2017 Estimated All-Cause Mortality Rate

Race	Year	Age Group					
		0-0	5-14	25-34	45-54	65-74	85-99
Asian	2020	0.98	0.99	0.95	0.95	0.98	0.97
	2030	0.92	0.83	0.76	0.82	0.90	0.88
	2040	0.86	0.72	0.63	0.72	0.82	0.83
	2050	0.80	0.63	0.51	0.60	0.76	0.84
	2060	0.75	0.56	0.41	0.51	0.72	0.82
Black	2020	0.95	0.94	0.92	0.91	0.95	0.98
	2030	0.79	0.77	0.66	0.67	0.80	0.89
	2040	0.66	0.63	0.47	0.52	0.66	0.82
	2050	0.56	0.52	0.34	0.40	0.54	0.82
	2060	0.47	0.43	0.24	0.30	0.46	0.83
Native American	2020	0.96	0.96	0.92	0.90	0.94	0.98
	2030	0.83	0.80	0.67	0.69	0.79	0.89
	2040	0.72	0.68	0.49	0.55	0.66	0.83
	2050	0.63	0.58	0.37	0.43	0.56	0.83
	2060	0.56	0.50	0.27	0.34	0.49	0.84
White	2020	0.98	0.97	0.93	0.93	0.97	1.01
	2030	0.92	0.86	0.70	0.73	0.85	0.91
	2040	0.86	0.76	0.54	0.59	0.74	0.86
	2050	0.81	0.68	0.42	0.47	0.64	0.86
	2060	0.76	0.61	0.32	0.38	0.57	0.87

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 hospitalizations 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.

- We then counted the number of discharges for the endpoint in the state excluding those records with missing age or county FIPS.
 - The adjustment factor is the ratio of the two counts.
- For California and West Virginia, patient county was unavailable for all observations. For these two states, we used hospital county in place of patient county.
- 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-, endpoint- specific hospitalization rates and applied them to each county in the state. We used the previously described procedure to adjust the “unreliable” rates.

- For states without discharge-level or state-level data:
 - We obtained the endpoint-specific hospitalization counts in each region from HCUPnet/NIS (we refer to this count for the i th endpoint in the j th region as “TOTAL ij ”)
 - For those states in the j th region that do have discharge-level or state-level data, we summed the hospital admissions by endpoint (we refer to this count for the i th endpoint in the j th region as “SUB ij ”).
 - We then estimated the hospitalization count for states without discharge or state data for the i th endpoint in the j th region as $TOTALij - SUB ij$. Note that while this count is endpoint- and region- specific, it is not age-specific. We obtained the distribution of hospital admission counts across age groups based on the 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 endpoint-, region-, and age-specific counts.

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

The endpoints 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 endpoints above, we developed a set of county level baseline incidence for one EHA endpoint, 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 endpoint 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 endpoint 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 endpoint is applicable for the additional codes. Table D-10 presents a summary of the national hospitalization rates for 2014 from HCUP.

Table D-10. Hospitalization Rates (per 100 people per year), by Health Endpoint 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.

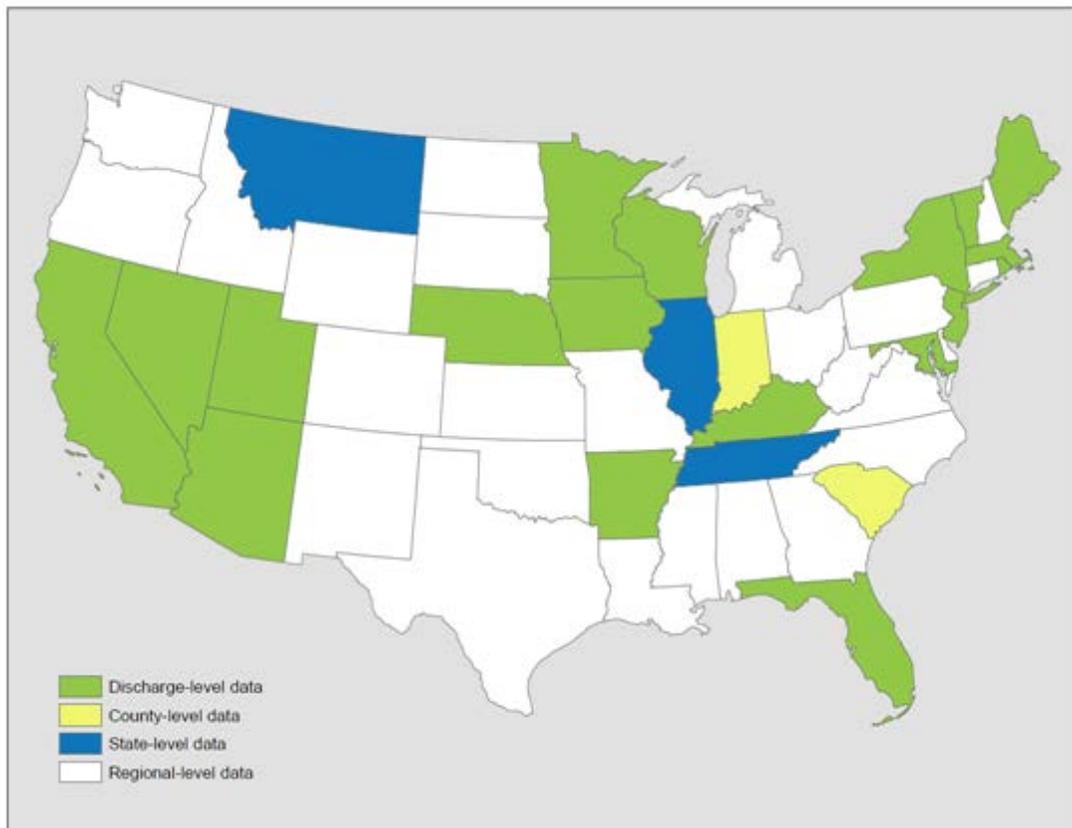
An alternative approach to the estimation of heart attack rates is to use data from the Healthcare Cost and Utilization Project (HCUP), assuming that all heart attacks that are not instantly fatal will result in a hospitalization. Details about HCUP data are described in Section D.2. According to the 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-10.

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, endpoint and county, we summed the ER visits that ended with discharge and those that resulted in hospitalization.
- The data year varies across the states from 2011 to 2014; 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-11 presents the estimated asthma emergency room rates by health endpoint and age group.

Table D-11. Emergency Department Visit Rates (per 100 people per year) by Health Endpoint 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-12 presents the estimated school loss day rates. Further detail is provided below on these rates.

Table D-12. 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 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 endpoints include: acute bronchitis, chronic bronchitis, upper respiratory symptoms, lower respiratory symptoms. Table D-13 presents a summary of these baseline rates.

Table D-13. Selected Acute and Chronic Incidence (Cases / Person-Year) & Prevalence (Percentage Population)

Endpoint	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.

Endpoint	Age	Parameter	Rate	Source
	18-24		1.971	Bureau of the Census (1997, No.22)
	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.6.1 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.6.2 Chronic Bronchitis Incidence Rate

The annual incidence rate for chronic bronchitis⁴¹ 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.6.3 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.6.4 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 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 =

⁴¹ Please note that this endpoint is not regularly considered in U.S. EPA analyses (July 2018).

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.6.5 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.6.6 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.7 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 endpoints are described below and summarized in Table D-14. The prevalence of asthma is summarized in Table D-15.

Table D-14. Asthma-Related Health Effects Incidence Rates

Endpoint	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)

Endpoint	Age	Parameter	Rate	Source
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), 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.7.1 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.7.2 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.7.3 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.7.4 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.7.5 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.7.6 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.7.7 Asthma Population Estimates

In studies examining the association between air pollution and the development or exacerbation of asthma, often times 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-15 presents asthma prevalence rates used to define asthmatic populations in the health impact functions.

Table D-15. Asthma Prevalence Rates Used to Estimate Asthmatic Populations

Endpoint	Population Group	Asthma Prevalence	Source
Asthma Symptoms	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%	American Lung Association (2010b, Table 9)
	African-American, <5	9.98%	
	African-American, 5 to 17	17.76%	
	African-American, <18	15.53%	American Lung Association*
New Onset Asthma**	0-4	3.80%	National Health Interview Survey (2018, Table 4-1)
	5-17	8.93%	
	4-17	8.56%	
	7-21	9.01%	
	Female, 35 to 64	9.90%	
	Female, 65+	10.00%	
Asthma Symptoms (Albuterol Use), Albuterol Use	6-13	8.6%	

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

** Asthma prevalence rates differ for the same age groups due to population weighting

D.8 Other Health Endpoint Occurrence

Baseline incidence estimates for health endpoint 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-16 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-16. 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-17).

Table D-17. 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 average from the stratified. This resulted in a rate of 0.004 strokes per person per year. Table D-18 presents the stratified incidence and survival rates.

Table D-18. 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 U.S. Setup

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-1. Core Health Impact Functions for Particulate Matter and Long-Term Mortality

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
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	
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24Hour Mean	0.01133	0.001602	Log-linear	
Mortality, All Cause	Wu et al.	2020	Nationwide	65-99		D24Hour Mean	0.00639	0.000383	Log-linear	

E.1.1 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 O₃ 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/m³.

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/m³ (Di et al., 2017, Table 2 Main Analysis, Cox PH with GEE).

E.1.2 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 O₃ 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/m³ 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 O₃ 1982-2004).

E.1.3 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 µg/m³, with 25% of the population experiencing concentrations below 12 µg/m³ and above 18.8 µg/m³. 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 µg/m³ 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.1.4 Pope et al. (2019)

Pope et al. (2019) examined the relationship between long-term PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} are estimated from the hazard ratio (1.12) and 95% confidence interval (1.08-1.15) associated with a change in annual mean PM_{2.5} exposure of 10.0 µg/m³ (Pope et al. 2019, Table 2, Subcohort).

E.1.5 Wu et al. (2020)

Wu et al. (2020) evaluated the relationship between long-term PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} are estimated from the hazard ratio (1.066) and 95% confidence interval (1.058-1.074) associated with a change in annual mean PM_{2.5} exposure of 10.0 ug/m³ (Wu et al. 2020, Table S3, Main analysis, 2000-2016 Cohort, Cox PH).

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 / severe health effects. We present a brief summary of each of the studies below.

Table E-2. 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	Sullivan et al.	2005	King County, WA	0-99		D24HourMean	0.0019	0.0022	Logistic	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Infarction, Nonfatal										
Acute Myocardial Infarction, Nonfatal	Wei et al.	2019	Continental U.S.	65-99		D24HourMean	0.0011	0.0002	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.

E.2.1 Ensor et al. (2013)

Ensor et al. (2013) studied the association between short-term ambient air pollution (PM_{2.5} and O₃) 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 O₃. 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 µg/m³ 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 O₃) 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 O₃) analyses. The study concluded that each 10 µg/m³ 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 O₃.

Incidence, Lung Cancer

In a two-pollutant multivariable model with O₃ (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/m³ 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/m³ 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 endpoint 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/m³ 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 PM₁₀, PM_{10-2.5}, PM_{2.5}, "black carbon", O₃, CO, NO₂, and SO₂ 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 PM₁₀ 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 $\mu\text{g}/\text{m}^3$ increase in twenty-four hour average $\text{PM}_{2.5}$ (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 $\text{PM}_{2.5}$ 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 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$. The researchers determined that a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ 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 $\mu\text{g}/\text{m}^3$ increase in daily 24-hour mean $\text{PM}_{2.5}$ (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 µg/m³ 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}, NO₂, SO₂, O₃, 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 cases of myocardial infarction through King County, Washington. In this case-crossover study from 1988-1994, air pollution exposure levels averaged 1 hour, 2 hours, 4 hours, and 24 hours before onset of myocardial infarction were compared to a set of time-stratified referent exposures from the same day of the week in the month of the case event. The authors estimated that an associated risk of 1.01 (95% CI: 0.98-1.05) for myocardial infarction onset could be attributed to a 10 µg/m³ 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/m³ 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 Wei et al. (2019)

Wei et al. (2019) evaluated the relationship between short-term PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} 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 $\mu\text{g}/\text{m}^3$ increase in 0-1 day lagged $\text{PM}_{2.5}$ exposure (Wei et al. 2019, Figure 3, CCS 100 Acute Myocardial Infarction).

E.2.11 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), $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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 endpoint 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 $\mu\text{g}/\text{m}^3$ increase in daily 24-hour mean $\text{PM}_{2.5}$ 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.2.12 Zanobetti et al. (2009)

Zanobetti et al. (2009) examined the relationship between daily $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ composition while controlling for seasonal temperature as a substitute for ventilation. Overall, the authors found that $\text{PM}_{2.5}$ mass higher in Ni, As, and Cr as well as Br and organic carbon significantly increased its effects on hospital admissions. For a 10 $\mu\text{g}/\text{m}^3$ increase in 2-day averaged $\text{PM}_{2.5}$, 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 endpoint 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/m³ 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.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-3. 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 hierarchical 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 µg/m³ 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 µg/m³ 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), NO₃, and SO₄ 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 $\mu\text{g}/\text{m}^3$ 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 impact 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-4. 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		D24HourMean	0.000612	0.000422	Logistic	
Respiratory	Krall et al.	2013	Atlanta, GA	0-99		D24HourMean	0.000545	0.000267	Log-linear	
Respiratory	Krall et al.	2013	Birmingham, AL	0-99		D24HourMean	0.000968	0.000352	Log-linear	
Respiratory	Krall et al.	2013	St. Louis, MO	0-99		D24HourMean	0.000832	0.000329	Log-linear	
Respiratory	Krall et al.	2013	Dallas, TX	0-99		D24HourMean	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 NO₃ and SO₄ 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 $\mu\text{g}/\text{m}^3$ (interquartile range) increase in daily mean $\text{PM}_{2.5}$ concentration, lagged by 2 days (Ostro et al. 2016, Table 4).

E.4.2 Krall et al. (2013)

Krall et al. (2013) investigated the associations between short-term, source-specific (traffic and coal combustion) ambient $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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. (2013) estimated source-specific $\text{PM}_{2.5}$ using apportionment models, which separate $\text{PM}_{2.5}$ 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. (2013) used Poisson time series regression models to analyze associations between short-term $\text{PM}_{2.5}$ exposure and emergency department visits for respiratory diseases. They then compared source-specific $\text{PM}_{2.5}$ 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 $\mu\text{g}/\text{m}^3$ increase in daily mean $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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-5. 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		D24HourMean	0.004600	0.000360	Log-linear
Minor	Ostro and	1989	Nationwide	18-64	Ozone	D24HourMean	0.007410	0.000700	Log-linear

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form
Restricted Activity Days	Rothschild								
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:

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

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

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

This eventually reduces down to:

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

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 endpoints 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:

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

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

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

This reduces down to:

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

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 SO₂, NO₂, PM_{2.5}, and PM_{2.5-10} and warm season (May to September) O₃ averages to participant's addresses through ambient air pollution and meteorological data (O₃, SO₂, NO₂, 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/m³ 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-6. 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), 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 E₄ 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), 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 µg/m³ 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}, NO₂, O₃) 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}, NO₂, and O₃.

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 $\mu\text{g}/\text{m}^3$ (interquartile range) increase in annual $\text{PM}_{2.5}$ concentration at the residential address (Tétreault et al. 2016, Table 5).

E.7 Sensitivity Analysis – General

Table E-7 summarizes the $\text{PM}_{2.5}$ 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-7. 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		D24HourMean	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	D24HourMean	0.000499	0.000355	Logistic	
Hospital Admissions, All Cardiac Outcomes	Talbott et al.	2014	New Jersey	0-99	O ₃	D24HourMean	0.001094	0.000227	Logistic	
Hospital Admissions, All Cardiac Outcomes	Talbott et al.	2014	New Mexico	0-99	O ₃	D24HourMean	0.001094	0.001943	Logistic	
Hospital	Talbott et al.	2014	New York	0-99	O ₃	D24HourMean	0.001094	0.000151	Logistic	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Admissions, All Cardiac Outcomes										
Hospital Admissions, All Cardiac Outcomes	Talbott et al.	2014	Florida	0-99	O ₃	D24HourMean	-0.000401	0.000307	Logistic	
Hospital Admissions, All Cardiac Outcomes	Talbott et al.	2014	New Hampshire	0-99	O ₃	D24HourMean	-0.001207	0.001238	Logistic	
Hospital Admissions, All Cardiac Outcomes	Talbott et al.	2014	Washington	0-99	O ₃	D24HourMean	-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		D24HourMean	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/m³ (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/m³ (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/m³ (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 (SO₄), ammonium (NH₄), nitrate (NO₃), 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 Climatic 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 µg/m³ 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}, PM₁₀, O₃, and NO₂) 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 $\mu\text{g}/\text{m}^3$ (range of exposure in the 13 communities) increase in annual average $\text{PM}_{2.5}$ 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 ($\text{PM}_{2.5}$, 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 $\text{PM}_{2.5}$ 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 $\mu\text{g}/\text{m}^3$ increase in average annual $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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+). $\text{PM}_{2.5}$ 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/m³ 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 O₃ (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 O₃, 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/m³ in the mean PM_{2.5} exposure level from 1999-2004 (Turner et al., 2016, Table E10 HBM PM_{2.5}, 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/m³ 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/m³ 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-8. 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,	Alhanti et	2016	Atlanta,	0-4		D24HourMean	0.0037	0.0012	Log-linear	Non-White

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Asthma	al.		Dallas, St. Louis							
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
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24HourMean	0.0095	0.0079	Log-linear	Native American, Non-Hispanic
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24HourMean	0.0104	0.0018	Log-linear	White, Non-Hispanic
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24HourMean	0.0140	0.0049	Log-linear	Black, Non-Hispanic
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24HourMean	0.0095	0.0079	Log-linear	Asian, Non-Hispanic
Mortality, All Cause	Pope et al.	2019	Nationwide	18-99		D24HourMean	0.0182	0.0040	Log-linear	Hispanic

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 AQ5 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 $8 \mu\text{g}/\text{m}^3$ increase in three-day moving average $\text{PM}_{2.5}$ 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 $8 \mu\text{g}/\text{m}^3$ increase in three-day moving average $\text{PM}_{2.5}$ concentrations (Alhanti et al. 2016, Supplemental Table 4).

E.8.2 Di et al. (2017)

See full study description under 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 $10 \mu\text{g}/\text{m}^3$ increase in annual mean $\text{PM}_{2.5}$ exposure (Di et al. 2017, Supplementary Table S3 (GEE, By Race)).

E.8.3 Pope et al. (2019)

See full study description under Pope et al. (2019) in Appendix E, Section E.1.4.

Mortality, All-Cause

The basic CPH model is used to find the coefficient and standard error from a hazard ratio of 1.11 (95% CI: 1.07-1.15) for Non-Hispanic White; 1.20 (95% CI: 1.11-1.30) for Hispanic; 1.15 (95% CI: 1.05-1.27) for Non-Hispanic Black; and 1.10 (95% CI: 0.94-1.28) for the “Other/unknown” group, which was applied for the Non-Hispanic Native American and Non-Hispanic Asian groups. Each hazard ratio is for a $10 \mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ (Pope et al. 2019, Supplementary Table S3).

Appendix F. Core Ozone Health Impact Functions in U.S. Setup

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-1. 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	PM _{2.5} , NO ₂	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 O₃ 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 O₃ 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 O₃ from distributed lag days (Katsouyanni et al. 2009, Table 24: Distributed Lags; Penalized splines; O₃ 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 O₃ exposure (1982-2004) and mortality (all-cause, cause-specific) in American Cancer Society Cancer Prevention Study-II participants (aged 30-99 years). A hierarchical 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 NO₂). 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 O₃ 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 O₃ concentrations (Turner et al. 2016, Table E9: Diseases of the respiratory system (cause of death), HBM O₃ (multipollutant model data, fully adjusted HR)).

F.1.3 Zanobetti and Schwartz (2008)

Zanobetti and Schwartz (2008) investigated the effects of short-term O₃ 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 Statistics 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 O₃ 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-2. 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 O₃ 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 O₃ 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 PM₁₀, 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 O₃ 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-3. 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 O₃ 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-4. 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

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
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, PM₁₀, and NO₂ 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 PM₁₀ nor NO₂ was significantly associated with illness-related school absences, but PM₁₀ 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:

$$Duration = \frac{TotalAbsences}{NewAbsences}$$

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

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 ($2.75/5 \times 5/7$).

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 ($2.75/5 \times 5/7$).)

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 endpoints in most years. The C-R function for ozone is based on the co-pollutant model with PM_{2.5}.

The study is based on a “convenience” sample of non-elderly individuals. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to ozone as individuals under 65. A number of studies have found that hospital admissions for the elderly are related to ozone exposures (e.g., Schwartz, 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).

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

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

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

This reduces down to:

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

F.4.3 Parker et al. (2009)

Parker et al. (2009) investigated the associations between long-term O₃ 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 SO₂, NO₂, PM_{2.5}, and PM_{2.5-10} and warm season (May to September) O₃ 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 O₃ (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-5. 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,	Lewis et al.	2013	Detroit, MI	5-12		D8HourMax	0.01140	0.00505	Logistic	All year

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Chest tightness										
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 O₃ 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 O₃ 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 O₃ 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 O₃ 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 O₃ 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 O₃ 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) O₃ 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 O₃ 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 O₃ 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-6. 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.

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
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 description under 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 O₃ lagged by 1 day (Katsouyanni et al. 2009, Table 24: Lag 1; Penalized splines; Controlling for PM₁₀).

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 O₃ lagged by 1 day (Katsouyanni et al. 2009, Table 24: Lag 1; Penalized splines; O₃ 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 O₃ concentrations (Turner et al. 2016, Table E9: HBM O₃, 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 O₃ concentrations (Turner et al. 2016, Table E5: HBM O₃, 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 O₃ concentrations (Turner et al. 2016, Table E7: HBM O₃, 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-7. 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,	Jerrett et al.	2009	Nationwide	30-99		Annual	0.000995	0.001257	Log-	Male, 8-hour max

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Respiratory			US, 96 MSAs			(D8HourMax)			linear	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 Visits, Asthma	Mar and Koenig	2009	Seattle, Washington	0-17		D8HourMax	0.010436	0.004358	Log-linear	Age <18
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 &	2008	Nationwide US, 48 cities	0-99		D8HourMean	0.000359	0.000038	Logistic	Male, Age 0-99

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
	Schwartz									
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 Visits, Asthma	Villeneuve et al.	2007	Edmonton, Canada	75-99		D8HourMax	-0.000558	0.006684	Logistic	Age 75-99
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	2008	Nationwide	81-99		D8HourMean	0.00029	0.000079	Logistic	Age 81-99

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
All Cause	and Schwartz		US, 48 cities							

F.7.1 Cakmak et al. (2006)

Cakmak et al. (2006) examined the relationship between daily average O₃ 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 O₃ 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 O₃ 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 O₃ from distributed lag days (Katsouyanni et al. 2009, Table 24: Distributed Lags; Penalized splines; O₃ 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 O₃ 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 O₃ 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 O₃ 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 O₃ 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 O₃-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:

$$ER_{\text{Total}} = \frac{ER_{\text{Male}}(\text{Pop}_{\text{Male}}) + ER_{\text{Female}}(\text{Pop}_{\text{Female}})}{\text{Pop}_{\text{Total}}}$$

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; $\text{Pop}_{\text{Total}}$ is the total sample population; and $\text{Pop}_{\text{Female}}$ 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:

$$ER_{\text{Female}} = ER_{\text{Male}} + 0.58 \%$$

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} :

$$0.65\% = \frac{ER_{\text{Male}}(1,365,937) + (0.58\% + ER_{\text{Male}})(1,363,703)}{2,729,640}$$

$$ER_{\text{Male}} = 0.36 \%$$

and

$$ER_{\text{Female}} = 0.36\% + 0.58\% = 0.94\%$$

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 O₃ 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 O₃ 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 O₃ 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 U.S. Setup

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 included in the U.S. Setup but are not currently used by the U.S. EPA in regulatory impact analyses. For the health impact functions currently used by EPA, see the following page: <https://www.epa.gov/benmap/benmap-community-edition>. For Ozone Health Impact Functions, click the “U.S. EPA approach for quantifying and valuing ozone effects” link. For PM_{2.5} Health Impact Functions, click the “U.S. EPA approach for quantifying and valuing PM effects” link.

G.1 Additional PM_{2.5} Health Impact Functions

Section G.1 summarizes the additional health impact functions for PM_{2.5} included in BenMAP-CE.

G.1.1 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 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 G-1 lists the additional long-term mortality health impact functions.

Table G-1. Additional Health Impact Functions for Particulate Matter and Long-Term Mortality*

Effect	Author	Year	Location	Age	Metric	Beta	Std Err	Form	Notes
Mortality, All Cause	Expert A	2006		30-99	Annual	0.015180		Log-linear	
Mortality, All Cause	Expert B	2006		30-99	Annual	0.012620		Log-linear	Range >10 to 30 µg. Unconditional dist. 2% no causality included.
Mortality, All Cause	Expert B	2006		30-99	Annual	0.011950		Log-linear	Range 4 to 10 µg. Unconditional dist. 2% no causality included.
Mortality, All Cause	Expert C	2006		30-99	Annual	0.011930		Log-linear	
Mortality, All Cause	Expert D	2006		30-99	Annual	0.008380		Log-linear	Unconditional dist. 5% no causality included
Mortality, All Cause	Expert E	2006		30-99	Annual	0.019670		Log-linear	Unconditional dist. 1% no causality included
Mortality, All Cause	Expert F	2006		30-99	Annual	0.011440		Log-linear	Range >7 to 30 µg
Mortality, All Cause	Expert F	2006		30-99	Annual	0.009370		Log-linear	Range 4 to 7 µg

Effect	Author	Year	Location	Age	Metric	Beta	Std Err	Form	Notes
Mortality, All Cause	Expert G	2006		30-99	Annual	0.006970		Log-linear	Unconditional dist. 30% no causality included
Mortality, All Cause	Expert H	2006		30-99	Annual	0.008700		Log-linear	
Mortality, All Cause	Expert I	2006		30-99	Annual	0.011810		Log-linear	Unconditional dist. 5% no causality included
Mortality, All Cause	Expert J	2006		30-99	Annual	0.009620		Log-linear	
Mortality, All Cause	Expert K	2006		30-99	Annual	0.006890		Log-linear	Range >16 to 30. No threshold. Conditional dist.
Mortality, All Cause	Expert K	2006		30-99	Annual	0.003940		Log-linear	Range 4 to 16 µg. No threshold. Conditional dist.
Mortality, All Cause	Expert K	2006		30-99	Annual	0.003940		Log-linear	Range 4 to 16 µg. Threshold 0 to 5 µg. Conditional dist.
Mortality, All Cause	Expert K	2006		30-99	Annual	0.003940		Log-linear	Range 4 to 16 ug. Threshold 5 to 10 µg. Conditional dist.
Mortality, All Cause	Expert K	2006		30-99	Annual	0			Range >16 to 30. Uniform distribution. No causality.
Mortality, All Cause	Expert K	2006		30-99	Annual	0			Range 4 to 16 ug. Uniform distribution. No causality.
Mortality, All Cause	Expert L	2006		30-99	Annual	0.009340		Log-linear	Range >10 to 30 µg. Unconditional dist. 1% no causality included.
Mortality, All Cause	Expert L	2006		30-99	Annual	0.007390		Log-linear	Range 4 to 10 µg. Unconditional dist. 25% no causality included
Short-term Mortality, Non-Accidental	Baxter et al.	2017	77 U.S. cities	0-99	D24HourMean	0.000329	0.000102	Log-linear	PM PA
Short-term Mortality, All Cause	Ito et al.	2013	150 U.S. cities	0-99	D24HourMean	0.000145	0.000075	Log-linear	PM PA
Mortality, IHD	Jerrett et al.	2017	Nationwide	30-99	Annual	0.013976	0.001775	Log-linear	PM PA
Mortality, All Cause	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.005827	0.000963	Log-linear	
Mortality, IHD	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.021511	0.002058	Log-linear	Mortality, IHD
Mortality, Lung Cancer	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.013103	0.003795	Log-linear	Mortality, Lung Cancer
Mortality, All Cause	Laden et al.	2006	6 cities	25-99	Annual	0.014842	0.004170	Log-linear	

Effect	Author	Year	Location	Age	Metric	Beta	Std Err	Form	Notes
Mortality, All Cause	Lepeule et al.	2012	6 Cities	25-99	Annual	0.013103	0.003347	Log-linear	
Mortality, All Cause	Pope et al.	2002	51 cities	30-99	Annual	0.005827	0.002157	Log-linear	
Mortality, IHD	Pope et al.	2015	Nationwide	30-99	Annual	0.013103	0.001791	Log-linear	PM PA
Mortality, All Cause	Thurston et al.	2016	100 U.S. metro areas	55-85	Annual	0.002956	0.001245	Log-linear	PM PA
Mortality, Lung Cancer	Turner et al.	2016	Nationwide	30-99	Annual	0.008618	0.003032	Log-linear	PM PA
Mortality, All Cause	Woodruff et al.	1997	86 cities	Infant	Annual	0.003922	0.001221	Logistic	
Mortality, All Cause	Woodruff et al.	2006	204 counties	Infant	Annual	0.006766	0.007339	Logistic	
Short-term Mortality, All Cause	Zanobetti et al.	2014	180 U.S. counties	65-99	D24HourMean	0.000638	0.000109	Log-linear	PM PA

*Unless specified as short-term mortality.

G.1.1.1 Expert Functions

In this section, we describe the approach taken to incorporate into BenMAP concentration- response (C-R) functions that were obtained through expert elicitation for EPA (IEc, 2006).

We have specified expert distributions for the PM_{2.5} effect either as truncated parametric distributions or as non-parametric distributions. Therefore they can only be included in BenMAP in the form of custom distribution tables containing 15,000 random draws (with replacement) from an underlying distribution. We first describe the way these custom distribution tables were created. Then we explain how these custom distribution tables should be handled in a configuration file to represent the expert-specified distribution as closely as possible.

Note that the table on page 3-30 of the expert elicitation report (IEc, 2006) refers to the non-parametric distributions as “custom” distributions. However, BenMAP refers to distribution tables that are supplied in the form of a simulated draw as “custom distribution tables”. In order to avoid confusion in terminology, we will call the expert-specified distributions, which did not have a parametric shape, “non-parametric” expert distributions.

We divided the experts into two groups - those who specified a parametric distribution and those who specified a non-parametric distribution. This division was necessary because the two groups required different methods for generating the custom distribution tables. We describe the respective algorithms below and then provide an assessment of the results for each expert.

G.1.1.1.1 Parametric Distributions

Experts A, C, D, E, G, I, J, and K chose parametric distribution functions to represent their subjective beliefs about the percent change in risk associated with an increase in PM_{2.5}. In particular, they specified the following characteristics of the distribution:

- The shape (e.g., Normal, Triangular, Weibull)
- The truncation points (i.e., minimum and/or maximum)
- Two or three percentile points
- The likelihood that the association is causal and whether the function includes that (i.e., whether the function is conditional on the association being causal or unconditional).

There were two types of inconsistencies encountered in these specifications:

(1) The experts who chose Normal or Weibull shapes for their distributions also specified minimum and/or maximum values at which there could be an effect. The Normal distribution has an unlimited support from -8 to + 8. The Weibull distribution has support $(l + 8)$, where l is a location parameter that can be any value on the real line. The specification of a minimum or a maximum value for the effect is therefore inconsistent with specifying these distributions. Therefore, we interpreted these experts' distributions as truncated Normal or truncated Weibull distributions. In other words, we assumed that the shape of the distribution is Normal or Weibull between the truncation points.

(2) Experts A, C, and J indicated that they included the likelihood of causality in their subjective distributions. However, the continuous parametric distributions specified were inconsistent with the causality likelihoods provided by these experts. Because there was no way to reconcile this, we chose to interpret the distributions of these experts as unconditional and ignore the additional information on the likelihood of causality. For example, Expert A specified a truncated Normal distribution with a minimum 0 and a maximum 4. The expert also indicated that the likelihood of causality is 95 percent and it is included in the distribution. This implies that the 5th percentile of the truncated Normal distribution should be zero. The minimum and 5th percentile of the distribution both being zero imply a density with a large (discrete) mass at zero. This, however, is not consistent with specifying a continuous Normal density. (In the case of Expert A, in addition, he specified a 5th percentile value of 0.29, whereas a 5 percent chance of non-causality would imply a 5th percentile value of 0.)

In order to create a random draw from a parametric distribution it is not sufficient to know its shape and truncation points. In addition, one needs to know the values of parameters that distinguish this particular distribution from a class of similarly shaped distributions with identical truncation points. Experts D and I reported parameter

values of their subjective distributions (see details in Table 1). Therefore, we simply drew 15,000 times from each of their distributions.

However, the only information, in addition to the shape and truncation points, which the other experts provided was the percentile points. To derive the parameter values of interest, we used this information as follows:

Let $F(x; \theta, \mathbf{min}, \mathbf{max})$ be a truncated continuous parametric (cumulative) distribution function with (vector of) parameters θ and truncation points \mathbf{min} and \mathbf{max} . The n th percentile point is defined as the value x_n such that $F(x_n; \theta, \mathbf{min}, \mathbf{max}) = n/100$. Thus, if we know that the expert distribution's n th percentile point is x_n and m th percentile point is x_m then the following has to hold:

$$F(x_n; \theta, \mathbf{min}, \mathbf{max}) = n/100$$

$$F(x_m; \theta, \mathbf{min}, \mathbf{max}) = m/100$$

This is a system of non-linear equations that can be solved for the unknown distribution parameters θ . We used the Nelder and Mead (1965) numeric optimization algorithm, available in R, to find the best-fitting estimates of parameters θ for the truncated distributions specified by the experts. Once estimates of θ were obtained, the distributions were specified fully and we had enough information to make 15,000 draws from each.

Table G-2 below summarizes the results for each expert who specified a parametric distribution. In each case, we provide an “input” line that has all the information that was provided by the expert. We also show the “output” line that contains the inferred parameters and five percentile points of the distribution from which draws were made.

Highlighted in yellow are the percentiles specified by the expert and used to create the equation system for the optimization. After finding the best-fitting parameters, we calculated the associated percentiles and confirmed that they are close to the input values.

Table G-2. Description of the Parametric Expert Functions

Expert	Information	Distribution	Min	P5	P25	P50	P75	P95	Max	Parameters
A	input	Normal	0	0.290				2.900	4	mean=? sd=?
	output			0.290	0.929	1.481	2.059	2.900		mean=1.42 sd=0.895
C	input	Normal	0			1.200		2.000	+8	mean=? sd=?
	output			0.423	0.875	1.200	1.528	2.000		mean = 1.196 sd=0.488
D	input	Triangular	0.100						1.600	mode =0.95
	output			0.350	0.662	0.897	1.107	1.382		

Expert	Information	Distribution	Min	P5	P25	P50	P75	P95	Max	Parameters
E	input	Normal	0			2.000		3.000	+8	mean=? sd=?
	output			1.002	1.590	2.000	2.410	3.000		mean=2 sd=0.608
G	input	Normal	-8			1.000		1.300	1.500	mean=? sd=?
	output			0.695	0.875	1.000	1.124	1.300		mean=1.001 sd=0.185
I	input	Normal	0.200						2.300	mean=1.25 sd=0.53
	output			0.473	0.912	1.250	1.588	2.027		
J	input	Weibull	0	0.150		0.900		2.000	3.000	shape=? scale=? location=?
	output			0.150	0.525	0.900	1.331	2.000		shape=2.21 scale=1.413 location=-0.326
K1 4-16 µg/m ³	input	Normal	-8	0.100		0.400			0.800	mean=? sd=?
	output			0.100	0.277	0.400	0.521	0.682		mean=0.404 sd=0.184
K2 >16-30 µg/m ³	input	Normal	-8	0.100		0.700			1.500	mean=? sd=?
	output			0.100	0.455	0.700	0.942	1.264		mean=0.707 sd=0.367

For example, Expert A indicated that the distribution of the effect is Normal, with minimum 0 and maximum 4. Under the assumption that this is actually a truncated Normal distribution, we looked for the corresponding mean and standard deviation for it. The 5th and the 95th percentile values (0.29 and 2.90, respectively) were used to specify the following equations:

$$N(0.29; \text{mean} = ?, \text{sd} = ?, \text{min} = 0, \text{max} = 4) = 0.05$$

$$N(2.90; \text{mean} = ?, \text{sd} = ?, \text{min} = 0, \text{max} = 4) = 0.95$$

The solution to this system was a mean of 1.42 and a standard deviation of 0.89. We also verified that these parameters produced percentile values consistent with the ones supplied by the expert. We similarly solved for the parameters of the other experts who specified parametric distributions, with the exception of experts D and I, who specified their distributions fully.

The experts were asked to describe uncertainty distributions for the percent change in mortality risk associated with a 1 µg/m³ change in PM_{2.5}. All of the experts assumed log-linear (or piecewise log-linear) C-R functions. If Z denotes the percent change elicited from an expert, the relative risk associated with a 1 µg/m³ change in PM_{2.5} is (1+Z/100), and the PM_{2.5} coefficient in the log-linear C-R function is ln(1+(Z/100)). We applied this transformation to the values drawn from each distribution.

Finally, some experts stated that their distribution does not incorporate the likelihood of causality - i.e., they specified conditional distributions. We made 15,000 draws from an expert's conditional distribution. BenMAP contains a function that is zero. If an expert specified, for example, a five percent chance that there is not a causal association, BenMAP will draw from this zero function with five percent probability and draw from the 15,000-draw custom distribution (of positive values) with 95 percent probability. Table G-3 below shows summary statistics for the draws from the parametric distributions that became BenMAP "custom" distribution tables. Additional details on the form of the distributions are below and in Belova et al. (2007).

Table G-3. Descriptive Statistics of the Random Draws from the Parametric Expert Distributions

Expert	Mean	Standard Deviation	Min	P25	P50	P75	Max
A	0.01518	0.00773	0.00000	0.00944	0.01483	0.02051	0.03917
C	0.01193	0.00466	0.00001	0.00870	0.01189	0.01509	0.02848
D (cond)	0.00884	0.00305	0.00105	0.00671	0.00899	0.01108	0.01577
D	0.00838	0.00354	0.00000	0.00623	0.00875	0.01092	0.01577
E (cond)	0.01975	0.00591	0.00026	0.01577	0.01986	0.02376	0.04534
E	0.01967	0.00619	0.00000	0.01575	0.01989	0.02381	0.04534
G (cond)	0.00996	0.00181	0.00256	0.00873	0.00996	0.01123	0.01489
G	0.00697	0.00480	0.00000	0.00000	0.00892	0.01062	0.01489
I (cond)	0.01240	0.00458	0.00200	0.00905	0.01244	0.01575	0.02273
I	0.01181	0.00523	0.00000	0.00845	0.01214	0.01559	0.02273
J	0.00962	0.00567	0.00000	0.00525	0.00902	0.01329	0.02936
K1 (cond)	0.00394	0.00175	-0.00262	0.00278	0.00398	0.00520	0.00797
K1	0.00139	0.00215	-0.00262	0.00000	0.00000	0.00298	0.00796
K2 (cond)	0.00689	0.00350	-0.00766	0.00452	0.00698	0.00937	1.01489
K2	0.00237	0.00382	-0.00402	0.00000	0.00000	0.00489	0.01488

G.1.1.1.2 Non-Parametric Distributions

Experts B, F, H, and L chose a non-parametric distribution function to represent their subjective beliefs about the percent change in risk associated with 1 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5}. They specified the following characteristics of the distribution:

- The truncation points (*i.e.*, minimum and/or maximum)
- Five percentile points
- The likelihood that the association is causal and whether the function includes that (*i.e.*, whether the function is conditional on the association being causal or unconditional)

The only information that we had about these distributions was the minimum, the maximum, and the five percentiles. The shape of the distribution was unknown. Therefore,

we made an assumption that the cumulative distribution function (CDF) is piece-wise linear. In other words, we assumed that all values between the percentiles are equally likely. Following this assumption, we used linear interpolation between the percentile points to derive the CDF for each expert. We then made 15,000 draws from each CDF.

Table G-4 shows the inputs and the outputs of this process for each expert. The inputs are the minimum, the maximum, and the percentiles. The outputs are the percentiles that we calculated from the draws from the respective linearly interpolated CDFs.

Table G-4. Description of the Non-Parametric Expert Functions

Expert	Information	Min	P5	P10	P25	P50	P75	P95	Max
B1 4-10 µg/m ³	input	0.010	0.100		0.200	1.200	2.100	2.600	2.800
	output		0.099		0.203	1.213	2.092	2.599	
B2 >10- 30 µg/m ³	input	0.100	0.200		0.500	1.200	2.100	2.600	2.800
	output		0.198		0.501	1.191	2.096	2.597	
F1 4-7 µg/m ³	input	0.370	0.580		0.730	0.930	1.100	1.400	1.700
	output		0.581		0.732	0.928	1.097	1.407	
F2 >7-30 µg/m ³	input	0.290	0.770		0.960	1.100	1.400	1.600	1.800
	output		0.771		0.958	1.100	1.398	1.606	
H	input	0	0	0	0.400	0.700	1.300	2.000	3.000
	output		0	0	0.407	0.710	1.320	2.010	
L1 4-10 µg/m ³	input	0	0.200		0.570	1.000	1.400	1.600	2.700
	output		0.201		0.570	0.996	1.400	1.619	
L2 >10- 30 µg/m ³	input	0.020	0.200		0.570	1.000	1.400	1.600	2.700
	output		0.018		0.568	1.003	1.396	1.634	

Table G-5 below shows summary statistics for the draws from the non-parametric distributions that became BenMAP “custom” distribution tables. The section below on distributional details contains histograms for all the experts’ distributions.

Table G-5. Descriptive Statistics of the Random Draws from the Non-Parametric Expert Distributions

Expert	Mean	Standard Deviation	Min	P25	P50	P75	Max
B1 (cond)	0.01217	0.00897	0.00010	0.00200	0.01195	0.02090	0.02761
B1	0.01195	0.00901	0.00000	0.00195	0.01167	0.02075	0.02761
B2 (cond)	0.01290	0.00813	0.00100	0.00489	0.01187	0.02068	0.02761
B2	0.01262	0.00827	0.00000	0.00464	0.01159	0.02042	0.02761
F1	0.00937	0.00268	0.00370	0.00727	0.00924	0.01092	0.01686
F2	0.01144	0.00292	0.00290	0.00951	0.01091	0.01387	0.01784

Expert	Mean	Standard Deviation	Min	P25	P50	P75	Max
H	0.00870	0.00662	0.00000	0.00406	0.00702	0.01302	0.02954
L1 (cond)	0.00985	0.00511	0.00001	0.00582	0.00999	0.01391	0.02662
L1	0.00739	0.00613	0.00000	0.00001	0.00727	0.01250	0.02659
L2 (cond)	0.00953	0.00544	0.00000	0.00567	0.00991	0.01389	0.02661
L2	0.00934	0.00549	0.00000	0.00531	0.00964	0.01371	0.02661

G.1.1.1.3 Using Expert Functions in BenMAP

When an expert has specified certain functional specifics with certain probabilities, the resulting “C-R function” becomes a set of possible functions, each with an associated probability. For example, expert K specified a piecewise log-linear function (i.e., two different log-linear functions on two different parts of the range of PM_{2.5}); this expert also specified a threshold within different ranges with different probabilities (and no threshold with a specified probability). BenMAP incorporates such a set of possible functions specified by an expert function by assigning appropriate weights to each specification. We illustrate this using expert K’s specification.

Expert K specified one log-linear function if the baseline PM_{2.5} value falls within the range from 4 µg/m³ to 16 µg/m³ and another log-linear function if the baseline value falls within the range from >16 µg/m³ to 30 µg/m³. BenMAP thus incorporates two sets of functions - one set for each of these two PM_{2.5} ranges - and selects from the set appropriate for a given PM_{2.5} baseline value. Expert K also specified a 64% probability that there is no causal relationship; an 18% probability that there is a causal relationship with no threshold, a 4% probability that there is a causal relationship with a threshold somewhere between 5 µg/m³ to 10 µg/m³, and a 14% probability that there is a causal relationship with a threshold somewhere between 0 µg/m³ to 5 µg/m³. Thus, the set of log-linear functions in BenMAP for expert K on the range from 4 µg/m³ to 16 µg/m³ contains a function with the:

- PM_{2.5} coefficient = 0 (no causality), which BenMAP selects with 65% probability;
- PM_{2.5} coefficient expert K specified for the log-linear function on that range and no threshold, which BenMAP selects with 18% probability;
- PM_{2.5} coefficient expert K specified for the log-linear function on that range and a threshold (with uniform probability) between 0 µg/m³ to 5 µg/m³, which BenMAP selects with 14% probability; and
- PM_{2.5} coefficient expert K specified for the log-linear function on that range and a threshold (with uniform probability) between 5 µg/m³ to 10 µg/m³, which BenMAP selects with 4% probability.

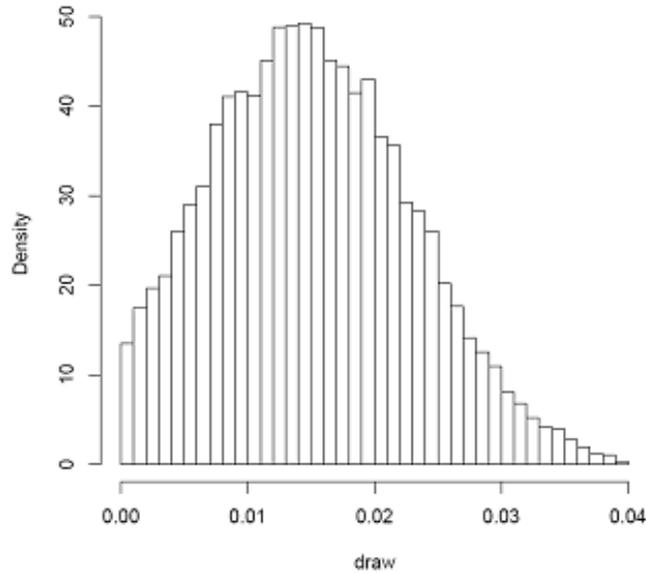
If the PM_{2.5} baseline value is greater than 16 µg/m³, BenMAP goes through an analogous procedure to select a function from among the two functions in that set.

G.1.1.1.4 Distributional Details by Expert

Distributional details on each expert distribution are presented below. The derivation of the distributions is described above with additional details provided by Belova et al. (2007).

G.1.1.1.4.1 Expert A

Figure G-1. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert A



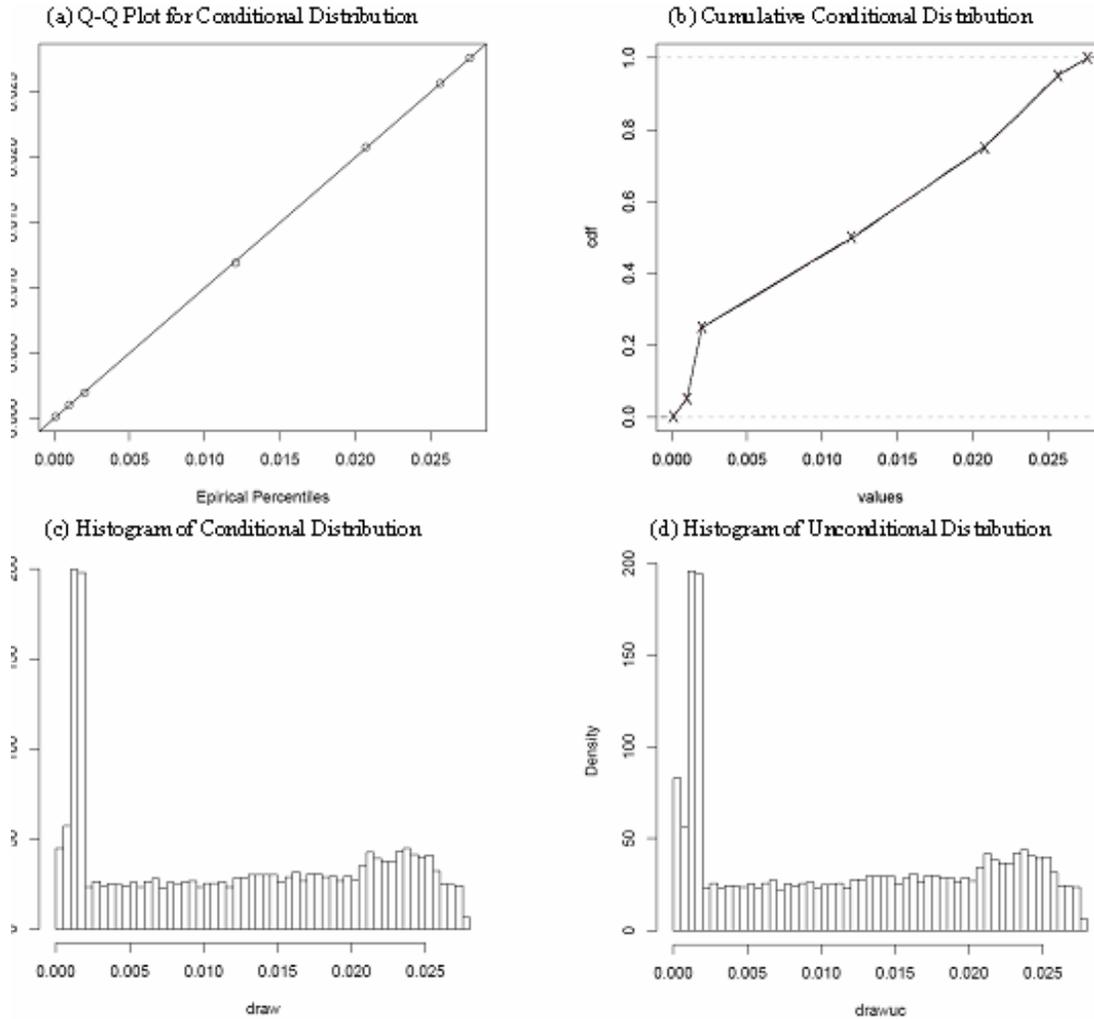
Notes:

Expert A specified a truncated Normal Distribution. We inferred the following values for the parameters of this distribution: mean=1.42 and standard deviation=0.89.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

G.1.1.1.4.2 Expert B

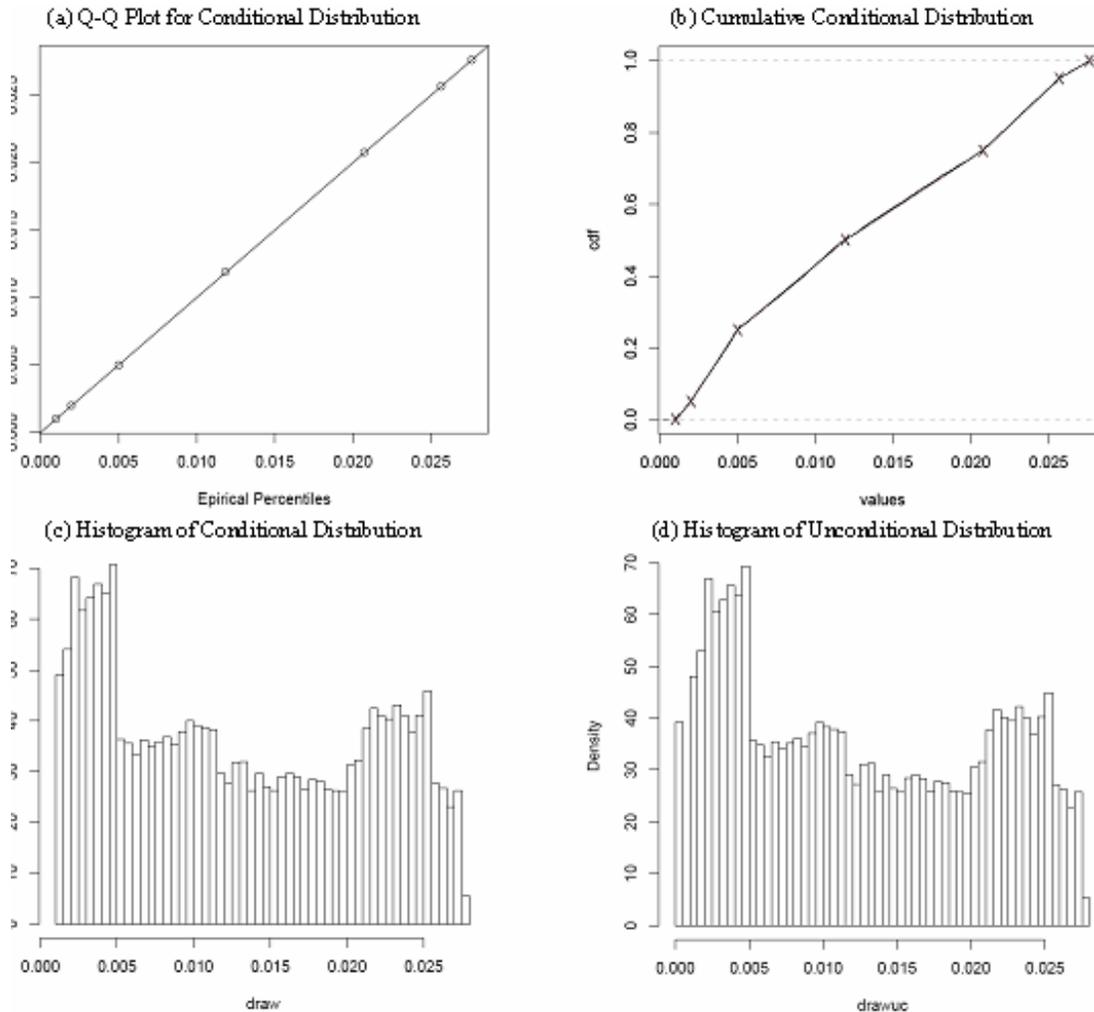
**Figure G-2. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert B
(1) Results for the range 4-10 $\mu\text{g}/\text{m}^3$**

**Notes:**

Expert B specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red "X" marks indicate corresponding expert percentiles. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 2 percent zeros to the draw. Panels (c) and (d) show the respective distributions.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

Figure G-2. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert B (continued)
(2) Results for the range >10-30 $\mu\text{g}/\text{m}^3$



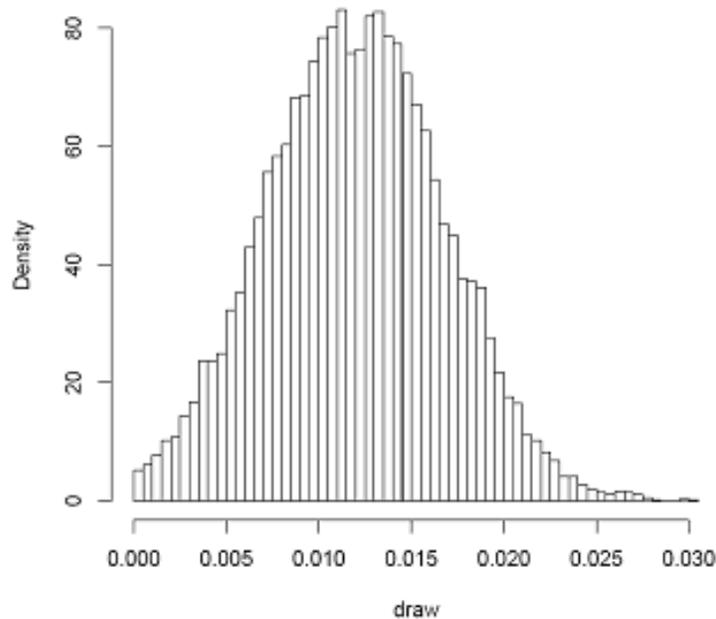
Notes:

Expert B specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 2 percent zeros to the draw. Panels (c) and (d) show the respective distributions.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

G.1.1.1.4.3 Expert C

Figure G-3. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert C

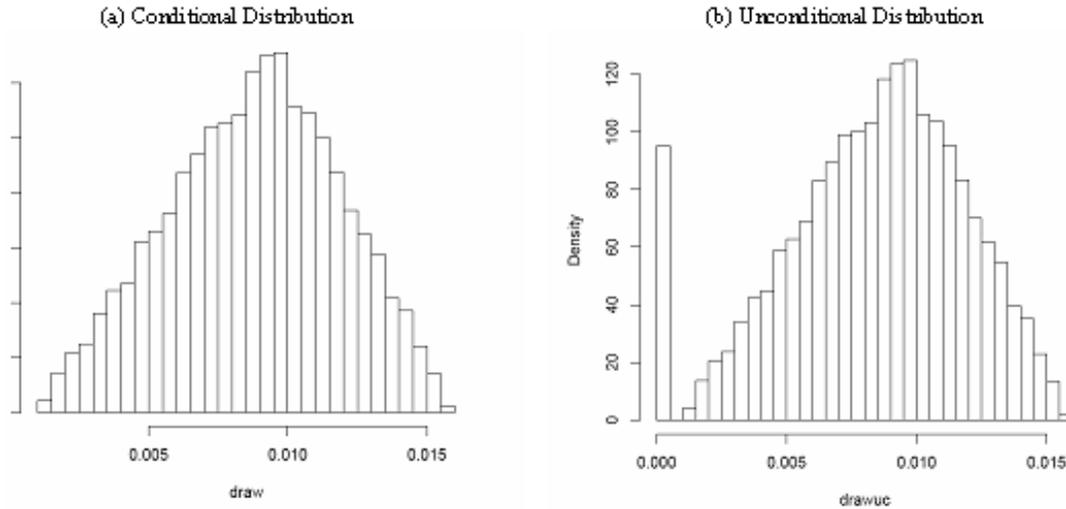


Notes:

Expert C specified a truncated Normal Distribution. We inferred the following values for the parameters of this distribution: mean=1.20 and standard deviation=0.49.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

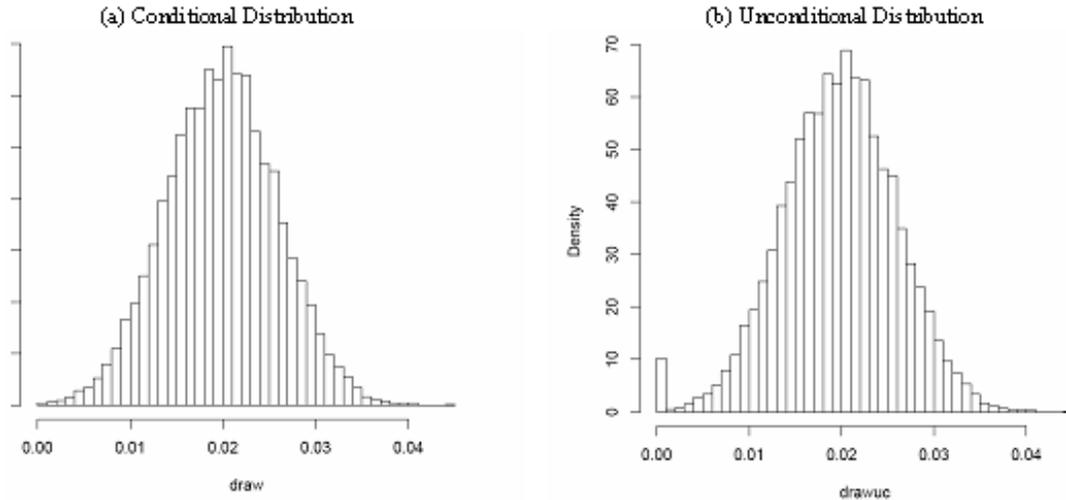
G.1.1.1.4.4 Expert D

Figure G-4. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert D**Notes:**

Expert D specified a Triangular Distribution with minimum=0.1, maximum=1.6, and mode=0.95. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 5 percent zeros to the draw.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

G.1.1.1.4.5 Expert E

Figure G-5. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert E**Notes:**

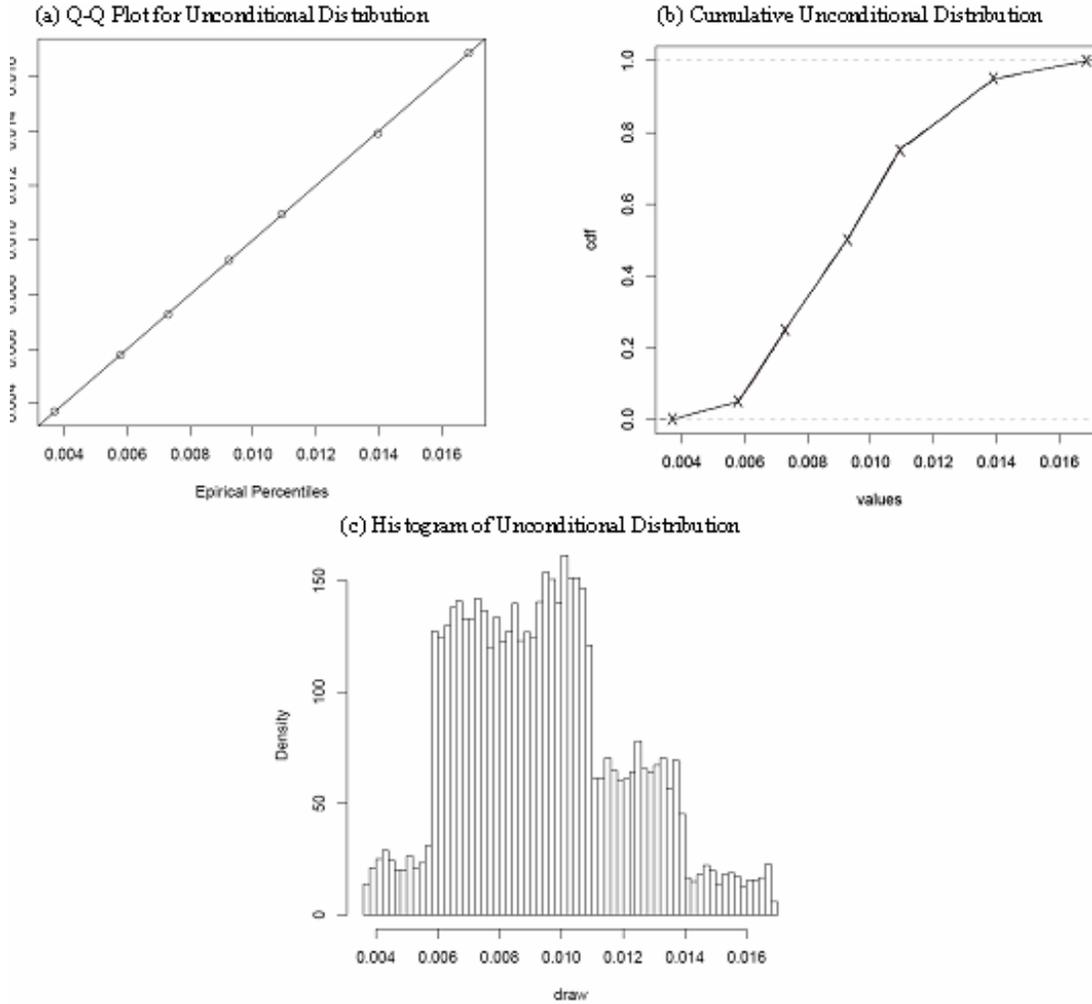
Expert E specified a truncated Normal Distribution. We inferred the following parameters for this distribution: mean=2.00 and standard deviation=0.61. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 1 percent zeros to the draw.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

G.1.1.1.4.6 Expert F

Figure G-6. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert F

(1) Results for the range 4-7 µg/m³

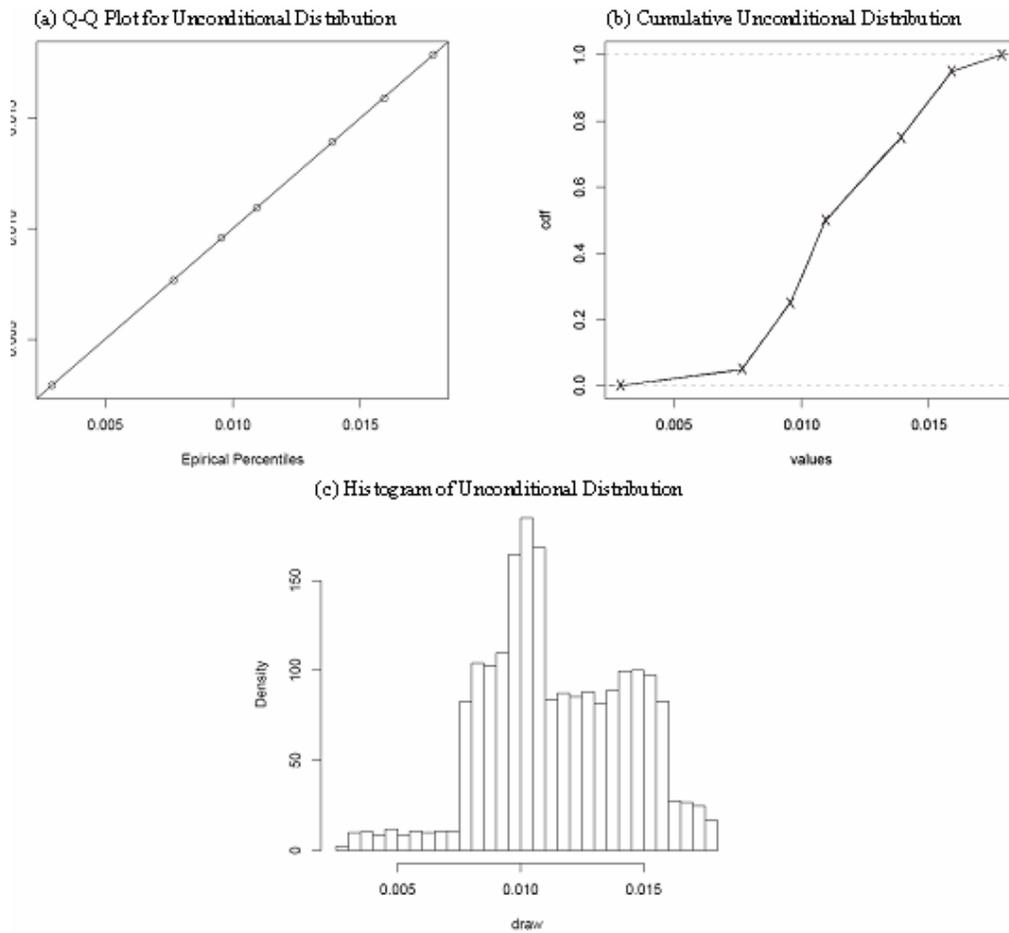


Notes:

Expert F specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. Panel (c) shows the histogram of the distribution.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

Figure G-6. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert F (continued)
(2) Results for the range >7-30 µg/m³



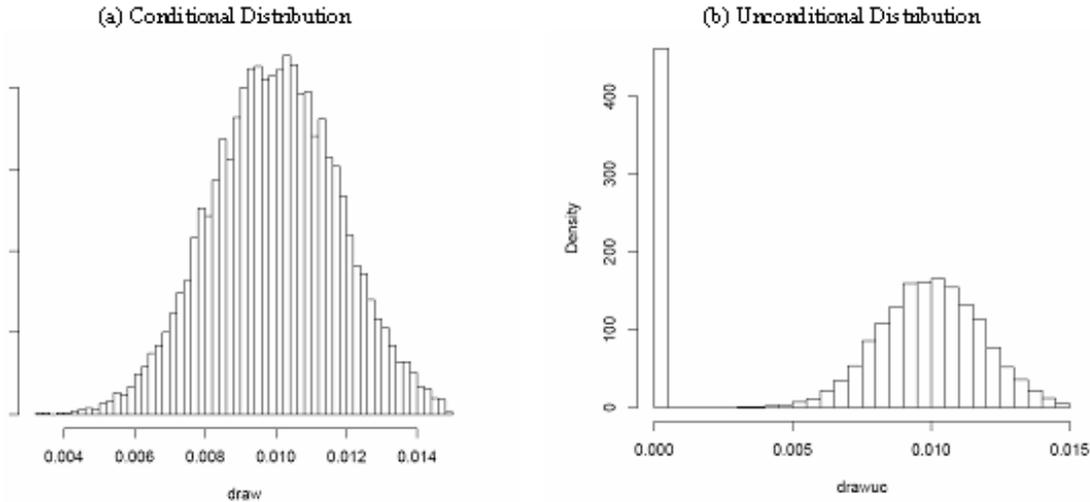
Notes:

Expert F specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. Panel (c) shows the histogram of the distribution.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

G.1.1.1.4.7 Expert G

Figure G-7. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert G

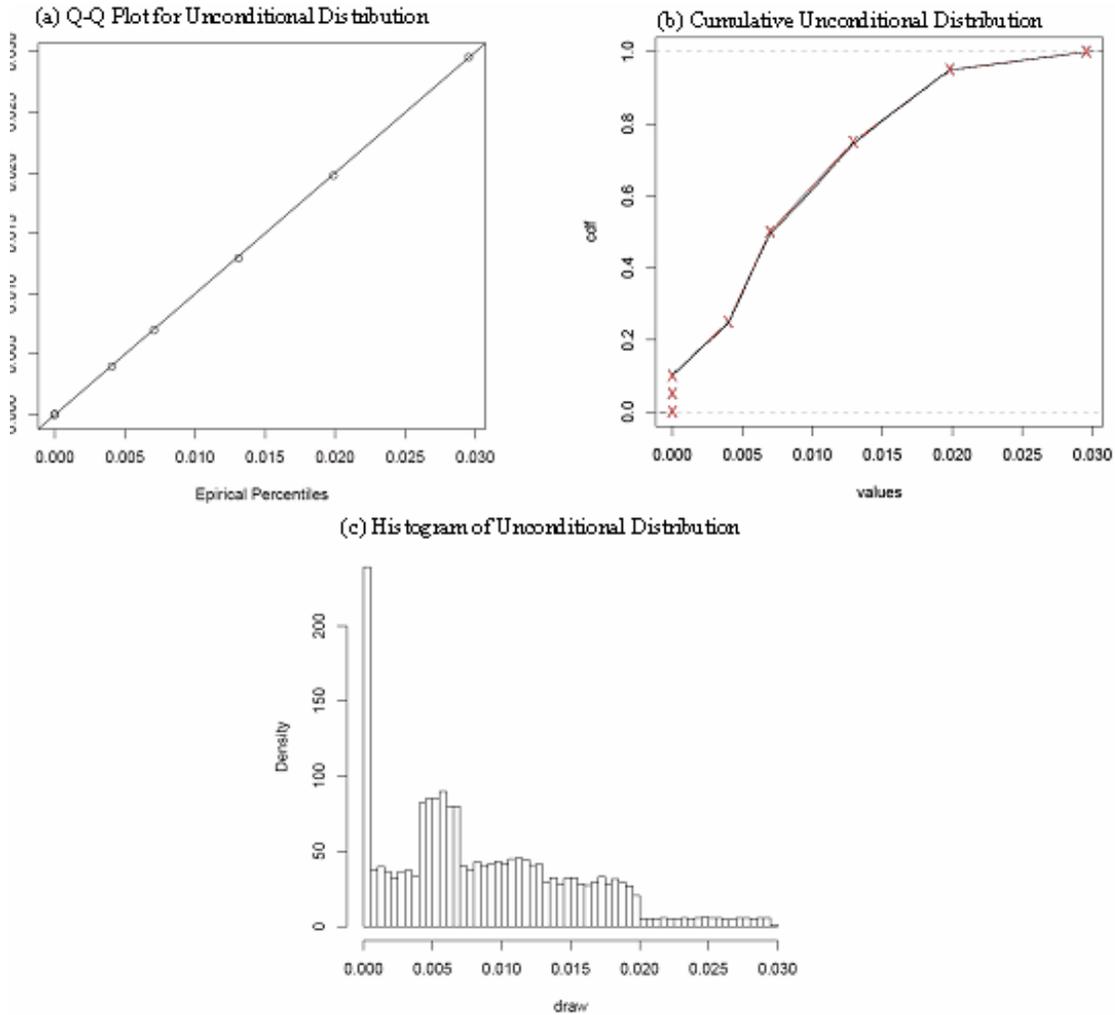


Notes:

Expert G specified a truncated Normal Distribution. We inferred the following parameters for this distribution: mean=1.00 and standard deviation=0.19. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 30 percent zeros to the draw.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

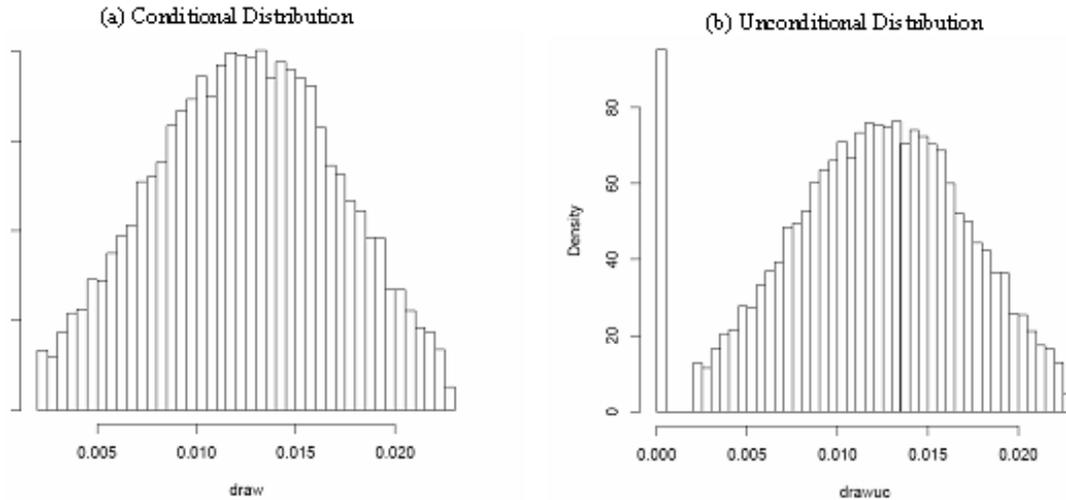
G.1.1.1.4.8 Expert H

Figure G-8. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert H**Notes:**

Expert H specified a non-parametric distribution using six percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. Panel (c) shows the histogram of the distribution.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

G.1.1.1.4.9 Expert I

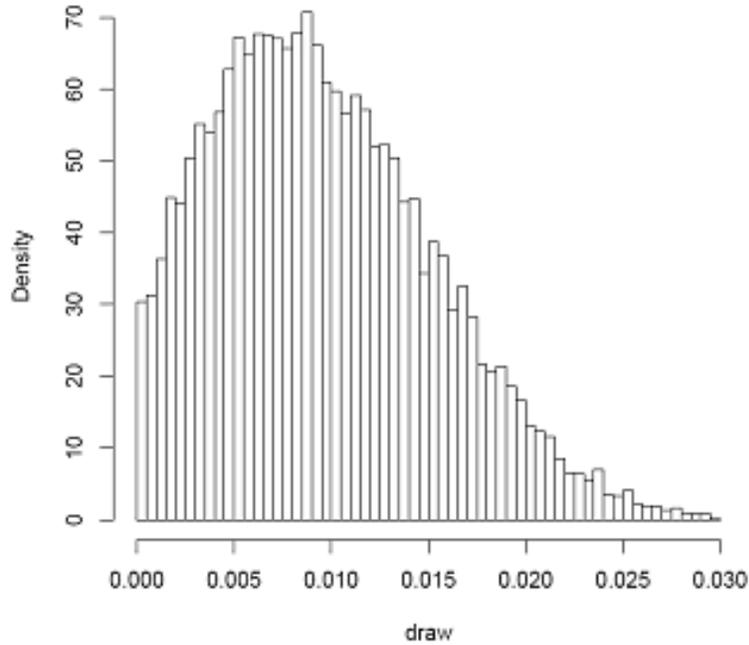
Figure G-9. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert I**Notes:**

Expert I specified a truncated Normal Distribution with mean=1.25 and standard deviation=0.53. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 5 percent zeros to the draw.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

G.1.1.1.4.10 Expert J

Figure G-10. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert J

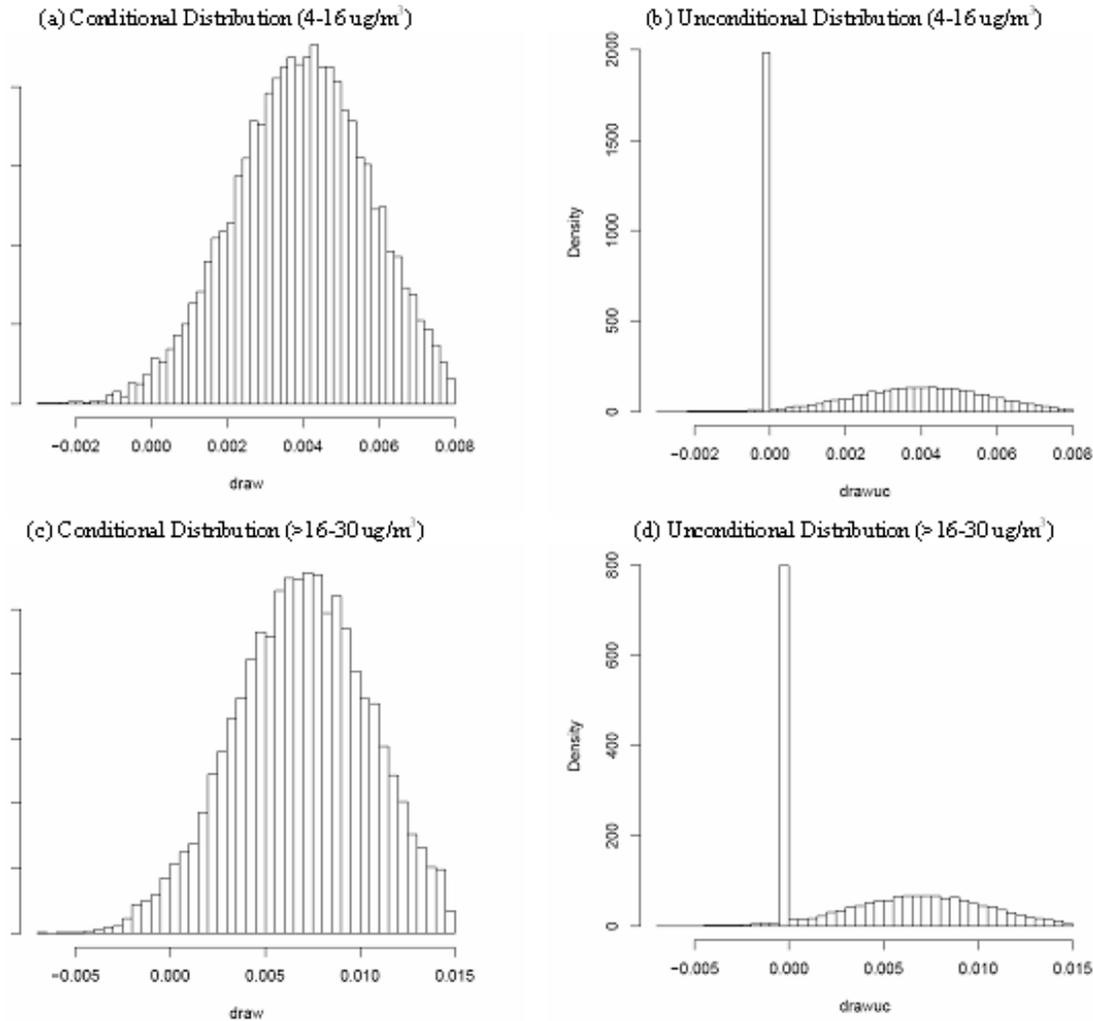


Notes:

Expert J specified a truncated Weibull Distribution. We inferred the following values for the parameters of this distribution: shape=2.21, scale=1.41, and location=-0.33.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

G.1.1.1.4.11 Expert K

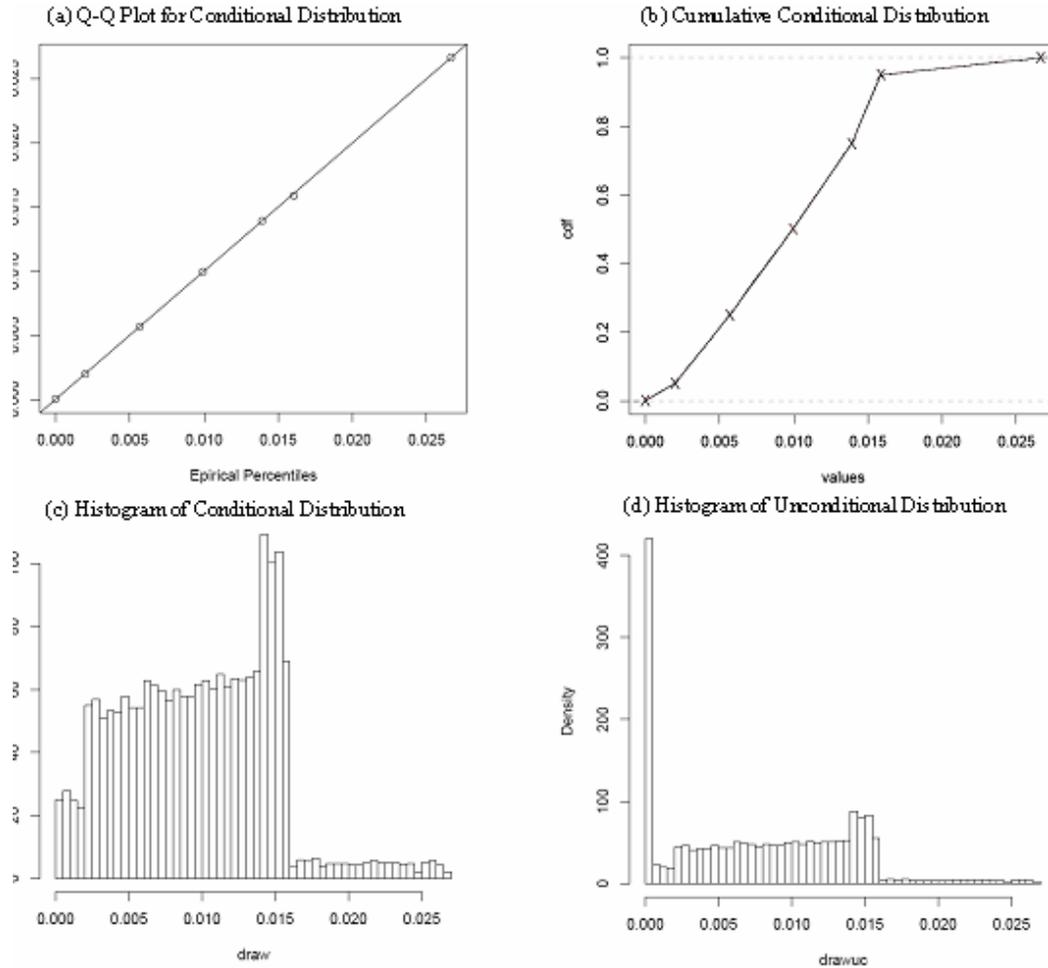
Figure G-11. Histogram of the Random Draw from the Distribution of the PM_{2.5} Effect Specified by Expert K**Notes:**

Expert K specified a truncated Normal Distribution for two ranges separately (4-16 $\mu\text{g}/\text{m}^3$ and >16-30 $\mu\text{g}/\text{m}^3$). We inferred the following parameters for this distribution: mean=0.40 and standard deviation=0.18 in the lower range and mean=0.71 and standard deviation=0.37 in the upper range. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 65 percent zeros to the draws in each range.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

G.1.1.1.4.12 Expert L

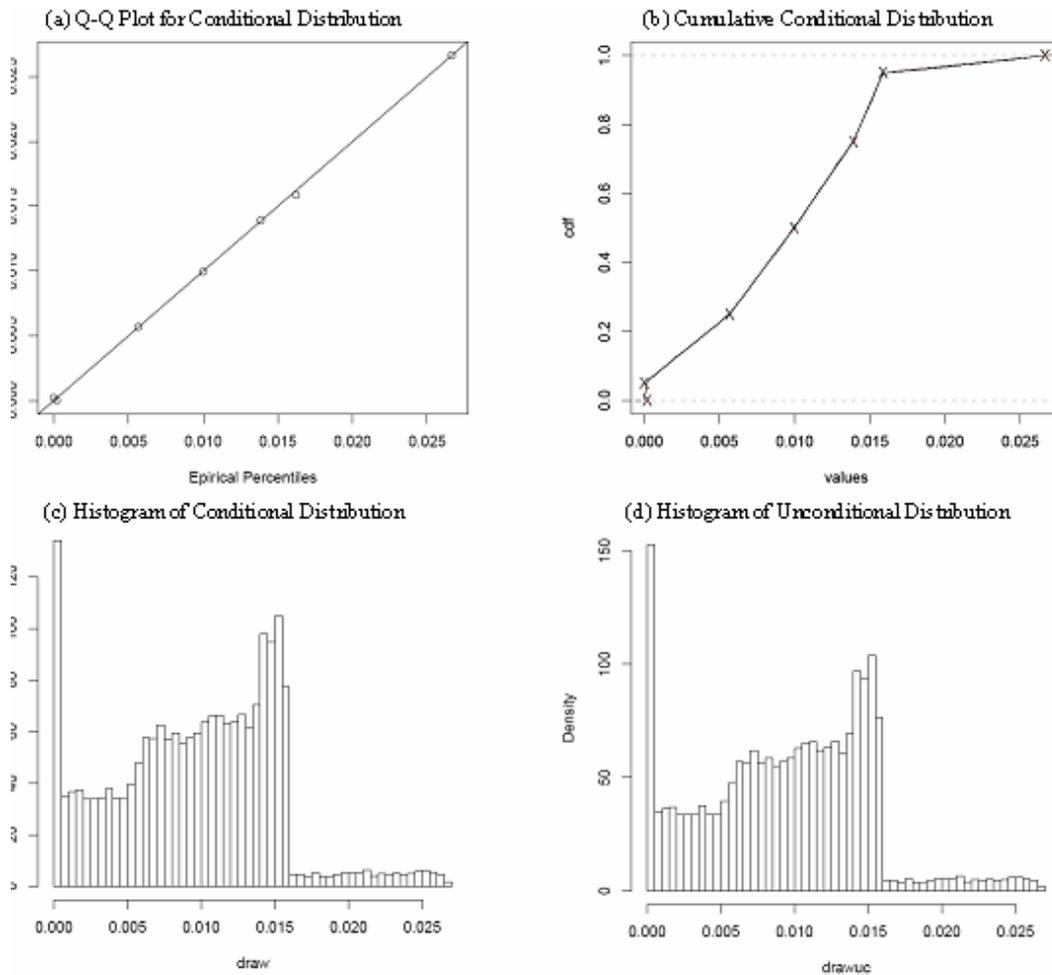
**Figure G-12. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert L
(1) Results for the range 4-10 µg/m³**

**Notes:**

Expert L specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 25 percent zeros to the draw. Panels (c) and (d) show the respective distributions.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk Z - $\log(1+(Z/100))$.

Figure G-12. Characteristics of the Random Draw from the Approximated Distribution of the PM_{2.5} Effect Specified by Expert L (continued)
(2) Results for the range >10-30 µg/m³



Notes:

Expert L specified a non-parametric distribution using five percentile points. We linearly interpolated the CDF between them. Panel (a) shows q-q plot of the expert percentiles and empirical percentiles for the draw. Panel (b) shows empirical CDF associated with the draw, the red “X” marks indicate corresponding expert percentiles. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 1 percent zeros to the draw. Panels (c) and (d) show the respective distributions.

The experts specified distributions for the percent changes in the relative risk. The distribution of the corresponding PM_{2.5} effects was the following transformation of the percent change in relative risk $Z - \log(1+(Z/100))$.

G.1.1.2 Baxter et al. (2017)

Baxter et al. (2017) used a cluster-based approach to evaluate the impact of residential infiltration factors on intercity heterogeneity in short-term PM_{2.5}-mortality associations. The authors performed a city-level time series assessment using mortality data from the National Center for Health Statistics (NCHS). They included 77 U.S. cities in their analysis of 24-hour non-accidental mortality across all ages. PM_{2.5} exposure estimates were based on average daily monitored PM_{2.5} concentrations in each city and the 2-day moving average (lag 0-1 days) of PM_{2.5} concentration was included in the model. The authors utilized Poisson regression models at the city-level and then a meta-regression model to aggregate the city-specific effect estimates into an overall effect estimate. Specifically, in the first stage, the authors ran single-city Poisson time-series models adjusting for temperature and dew point temperature and including variables for previous day temperature, temporal trends, and trends by age. The second stage was a meta-regression with cluster analysis (5 clusters) based on characteristics of residential infiltration.

Short-term Mortality, Non-Accidental

In a single-pollutant model, the coefficient and standard error are estimated from the percent increase in 24-hour non-accidental mortality (0.33%) and 95% confidence intervals (95% CI: 0.13-0.53%) for a 10 µg/m³ increase in daily 24-hour mean PM_{2.5} for a 2-day moving average (lag 0-1 days) (Baxter, et al., 2017, in results section text).

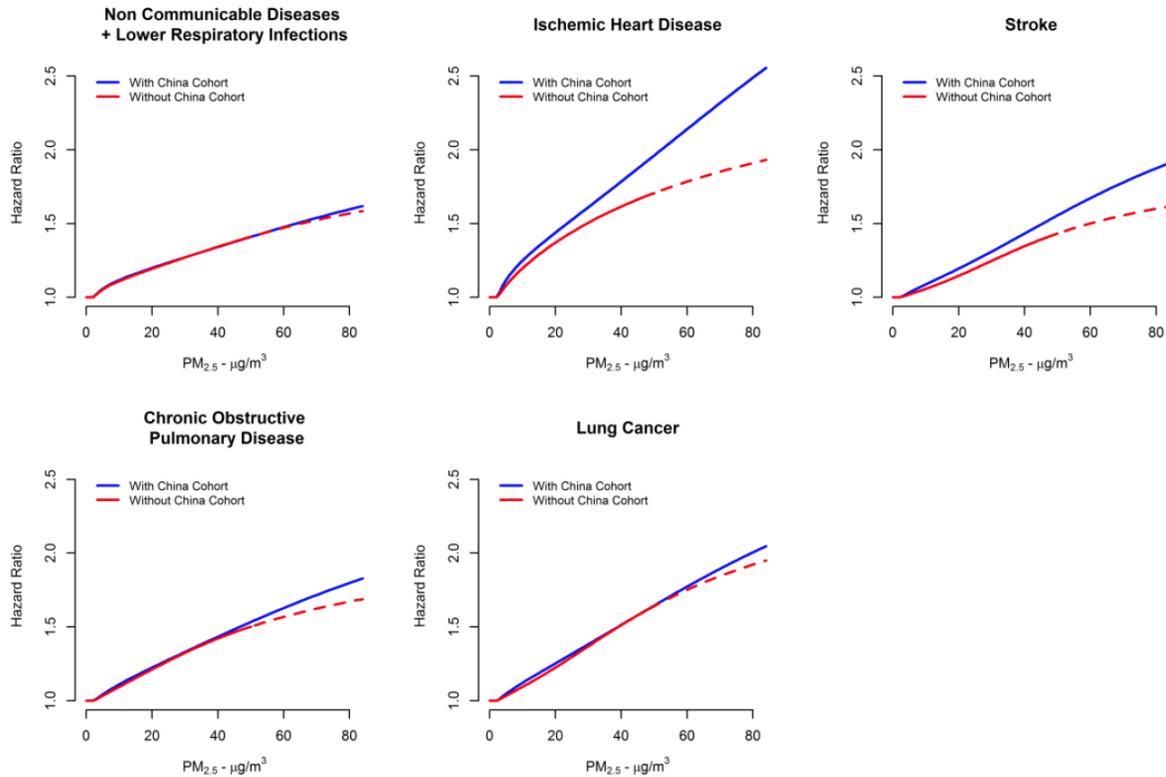
G.1.1.3 Global Exposure Mortality Model (Burnett et al., 2018)

The Global Exposure Mortality Model (GEMM) is a family of functions that can be used to estimate the global burden of disease attributable to ambient fine particle (PM_{2.5}) exposure over the entire global exposure range. The GEMM is the latest iteration of earlier models with the same purpose, such as the Integrated Exposure Response (IER) models (Burnett et al., 2014). The GEMM improves upon these models by excluding studies of non-ambient particle exposure, identifying a single counterfactual concentration (2.4µg/m³), and quantifying all non-accidental mortality related to ambient PM_{2.5} concentrations relative to that level. The GEMM was constructed by combining data from 41 cohort studies in 16 countries that examined the relationship between ambient PM_{2.5} concentrations and mortality. These studies spanned a PM_{2.5} range of 2.4µg/m³ to 83.7µg/m³, which encompasses the 2015 population weighted outdoor exposure levels for 97% of the global population.

The GEMM consists of 83 non-linear relative risk functions that relate changes in ambient PM_{2.5} concentrations to changes in six different mortality endpoints: non-communicable diseases plus lower respiratory infections (NCD+LRI), cerebrovascular disease (CEV), chronic obstructive pulmonary disease (COPD), ischemic heart disease (IHD), lung cancer (LC), and lower respiratory infections (LRI). The functions are applicable to adults age 25-99, and age-specific functions are also provided for NCD+LRI, CEV, and IHD because cardiovascular risk factors, including PM_{2.5}, are known to decline with age. The authors also prepared one set of functions that exclude the

Chinese male cohort study because it had the largest PM_{2.5} range (15.4 µg/m³ to 83.7 µg/m³). The two sets of functions do not differ significantly for NCD+LRI, COPD, and LC; however, excluding the Chinese male cohort yielded lower hazard ratio predictions for CEV and IHD (Figure G-13).

Figure G-13. Figure S6 from Burnett et al., 2018 depicting GEMM hazard ratio predictions for NCD+LRI, IHD, CEV (stroke), COPD), and LC with (blue line) and without Chinese Male Cohort (red line).



Within BenMAP-CE, each GEMM relative risk function is applied to an air quality scenario by first using the applicable GEMM equation to estimate the log-linear beta coefficient for the PM_{2.5} change in each grid cell, and then inserting that beta value into the standard BenMAP-CE formula $(1 - (1/\text{EXP}(\text{Beta} \cdot \text{DELTAQ}))) \cdot \text{Incidence} \cdot \text{POP}$. Additional function logic prevents the GEMM from predicting health benefits below the counterfactual concentration of 2.4 µg/m³. As a result of this linear approximation approach, the calculated BenMAP point estimate(s) are not a perfect reflection of the non-linear GEMM functions. However, this approach greatly reduces the implementation complexity of these functions while maintaining a reasonable level of accuracy, typically within 10% of the non-linear function.

G.1.1.4 Ito et al. (2013)

Ito et al. (2013) used factor analysis to characterize pollution sources and assess the association between short-term PM_{2.5} and PM_{2.5} components with morbidity and mortality outcomes. The authors performed a city-level time series analysis using mortality data from the National Center for Health Statistics (NCHS). They included 150 U.S. cities (and 64 cities in a second analysis) in their assessment of 24-hour all-cause mortality for all ages. PM_{2.5} exposure estimates were based on average daily monitored PM_{2.5} mass data in each city. PM_{2.5} concentrations were expressed in the model as a deviation from the monthly mean to reduce the influence of the seasonal cycles of the pollutants on the overall associations and help focus on the short-term associations. The authors first ran city- and season-specific Poisson generalized linear models and then combined the city-specific estimates using a random effects approach. The model adjusted for temporal trends (annual cycles and influenza epidemics), immediate and delayed temperature, and day-of-week pattern, for entire years (2001-2006) and for warm (April-September) and cold (October-March) seasons. The authors also assessed effect modification using land-use variables and average air pollution levels.

Short-term Mortality, All Cause

Ito et al. (2013) provided regression coefficients and standard errors which are directly used in BenMAP-CE. The selected effect estimate is the lag-1 day (all year) beta of 0.000145 (0.0000747) (Ito, et al., 2013, Appendix G, Table G.6 for Figure 4).

G.1.1.5 Jerrett et al. (2017)

Jerrett et al. (2017) compared mortality effect estimates for PM_{2.5} modeled from remote sensing to those for PM_{2.5} modeled using ground-level information. Multiple exposure estimation approaches were evaluated and the risk assessment used results based on an ensemble approach that incorporated chemical transport modeling, land use data, satellite data, and data from ground-based monitors. This cohort study evaluated long-term PM_{2.5} exposure and mortality from IHD and diseases of the circulatory system in the ACS Cancer Prevention Study II cohort (ages 30+). The authors utilized a Cox proportional hazards model controlling for individual-level confounders which included current and former smoking status as well as smoking duration, amount, age started, second hand cigarette smoke (hours/day exposed), exposure to PM_{2.5} in the workplace for each of the subject's major lifetime occupation, self-reported exposure to dust/fumes at work, marital status, level of education, BMI, alcohol consumption, dietary vegetable/fruit/fiber index, dietary fat index, and missing nutrition information. Ecologic characteristics included median household income, percentage of people with <125% of poverty-level income, percentage of persons >16 who are unemployed, percentage of adults with <12th grade education, and percentage of population who were Black or Hispanic.

Mortality, Ischemic Heart Disease (ICD-10 code I20-I25)

In a single-pollutant model, the coefficient and standard error are estimated from the hazard ratio (1.15) and 95% confidence intervals (95% CI: 1.11-1.19) for a 10 $\mu\text{g}/\text{m}^3$ increase in the ensemble estimate of $\text{PM}_{2.5}$ exposure level from 2002-2004 (Jerrett, et al., 2017, Table 4 IHD). The model was fully adjusted for ecological confounders.

G.1.1.6 Krewski et al. (2009)

This cohort study consists of approximately 360,000 participants residing in areas of the country that have adequate monitoring information on levels of $\text{PM}_{2.5}$ for 1980 and about 500,000 participants in areas with adequate information for 2000. The causes of death that were analyzed included all causes, cardiopulmonary disease (CPD), ischemic heart disease (IHD), lung cancer, and all remaining causes. Data for 44 personal, individual-level covariates, based on participants' answers to a 1982 enrollment questionnaire, were also used for the analyses. The authors also collected data for seven ecologic (neighborhood-level) covariates, each of which represents local factors known or suspected to influence mortality, such as poverty level, level of education, and unemployment (at both Zip Code and city levels). Long-term average exposure variables were constructed for $\text{PM}_{2.5}$ from monitoring data for two periods: 1979-1983 and 1999-2000. Similar variables were constructed for long-term exposure to other pollutants of interest from single-year (1980) averages, including total suspended particles, ozone, nitrogen dioxide, and sulfur dioxide. Exposure was averaged for all monitors within a metropolitan statistical area (MSA) and assigned to participants according to their Zip Code area (ZCA) of residence. The authors chose the standard Cox proportional-hazards model (and a variation to allow for random effects) to calculate hazard ratios for various cause-of-death categories associated with the levels of air pollution exposure in the cohort. They extended the random effects Cox model to accommodate two levels of information for clustering and for ecologic covariates. Three main analyses were conducted: a Nationwide Analysis, Intra-Urban Analyses in the New York City (NYC) and Los Angeles (LA) regions, and an analysis designed to investigate whether critical time windows of exposure to pollutants might have affected mortality in the cohort.

Mortality, All-Cause

In a random effects Cox model, the coefficient and standard error in BenMAP-CE are estimated from the relative risks (1.06) and 95% confidence intervals (95% CI: 1.04-1.08) for a 10 $\mu\text{g}/\text{m}^3$ increase in the average of $\text{PM}_{2.5}$ exposure level for 1999-2000 (Krewski, et al., 2009, Commentary Table 4). The results were adjusted for the 44 individual-level covariates and the 7 ecologic covariates at the MSA & DIFF levels.

Mortality, Ischemic Heart Disease (ICD-10 code I20-I25)

In a random effects Cox model, the coefficient and standard error are estimated from the relative risks (1.24) and 95% confidence intervals (95% CI: 1.19-1.29) for a 10 $\mu\text{g}/\text{m}^3$ increase in the average of $\text{PM}_{2.5}$ exposure level for 1999-2000 (Krewski, et al.,

2009, Commentary Table 4). The results were adjusted for the 44 individual-level covariates and the 7 ecologic covariates at the MSA & DIFF levels.

Mortality, Lung Cancer (ICD-10 code C30-C39)

In a random effects Cox model, the coefficient and standard error are estimated from the relative risks (1.14) and 95% confidence intervals (95% CI: 1.06-1.23) for a 10 $\mu\text{g}/\text{m}^3$ increase in the average of $\text{PM}_{2.5}$ exposure level for 1999-2000 (Krewski, et al., 2009, Commentary Table 4). The results were adjusted for the 44 individual-level covariates and the 7 ecologic covariates at the MSA & DIFF levels.

G.1.1.7 Laden et al. (2006)

A large body of epidemiologic literature has found an association of increased fine particulate air pollution ($\text{PM}_{2.5}$) with acute and chronic mortality. The effect of improvements in particle exposure is less clear. Earlier analysis of the Harvard Six Cities adult cohort study showed an association between long-term ambient $\text{PM}_{2.5}$ and mortality between enrollment in the mid-1970's and follow-up until 1990. The authors extended mortality follow-up for eight years in a period of reduced air pollution concentrations. Annual city-specific $\text{PM}_{2.5}$ concentrations were measured between 1979-1988, and estimated for later years from publicly available data. Exposure was defined as (1) city-specific mean $\text{PM}_{2.5}$ during the two follow-up periods, (2) mean $\text{PM}_{2.5}$ in the first period and change between these periods, (3) overall mean $\text{PM}_{2.5}$ across the entire follow-up, and (4) year-specific mean $\text{PM}_{2.5}$. Mortality rate ratios were estimated with Cox proportional hazards regression controlling for individual risk factors. The authors found an increase in overall mortality associated with each 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ modeled either as the overall mean (RR=1.16, 95%CI=1.07-1.26) or as exposure in the year of death (RR=1.14, 95%CI=1.06-1.22). $\text{PM}_{2.5}$ exposure was associated with lung cancer (RR=1.27, 95%CI=0.96-1.69) and cardiovascular deaths (RR=1.28, 95%CI=1.13-1.44). Improved overall mortality was associated with decreased mean $\text{PM}_{2.5}$ (10 microg/m³) between periods (RR=0.73, 95% CI=0.57-0.95). Total, cardiovascular, and lung cancer mortality were each positively associated with ambient $\text{PM}_{2.5}$ concentrations. Reduced $\text{PM}_{2.5}$ concentrations were associated with reduced mortality risk.

All-Cause Mortality

The coefficient and standard error for $\text{PM}_{2.5}$ are estimated from the relative risk (1.16) and 95% confidence interval of (1.07-1.26) associated with a change in annual mean exposure of 10.0 $\mu\text{g}/\text{m}^3$ (Laden et al., 2006, p. 667).

G.1.1.8 Lepeule et al. (2012)

Lepeule et al. (2012) evaluated the sensitivity of previous Six Cities results to model specifications, lower exposures, and averaging time using eleven additional years of cohort follow-up that incorporated recent lower exposures. The authors found significant associations between $\text{PM}_{2.5}$ exposure and increased risk of all-cause, cardiovascular and lung cancer mortality. The authors also concluded that the

concentration-response relationship was linear down to PM_{2.5} concentrations of 8 µg/m³, and that mortality rate ratios for PM_{2.5} fluctuated over time, but without clear trends, despite a substantial drop in the sulfate fraction.

G.1.1.9 Pope et al. (2002)

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

They used a Cox proportional hazard model to estimate the impact of long-term PM exposure using three alternative measures of PM_{2.5} exposure; metropolitan area-wide annual mean PM levels from the beginning of tracking period (1979-1983 PM data, conducted for 61 metropolitan areas with 359,000 individuals), annual mean PM from the end of the tracking period (1999-2000, for 116 areas with 500,000 individuals), and the average annual mean PM levels of the two periods (for 51 metropolitan areas, with 319,000 individuals). PM levels were lower in 1999-2000 than in 1979-1983 in most cities, with the largest improvements occurring in cities with the highest original levels.

Pope et al. (2002) followed Krewski et al. (2000) and Pope et al. (1995, Table 2) and reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and “all other” deaths. All-cause mortality includes accidents, suicides, homicides and legal interventions. The category “all other” deaths is all-cause mortality less lung cancer and cardiopulmonary deaths. Like the earlier studies, Pope et al. (2002) found that mean PM_{2.5} is significantly related to all-cause and cardiopulmonary mortality. In addition, Pope et al. (2002) found a significant relationship with lung cancer mortality, which was not found in the earlier studies. None of the three studies found a significant relationship with “all other” deaths.

Pope et al. (2002) obtained ambient data on gaseous pollutants routinely monitored by EPA during the 1982-1998 observation period, including SO₂, NO₂, CO, and ozone. They did not find significant relationships between NO₂, CO, and ozone and premature mortality, but there were significant relationships between SO₄ (as well as SO₂), and all-cause, cardiopulmonary, lung cancer and “all other” mortality.

All-Cause Mortality, 1979-1983 Exposure

The coefficient and standard error for PM_{2.5} using the 1979-1983 PM data are estimated from the relative risk (1.04) and 95% confidence interval (1.01-1.08) associated with a change in annual mean exposure of 10.0 µg/m³ (Pope et al., 2002, Table 2).

All-Cause Mortality, Average of 1979-1983 and 1999-2000 Exposure

The coefficient and standard error for PM_{2.5} using the average of 1979-1983 and 1999-2000 PM data are estimated from the relative risk (1.06) and 95% confidence interval (1.02-1.11) associated with a change in annual mean exposure of 10.0 µg/m³ (Pope et al., 2002, Table 2).

G.1.1.10 Pope et al. (2015)

This cohort study 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 and above). PM_{2.5} exposures were estimated at home addresses based on a land use regression and Bayesian maximum entropy (LUR-BME) interpolation model that incorporated data from ground-based monitors. 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, Ischemic Heart Disease (ICD-10 code I20-I25)

In a single-pollutant model, the coefficient and standard error are estimated from the hazard ratio (1.14) and 95% confidence intervals (95% CI: 1.10-1.18) for a 10 µg/m³ 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).

G.1.1.11 Thurston et al. (2016)

Thurston et al. (2016) reevaluated the relationship between long-term exposure to ambient PM_{2.5} and mortality given the recent decline in U.S. ambient PM concentrations and differentiated risk for fossil fuel PM_{2.5} versus total PM_{2.5}. This cohort study analyzed all-cause, CVD, and respiratory mortality in the NIH-AARP cohort (ages 55-85) in 100 metropolitan areas in CA, GA, FL, LA, MI, NC, NJ, and PA. PM_{2.5} exposures were estimated at census tract centroids based on land use data and U.S. EPA AQS ground-based monitors. Participant exposure was estimated at census-tract of residence and included annual mean concentration in the year of mortality and 1-year lag average in a time-dependent model. Thurston et al. utilized a Cox proportional hazards model adjusted for race, education, marital status, BMI, alcohol consumption, smoking history,

and contextual variables such as median household income and percentage of population with less than a high school education.

Mortality, All-Cause

In a single-pollutant model, the coefficient and standard error are estimated from the hazard ratio (1.03) and 95% confidence intervals (95% CI: 1.00-1.05) for a 10 $\mu\text{g}/\text{m}^3$ increase in the annual mean $\text{PM}_{2.5}$ exposure level (Thurston, et al., 2016, Table 2. NIH-AARP cohort time independent Cox model $\text{PM}_{2.5}$ mortality hazard ratios (and 95th percentile CI) per 10 $\mu\text{g}/\text{m}^3$, by cause and cohort subgroup; cohort: ALL).

G.1.1.12 Turner et al. (2016)

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

Mortality, Lung Cancer

In a single-pollutant model, the coefficient and standard error are estimated from the hazard ratio (1.09) and 95% confidence intervals (95% CI: 1.03-1.16) for a 10 $\mu\text{g}/\text{m}^3$ increase in the mean $\text{PM}_{2.5}$ exposure level from 1999-2004 (Turner, et al., 2016, Table E4: Lung cancer, fully-adjusted HR, LURBME $\text{PM}_{2.5}$).

G.1.1.13 Woodruff et al. (1997)

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

Post-Neonatal Mortality

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

G.1.1.14 Woodruff et al. (2006)

Studies suggest that airborne particulate matter (PM) may be associated with postneonatal infant mortality, particularly with respiratory causes and sudden infant death syndrome (SIDS). To further explore this issue, the authors examined the relationship between long-term exposure to fine PM air pollution and postneonatal infant mortality in California. They linked monitoring data for $\text{PM}_{2.5}$ to infants born in California in 1999 and 2000 using maternal addresses for mothers who lived within 5 miles of a $\text{PM}_{2.5}$ monitor. They matched each postneonatal infant death to four infants surviving to 1 year of age, by birth weight category and date of birth (within 2 weeks). For each matched set, they calculated exposure as the average $\text{PM}_{2.5}$ concentration over the period of life for the infant who died. They used conditional logistic regression to

estimate the odds of postneonatal all-cause, respiratory-related, SIDS, and external-cause (a control category) mortality by exposure to PM_{2.5}, controlling for the matched sets and maternal demographic factors. They matched 788 postneonatal infant deaths to 3,089 infant survivors, with 51 and 120 postneonatal deaths due to respiratory causes and SIDS, respectively. They found an adjusted odds ratio for a 10-microg/m³ increase in PM_{2.5} of 1.07 [95% confidence interval (CI), 0.93-1.24] for overall postneonatal mortality, 2.13 (95% CI, 1.12-4.05) for respiratory-related postneonatal mortality, 0.82 (95% CI, 0.55-1.23) for SIDS, and 0.83 (95% CI, 0.50-1.39) for external causes.

Post-Neonatal Mortality

The coefficient and standard error for PM_{2.5} are estimated from the relative risk (1.07) and the 95% confidence interval (0.93-1.24) associated with a change in annual mean exposure of 10.0 µg/m³ (Woodruff et al., 2006, p. 786).

G.1.1.15 Zanobetti et al. (2014)

Zanobetti et al. (2014) estimated the effect of short-term exposure to PM_{2.5} on all-cause mortality and also assessed the potential for pre-existing diseases (neurological disorders and diabetes) to modify the association between PM_{2.5} and mortality. This case-crossover analysis was a community-level assessment (community defined as the county or contiguous counties encompassing a city's population) of a Medicare cohort (age 65+) in 121 U.S. communities. PM_{2.5} exposures were estimated based on data from U.S. EPA AQS ground-based monitors. Daily PM_{2.5} data available for various monitors were averaged over the communities and participants were assigned a 2-day moving average (lag 0 and 1) based on community of residence. Zanobetti et al. (2014) first performed logistic regression at the community level, controlling for confounders such as the average temperature for the same and previous day. In a second stage of analysis, the community specific results were combined using multivariate meta-analysis techniques.

Short-term Mortality, All-Cause

In a single-pollutant model, the coefficient and standard error are estimated from the percent increase (0.64%) and 95% confidence intervals (95% CI: 0.42-0.85%) for a 10 µg/m³ increase in the 2-day average PM_{2.5} concentration (Zanobetti, et al., 2014, Table 2. Percent increase for 10 µg/m³ increase in the two days average PM_{2.5}: Combined across the 121 communities).

G.1.2 Chronic/Severe Illness

Table G-6 below summarizes the additional health impacts functions used to estimate the relationship between PM_{2.5} and chronic / severe health effects. We present a brief summary of each of the studies and any items that are unique to the study.

Table G-6. Additional Health Impact Functions for Particulate Matter and Chronic Illness

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Chronic Bronchitis	Abbey et al.	1995	SF, SD, South Coast Air Basin	27-99		Annual	0.013185	0.006796	Logistic	
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.

G.1.2.1 Abbey et al. (1995b)

Abbey et al. (1995b) examined the relationship between estimated PM_{2.5} (annual mean from 1966 to 1977), PM₁₀ (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant PM_{2.5} relationship with development of chronic bronchitis, but not for AOD or asthma; PM₁₀ was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

Chronic Bronchitis

The estimated coefficient (0.0137) is presented for a one µg/m³ change in PM_{2.5} (Abbey et al., 1995b, Table 2). The standard error is calculated from the reported relative risk (1.81) and 95% confidence interval (0.98-3.25) for a 45 µg/m³ change in PM_{2.5}.

Incidence Rate: annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378

Population: population of ages 27 and older without chronic bronchitis = 95.57% of population 27+. Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95. The American Lung Association (2010a, Table 4) reports a chronic bronchitis prevalence rate for ages 18 and over of 4.37%.

G.1.2.2 Peters et al. (2001)

See full study description under Peters et al. (2001) in Appendix E, Section E.2.5.

G.1.2.3 Pope et al. (2006)

See full study description under Pope et al. (2006) in Appendix E, Section E.2.6.

G.1.2.4 Sullivan et al. (2005)

See full study description under Sullivan et al. (2005) in Appendix E, Section E.2.9.

G.1.2.5 Zanobetti and Schwartz (2006)

See full study description under Zanobetti and Schwartz (2006) in Appendix E, Section E.2.10.

G.1.2.6 Zanobetti et al. (2009)

See full study description under Zanobetti et al. (2009) in Appendix E, Section E.2.11.

G.1.3 Hospitalizations

Table G-7 summarizes the additional 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 G-7. Additional Health Impact Functions for Particulate Matter and Hospital Admissions

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Asthma	Babin et al.	2007	Washington, DC	0-17		D24HourMean	0.0020	0.0043	Log-linear	Age range adjusted Admission from emergency department only.
All Cardiovascular (less Myocardial Infarctions)	Bell et al.	2008	202 U.S. Counties	65-99		D24HourMean	0.0008	0.0001	Log-linear	Urgent admission only. Yearly national estimates
All Cardiovascular (less Myocardial Infarctions)	Bell et al.	2012	187 U.S. Counties	65-99		D24HourMean	0.0008	0.0001071	Log-linear	
Chronic Lung	Ito	2003	Detroit, MI	65-99		D24HourMean	0.001169	0.002064	Log-linear	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Congestive Heart Failure	Ito	2003	Detroit, MI	65-99		D24HourMean	0.003074	0.001292	Log-linear	
Dysrhythmia	Ito	2003	Detroit, MI	65-99		D24HourMean	0.001249	0.002033	Log-linear	
Ischemic Heart (less Myocardial Infarctions)	Ito	2003	Detroit, MI	65-99		D24HourMean	0.001435	0.001156	Log-linear	
Pneumonia	Ito	2003	Detroit, MI	65-99		D24HourMean	0.003979	0.001659	Log-linear	
All Respiratory	Kloog et al.	2012	New England	65-99		D24HourMean	0.0007	0.0010	Log-linear	
Chronic Lung (less Asthma)	Moolgavkar	2000	Los Angeles, CA	18-64		D24HourMean	0.002200	0.000733	Log-linear	
All Cardiovascular (less Myocardial Infarctions)	Moolgavkar	2000	Los Angeles, CA	18-64		D24HourMean	0.001400	0.000341	Log-linear	
Chronic Lung	Moolgavkar	2003	Los Angeles, CA	65-99		D24HourMean	0.001850	0.000524	Log-linear	
All Cardiovascular (less Myocardial Infarctions)	Moolgavkar	2003	Los Angeles, CA	65-99		D24HourMean	0.001580	0.000344	Log-linear	
All Cardiovascular (less Myocardial Infarctions)	Peng et al.	2008	108 U.S. Counties	65-99		D24HourMean	0.0007	0.0001	Log-linear	Emergency HA
All Cardiovascular (less Myocardial Infarctions)	Peng et al.	2009	119 U.S. Counties	65-99		D24HourMean	0.0007	0.0002	Log-linear	Urgent or emergency HA
Asthma	Sheppard	2003	Seattle, WA	0-64		D24HourMean	0.003324	0.001045	Log-linear	
All Cardiovascular (less Myocardial Infarctions)	Zanobetti et al.	2009	26 U.S. Communities	65-99		D24HourMean	0.00019	0.0003	Log-linear	All seasons
All Respiratory	Zanobetti et al.	2009	26 U.S. Communities	65-99		D24HourMean	0.0021	0.0004	Log-linear	All seasons

G.1.3.1 Babin et al. (2007)

Babin et al. (2007) examined pediatric asthma-related emergency room (ER) visits and hospital admissions (ICD-9 code 493) in Washington, D.C. from 2001-2004 and their short-term associations with ozone, particulate matter, socioeconomic status, and age group. The association between PM_{2.5} and asthma hospitalization was found statistically insignificant.

Hospital Admissions, Asthma (ICD-9 code 493)

In a single-pollutant model, the coefficient and standard error are estimated from the average percent increase in risk (0.2%) and 95% confidence interval (95% CI: -0.6% - 1.1%) for a 1 µg/m³ increase in same-day daily 24-hour mean PM_{2.5} (Babin et al., 2007, Table 2).

Note that although Babin et al. (2007) reports results for the 1-17 year old age range, for comparability to other studies, we apply the results to the population of ages 0 to 17.

G.1.3.2 Bell et al. (2008, 2012)

Bell et al. (2008) evaluated the association between short-term exposure to PM_{2.5} and the risk of cardiovascular (ICD-9 codes 410-414, 26-427, 428, 429, 430-438, and 440-449) and respiratory (ICD-9 codes 464-466, 480-487, and 490-492) hospital admissions among Medicare enrollees ≥ 65 years old varied by season and geographic region in 202 U.S. counties with populations greater than 200,000 from 1999-2005. Three time-series models were used to provide three key variables: consistent PM effects across the year, different PM effects by season, and smoothly varying PM effects throughout the year. A two-stage Bayesian hierarchical model was used to estimate the association between PM_{2.5} and hospitalization rates, with the first stage estimating the association within a single county and the second stage combining county-specific estimates. The authors found statistically significant evidence of seasonal and regional variation. Respiratory hospitalizations were highest in winter with a 1.05% increase (95%PI: 0.29-1.82) in hospitalizations per 10 $\mu\text{g}/\text{m}^3$ increase in same-day PM_{2.5}. A 1.49% increase (95% PI: 1.09-1.89) in cardiovascular hospital admissions were also found for the winter season, and associations were observed in other seasons as well. The strongest association was for the northeast for both respiratory and cardiovascular admissions.

Hospital Admissions, Cardio-, Cerebro- and Peripheral Vascular Disease (ICD-9 codes 426-427, 428, 430-438, 410-414, 429; 440-449)

For different seasons (i.e., autumn, spring, summer, winter, and all-year) and regions (i.e., southwest, northwest, southeast, southwest, and nationwide), the coefficient and standard error are estimated from the average percent increase in risk and 95% confidence interval for a 10 $\mu\text{g}/\text{m}^3$ increase in same-day (lag 0) daily 24-hour mean PM_{2.5} (Bell et al., 2008, Table 2).

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

G.1.3.3 Ito (2003)

Lippmann et al. (2000) studied the association between particulate matter and daily mortality and hospitalizations among the elderly in Detroit, MI. Data were analyzed for two separate study periods, 1985-1990 and 1992-1994. The 1992-1994 study period had a greater variety of data on PM size and was the main focus of the report. The authors collected hospitalization data for a variety of cardiovascular and respiratory endpoints. They used daily air quality data for PM₁₀, PM_{2.5}, and PM_{10-2.5} in a Poisson regression model with generalized additive models (GAM) to adjust for nonlinear

relationships and temporal trends. In single pollutant models, all PM metrics were statistically significant for pneumonia (ICD codes 480-486), PM_{10-2.5} and PM₁₀ were significant for ischemic heart disease (ICD code 410-414), and PM_{2.5} and PM₁₀ were significant for heart failure (ICD code 428). There were positive, but not statistically significant associations, between the PM metrics and COPD (ICD codes 490-496) and dysrhythmia (ICD code 427). In separate co-pollutant models with PM and either ozone, SO₂, NO₂, or CO, the results were generally comparable.

In response to concerns with the Splus issue, Ito (2003) reanalyzed the study by Lippmann et al. (2000). The reanalysis by Ito reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

Chronic Lung Disease (ICD-9 codes 490-496)

The coefficient and standard error are based on the relative risk (1.043) and 95% confidence interval (0.902-1.207) for a 36 µg/m³ increase in PM_{2.5} in the 3-day lag GAM stringent model (Ito, 2003, Table 8).

Congestive Heart Failure (ICD-9 code 428)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.117 (95% CI 1.020-1.224) for a 36 µg/m³ increase in PM_{2.5} in the 1-day lag GAM stringent model (Ito, 2003, Table 11).

Dysrhythmia (ICD-9 code 427)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.046 (95% CI 0.906-1.207) for a 36 µg/m³ increase in PM_{2.5} in the 1-day lag GAM stringent model (Ito, 2003, Table 10).

Ischemic Heart Disease (ICD-9 codes 411-414)

The co-pollutant coefficient and standard error are calculated from a relative risk of 1.053 (95% CI 0.971-1.143) for a 36 µg/m³ increase in PM_{2.5} in the 1-day lag GAM stringent model (Ito, 2003, Table 9). Note that Lippmann et al. (2000) report results for ICD codes 410-414. In the benefit analysis, avoided nonfatal heart attacks are estimated using the results reported by Peters et al. (2001). The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

Pneumonia (ICD-9 codes 480-487)

The estimated PM_{2.5} coefficient and standard error are based on a relative risk of 1.154 (95% CI -1.027, 1.298) due to a PM_{2.5} change of 36 µg/m³ in the 1-day lag GAM stringent model (Ito, 2003, Table 7).

G.1.3.4 Kloog et al. (2012)

Kloog et al. (2012) examined the relationship between both short-term and long-term PM_{2.5} exposure and emergent hospital admissions for respiratory (ICD-9 codes 460-519) and cardiovascular (ICD-9 codes 390-429) diseases in the New-England states of Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island and Vermont. The study used hospital admission data from the U.S. Medicare program for only patients that were at least 65 years old. Short-term exposure models looked at the mean 24-hour PM_{2.5} concentration on the day of a patient's hospital admission, while long-term exposure was defined to be the mean PM_{2.5} concentration over the entire study period (2000-2006). Results of the study showed an association between PM_{2.5} and hospital admissions for all disease types in both the short and long term.

Hospital Admissions, Respiratory (ICD Codes 460-519)

In a single pollutant model for patients over the age of 65, the coefficient and standard error were estimated from the percent change (0.70%) and 95% confidence interval (0.35%-1.05%) for a 10 µg/m³ increase in same day (0 lag) 24-hour mean PM_{2.5} concentration.

G.1.3.5 Moolgavkar (2000a)

Moolgavkar (2000a) examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO₂, NO₂, CO, and PM₁₀ in all three areas. PM_{2.5} data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0- to 5-day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM₁₀. In Los Angeles, marginally significant associations were observed for PM_{2.5}, which were generally lower than for the gases. In co-pollutant models with CO, the PM_{2.5} effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

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

Hospital Admissions, Chronic Lung Disease Less Asthma (ICD-9 codes 490-492, 494-496)

In a model with CO, the coefficient and standard error are calculated from an estimated percent change of 2.0 and t-statistic of 2.2 for a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} in the two-day lag model (Moolgavkar, 2000a, Table 4, p. 81). In a log-linear model, the percent change is equal to $(\text{RR} - 1) * 100$.

In this study, Moolgavkar defines and reports the “estimated” percent change as $(\log \text{RR} * 100)$. Because the relative risk is close to 1, RR-1 and log RR are essentially the same. For example, a true percent change of 2.0 would result in a relative risk of 1.020 and coefficient of 0.001980. The “estimated” percent change, as reported by Moolgavkar, of 2.0 results in a relative risk of 1.020201 and coefficient of 0.002.

Note that although Moolgavkar (2000a) reports results for the 20-64 year old age range, for comparability to other studies, we apply the results to the population of ages 18 to 64. Note also that in order to avoid double counting non-elderly asthma hospitalizations (ICD code 493), which are typically estimated separately in EPA benefit analyses, we have excluded ICD code 493 from the baseline incidence rate used in this function.

G.1.3.6 Moolgavkar (2000b), Cardiovascular

Moolgavkar (2000b) examined the association between air pollution and cardiovascular hospital admissions (ICD 390-429) in the Chicago, Los Angeles, and Phoenix metropolitan areas. He collected daily air pollution data for ozone, SO₂, NO₂, CO, and PM₁₀ in all three areas. PM_{2.5} data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM₁₀ or PM_{2.5}. The strongest overall effects were observed for SO₂ and CO. In a single pollutant model, PM_{2.5} was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the PM_{2.5} effect dropped out and CO remained significant. For ages 20-64, SO₂ and CO exhibited the strongest effect and any PM_{2.5} effect dropped out in co-pollutant models with CO.

Hospital Admissions, All Cardiovascular (ICD codes 390-409, 411-429)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.4 and t-statistic of 4.1 for a 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} in the zero lag model (Moolgavkar, 2000b, Table 4, p. 1203).

Note that (Moolgavkar (2000b) report results that include ICD code 410 (heart attack). In a benefit analysis, avoided nonfatal heart attacks are typically estimated separately. The baseline rate in the Peters et al. function is a modified heart attack hospitalization rate (ICD code 410), since most, if not all, nonfatal heart attacks will require

hospitalization. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

G.1.3.7 Moolgavkar (2003)

Moolgavkar (2000a) examined the association between air pollution and COPD hospital admissions (ICD 490-496) in the Chicago, Los Angeles, and Phoenix metropolitan areas. In response to concerns with Splus issue, Moolgavkar (2003) reanalyzed his earlier studies. In the reanalysis, he reported that more generalized additive models with stringent convergence criteria and generalized linear models resulted in smaller relative risk estimates.

Hospital Admissions, Chronic Lung (ICD-9 codes 490-496)

The coefficient and standard error are calculated from an estimated percentage change of 1.85 and a t-statistic of 3.53 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 2-day lag GAM-30df stringent (10-8) model (Moolgavkar, 2003, Table 17). In a log-linear model, the percent change is equal to $(\text{RR} - 1) * 100$.

The $\text{PM}_{2.5}$ C-R functions for the 65+ age group are based on the reanalysis in Moolgavkar (2003) of the single and co-pollutant models ($\text{PM}_{2.5}$ and CO). The true PM effect is most likely best represented by a distributed lag model, then any single lag model should underestimate the total PM effect. As a result, we selected the lag models with the greatest effect estimates for use in the C-R functions.

Hospital Admissions, All Cardiovascular (ICD-9 codes 390-429)

The single pollutant coefficient and standard error are calculated from an estimated percent change of 1.58 and t-statistic of 4.59 for a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 0-day lag GAM-30df stringent (10-8) model (Moolgavkar, 2003, Table 12). In a log-linear model, the percent change is equal to $(\text{RR} - 1) * 100$.

G.1.3.8 Peng et al. (2008)

Peng et al. (2008) examined the risk of hospital admissions for cardiovascular and respiratory diseases in relation to particulate matter ($\text{PM}_{10-2.5}$ and $\text{PM}_{2.5}$). To accomplish this, the authors utilized a database of 108 U.S. counties with daily emergency hospital admission rates for cardiovascular and respiratory diseases among Medicare enrollees living 9 miles from air monitors, temperature, and dew-point temperature. $\text{PM}_{10-2.5}$ and $\text{PM}_{2.5}$ concentrations were calculated by using monitoring data from January 1, 1999 through December 31, 2005. Overall, there were 3.7 million cardiovascular disease and 1.4 million respiratory disease-related hospital admissions for the time period assessed. The authors found that a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{10-2.5}$ was associated with a 0.36% increase (95% PI: 0.05-0.68%) in cardiovascular disease admissions on the same day, and a 0.25% increase (95% PI: -0.11-0.60%) after adjusting for $\text{PM}_{2.5}$. For respiratory disease admissions, a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{10-2.5}$ was found to be associated with an unadjusted 0.33% increase in respiratory disease admissions (95% PI: -0.21- 0.86%) and an adjusted 0.26% increase (95% PI: -0.32-

0.84%) in emergency admissions. Also, unadjusted associations of PM_{2.5} with cardiovascular and respiratory disease admissions were 0.71% (95% PI: 0.45-0.96%) for same-day exposure and 0.44% (95% PI: 0.06-0.82%) for exposure lagged by 2 days prior to hospital admission.

Hospital Admissions, Cardio-, Cerebro-, and Peripheral Vascular Disease (ICD-9 codes 426-427, 428, 430-438, 410-414, 429, 440-448)

In a single-pollutant model, the coefficient and standard error are estimated from the percent change in daily admission (0.44%) and 95% posterior interval (95% PI: 0.06-0.82%) for a 10 µg/m³ increase in daily 24-hour mean PM_{2.5} concentrations for the same day (Peng et al., 2008, page 2175).

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

G.1.3.9 Peng et al. (2009)

Peng et al. (2009) investigated the relationship between hospital admissions for cardiovascular and respiratory disease and the chemical components of PM_{2.5} across 119 U.S. urban communities for 12 million Medicare enrollees using log-linear Poisson regression models. This was achieved using a national database with daily data from 2000-2006 on emergency hospital admissions of cardiovascular and respiratory outcomes, ambient levels of PM_{2.5} components and weather variables. Bayesian hierarchical statistical models were used to estimate the associations. Three scenarios for PM_{2.5} exposure were assessed which were as follows: 1) for the period 2000-2006 and including only days with available measurements for all 7 PM_{2.5} components from the Speciation Trends network (STN); 2) PM_{2.5} measured by the STN for the period 2000-2006 and including only days with available measurements for all 7 PM_{2.5} components from the STN and 3) PM_{2.5} estimated as the sum of the 7 largest components of PM_{2.5} mass for the period 2000-2006. Results of percent increases in emergency admissions associated with PM_{2.5} at lag 0 under these scenarios were showed in Figure 2 and the results for the components of PM_{2.5} from both single and multi-pollutant models were showed in Figure 3. In multi-pollutant models that adjusted for the levels of other pollutants, the authors found that an interquartile range increase in elemental carbon was associated with a 0.80% increase (95% PI: 0.34-1.27%) in risk of same-day cardiovascular admissions. Similarly, an interquartile range increase in organic carbon matter was associated with a 1.01% increase (95% PI: 0.04-1.98%) risk of respiratory admissions on the same day.

Hospital Admissions, Cardio-, Cerebro-, and Peripheral Vascular Disease (ICD-9 codes 426-427, 428, 430-438, 410-414, 429, 440-448)

In a single-pollutant model, the coefficient and standard error are estimated from the percent change in daily admission (0.68%) and 95% posterior interval (95% PI: 0.26-

1.10%) for a 10 $\mu\text{g}/\text{m}^3$ increase in daily 24-hour mean $\text{PM}_{2.5}$ concentrations for the same day (Peng et al., 2009, page 960).

Note that Peng et al. (2009) considered a broader range of ICD-9 codes and estimated the risk of both cardiovascular events and cerebro- and peripheral vascular disease. For comparability to other studies, EPA decided to apply a baseline hospitalization rate for ICD-9 codes 390-409 and 411-429 when using this C-R function in quantifying impacts.

G.1.3.10 Sheppard (2003)

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

In response to concerns that the work by Sheppard et al. (1999) may be biased because of the Splus issue, Sheppard (2003) reanalyzed some of this work, in particular Sheppard reanalyzed the original study's $\text{PM}_{2.5}$ single pollutant model.

Hospital Admissions, Asthma (ICD-9 code 493)

The coefficient and standard error are based on the relative risk (1.04) and 95% confidence interval (1.01-1.06) for a 11.8 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ in the 1-day lag GAM stringent model (Sheppard, 2003, pp. 228-229).

G.1.3.11 Zanobetti et al. (2009)

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

Hospital Admissions, All Cardiovascular (ICD-9 codes 390-429)

In a single-pollutant model, the coefficient and standard error are estimated from the percent change in risk (1.89%) and 95% confidence interval (1.34%-2.45%) for a 10 $\mu\text{g}/\text{m}^3$ increase in 2-day averaged $\text{PM}_{2.5}$ (Zanobetti et al., 2009, Table 3).

Note that Zanobetti et al. (2009) report results for ICD codes 390-429. In the benefit analysis, avoided nonfatal heart attacks are estimated separately. In order to avoid double counting heart attack hospitalizations, we have excluded ICD code 410 from the baseline incidence rate used in this function.

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 $\mu\text{g}/\text{m}^3$ increase in 2-day averaged $\text{PM}_{2.5}$ (Zanobetti et al., 2009, Table 3).

G.1.4 Emergency Room Visits

Table G-8 summarizes the additional health impacts functions used to estimate the relationship between $\text{PM}_{2.5}$ 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 G-8. Additional Health Impact Functions for Particulate Matter and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Asthma	Glad et al.	2012	Pittsburgh, PA	0-99		D24HourMean	0.00392	0.00284	Logistic	All Races
Asthma	Mar et al.	2010	Greater Tacoma, WA	0-99		D24HourMean	0.0056	0.0021	Log-linear	
Emergency Room Visits, Asthma	Norris et al.	1999	Seattle, WA	0-17	NO_2 , SO_2	D24HourMean	0.016527	0.004139	Log-linear	
Asthma	Slaughter et al.	2005	Spokane, WA	0-99		D24HourMean	0.0029	0.0027	Log-linear	

G.1.4.1 Glad et al. (2012)

Glad et al. (2012) examined the relationship between air pollution and emergency department visits for asthma (ICD-9 code 493) in six Pittsburgh, PA hospitals between 2002 and 2005. The study includes a total of 6,979 individuals with a primary discharge diagnosis of asthma. Using a case-crossover methodology, particulate matter with an aerodynamic diameter $\leq 2.5\mu\text{m}$ ($\text{PM}_{2.5}$) had an effect both on the total population on day 1 after exposure (1.036, $p < 0.05$), and on African Americans for day-1 lag (OR = 1.055;

95% CI, 1.001–1.112), day-2 lag (OR=1.067; 95% CI, 1.015–1.122), day-3 lag (OR=1.053; 95% CI, 1.002–1.106), and the 6-day average (OR =1.088; 95% CI, 1.001–1.184). PM_{2.5} had no significant effect on Caucasian Americans alone. The disparity in risk estimates by race may reflect differences in residential characteristics, exposure to ambient air pollution, or a differential effect of pollution by race.

Emergency Room Visits, Asthma (ICD-9 Code 493) – by Age and Racial Group

In a single pollutant model for patients of all ages and all races, the coefficient and standard error were estimated from the odds ratio (1.04) and 95% confidence interval (0.984 – 1.10) for a 10 µg/m³ increase in 24-hour mean PM_{2.5} concentration averaged over lags 0-6 days (Glad et al., 2012, Table 3).

G.1.4.2 Mar et al. (2010)

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

Emergency Room Visits, Asthma (ICD-9 code not reported)

In a single-pollutant model, the coefficient and standard error are estimated from the relative risk (1.04) and 95% confidence interval (95% CI: 1.01-1.07) for a 7 µg/m³ increase in daily 24-hour mean PM_{2.5} at lag day 2 (Mar et al., 2010, Table 4).

G.1.4.3 Norris et al. (1999)

Norris et al. (1999) examined the relation between air pollution in Seattle and childhood (<18) hospital admissions for asthma from 1995 to 1996. The authors used air quality data for PM₁₀, light scattering (used to estimate fine PM), CO, SO₂, NO₂, and O₃ in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to PM_{2.5}), PM₁₀, and CO. No association was found between O₃, NO₂, or SO₂ and asthma ER visits, although O₃ had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM₁₀) and NO₂ and SO₂, the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits. The PM C-R functions are based on results of the single and multipollutant models reported.

Emergency Room Visits, Asthma

In a model with NO₂ and SO₂, the PM_{2.5} coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 µg/m³ increase in PM_{2.5} (Norris et al., 1999, p.491).

G.1.4.4 Slaughter et al. (2005)

Slaughter et al. (2005) examined the short-term association of particulate matter (PM₁, PM_{2.5}, PM₁₀, and PM_{10-2.5}) and carbon monoxide with hospital admissions and emergency room visits for respiratory and cardiac outcomes and mortality in Spokane, Washington, from January 1995 to June 2001 using a log-linear generalized linear model. The authors found no association between respiratory emergency room visits and any size fraction of PM, but there was a suggestive relationship between fine PM and respiratory effects when compared to coarse PM. No association between cardiac hospital admissions or mortality and any size fraction of PM or CO was observed at the 0- to 3-day lag. CO, on the other hand, was found to be associated with all respiratory emergency room visits and visits for asthma at the 3-day lag.

Emergency Room Visits, Asthma (ICD-9 code 493)

In a single-pollutant model, the coefficient and standard error are estimated from the relative risk (1.03) and 95% confidence interval (95% CI: 0.98-1.09) for a 10 µg/m³ increase in daily 24-hour mean PM_{2.5} at 1-day lag (Slaughter et al., 2005, Table 4).

G.1.5 Minor Effects

Table G-9 summarizes the additional 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 G-9. Additional Health Impact Functions for Particulate Matter and Minor Effects

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form
Acute Bronchitis	Dockery et al.	1996	24 communities	8-12		Annual	0.027212	0.017096	Logistic
Work Loss Days	Ostro	1987	Nationwide	18-64		D24HourMean	0.004600	0.000360	Log-linear
Minor Restricted Activity Days	Ostro and Rothschild	1989	Nationwide	18-64	Ozone	D24HourMean	0.007410	0.000700	Log-linear
Lower Respiratory Symptoms	Schwartz and Neas	2000	6 U.S. cities	7-14		D24HourMean	0.019012	0.006005	Logistic

G.1.5.1 Dockery et al. (1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a

study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis. The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable. They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes. Earlier work, by Dockery et al. (1989), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15} . Because it is based on a larger sample, the Dockery et al. (1996) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis.

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

Acute Bronchitis

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

Incidence Rate: annual bronchitis incidence rate per person = 0.043 (American Lung Association, 2002a, Table 11)

Population: population of ages 8-12.

G.1.5.2 Ostro (1987)

See full study description under Ostro (1987) in Appendix E, Section E.5.1.

G.1.5.3 Ostro and Rothschild (1989)

See full study description under Ostro and Rothschild (1989) in Appendix E, Section E.5.2.

G.1.5.4 Schwartz and Neas (2000)

Schwartz et al. (2000) replicated a previous analysis (Schwartz et al., 1994) linking PM levels to lower respiratory symptoms in children in six cities in the U.S. The original

study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14. The previous study focused on PM₁₀, acid aerosols, and gaseous pollutants, although single-pollutant PM_{2.5} results were reported. Schwartz et al. (2000) focused more on the associations between PM_{2.5} and PM_{10-2.5} and lower respiratory symptoms. In single and co-pollutant models, PM_{2.5} was significantly associated with lower respiratory symptoms, while PM_{10-2.5} was not. PM_{10-2.5} exhibited a stronger association with cough than did PM_{2.5}. The PM_{2.5} C-R functions for lower respiratory symptoms are based on the results of the reported single pollutant and co-pollutant model (PM_{2.5} and PM_{10-2.5}).

Lower Respiratory Symptoms

The coefficient and standard error are calculated from the reported odds ratio (1.33) and 95% confidence interval (1.11-1.58) associated with a 15 µg/m³ change in PM_{2.5} (Schwartz and Neas, 2000, Table 2).

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

Population: population of ages 7 to 14

G.1.6 Asthma-Related Effects

Table G-10 summarizes the additional 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 G-10. Additional Health Impact Functions for Particulate Matter and Asthma-Related Effects

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Cough	Mar et al.	2004	Spokane, WA	6-18		D24HourMean	0.0191	0.0098	Logistic	Uses incidence rate from Ostro et al. (2001). Age range adjusted.
Shortness of Breath	Mar et al.	2004	Spokane, WA	6-18		D24HourMean	0.0122	0.0138	Logistic	Uses incidence rate from Ostro et al. (2001). Age range adjusted.
Cough	Ostro et al.	2001	Los Angeles, CA	6-18		D24HourMean	0.000985	0.000747	Logistic	
Shortness of Breath	Ostro et al.	2001	Los Angeles, CA	6-18		D24HourMean	0.002565	0.001335	Logistic	
Wheeze	Ostro et al.	2001	Los Angeles, CA	6-18		D24HourMean	0.001942	0.000803	Logistic	
Upper Respiratory Symptoms	Pope et al.	1991	Utah Valley	9-11		D24HourMean	0.003600	0.001500	Logistic	

G.1.6.1 Mar et al. (2004)

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

Asthma Exacerbation, Cough

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.21) and 95% confidence interval (1.00-1.47) for a 10.0 µg/m³ increase in 1-day lagged concentration of PM_{2.5} (Mar et al., 2004, Table 7).

Incidence Rate: Daily cough rate per person = 14.5%. Mar et al. (2004) did not report the incidence rate for each type of asthma exacerbation. The daily cough rate from Ostro et al. (2001, p.202) is applied here.

Population: The study reported results for population ages 7-12. For comparability to other studies, we apply the results to the population of ages 6 to 18. We treat this as two groups based on the available information from American Lung Association (2010b, Table 7). Asthmatic population ages 6 to 17 = 10.70% of population ages 6 to 17 and asthmatic population age 18 = 7.19% of population age 18. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 and adults 18-44 at 10.70% and 7.19% respectively (based on data from the 2008 National Health Interview Survey).

Asthma Exacerbation, Shortness of Breath

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.13) and 95% confidence interval (0.86-1.48) for a 10.0 µg/m³ increase in current-day concentration of PM_{2.5} (Mar et al., 2004, Table 7).

Incidence Rate: Daily shortness of breath rate per person = 7.4%. Mar et al. (2004) did not report the incidence rate for each type of asthma exacerbation. The daily rate of shortness of breath from Ostro et al. (2001, p.202) is applied here.

Population: See the population description for “Asthma Exacerbation, Cough” from Mar et al. (2004).

G.1.6.2 Ostro et al. (2001)

Ostro et al. (2001) studied the relation between air pollution in Los Angeles and asthma exacerbation in African-American children (8 to 13 years old) from August to November 1993. They used air quality data for PM₁₀, PM_{2.5}, NO₂, and O₃ in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. The authors note that there were 26 days in which PM_{2.5} concentrations were reported higher than PM₁₀ concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions. Asthma symptom endpoints were defined in two ways: “probability of a day with symptoms” and “onset of symptom episodes”. New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day.

The authors found cough prevalence associated with PM₁₀ and PM_{2.5} and cough incidence associated with PM_{2.5}, PM₁₀, and NO₂. Ozone was not significantly associated with cough among asthmatics. The authors found that both the prevalent and incident episodes of shortness of breath were associated with PM_{2.5} and PM₁₀. Neither ozone nor NO₂ were significantly associated with shortness of breath among asthmatics. The authors found both the prevalence and incidence of wheeze associated with PM_{2.5}, PM₁₀, and NO₂. Ozone was not significantly associated with wheeze among asthmatics.

The derived health impact functions are based on the results of single pollutant models looking at the probability of symptoms.

Asthma Exacerbation, Cough

The coefficient and standard error are based on an odds ratio of 1.03 (95% CI 0.98-1.07) for a 30 µg/m³ increase in 12-hour average PM_{2.5} concentration (Ostro et al., 2001, Table 4, p.204).

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

Population: The study reported results for African-American population ages 8-13. For comparability to other studies, we apply the results to the African-American population of ages 6 to 18. We treat this as two groups based on the available information from American Lung Association (2010b, Table 9). Asthmatic African-American population ages 6 to 17 = 17.76% of African-American population ages 6 to 17 and asthmatic African-American population age 18 = 7.52% of African-American population age 18. The American Lung Association (2010b, Table 9) estimates asthma prevalence for children 5- 17 and adults 18-44 at 17.76% and 7.52% respectively (based on data from the 2008 National Health Interview Survey).

Asthma Exacerbation, Shortness of Breath

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

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

Population: The study reported results for African-American population ages 8-13. For comparability to other studies, we apply the results to the African-American population of ages 6 to 18. We treat this as two groups based on the available information from American Lung Association (2010b, Table 9). Asthmatic African-American population ages 6 to 17 = 17.76% of African-American population ages 6 to 17 and asthmatic African-American population age 18 = 7.52% of African-American population age 18. The American Lung Association (2010b, Table 9) estimates asthma prevalence for children 5- 17 and adults 18-44 at 17.76% and 7.52% respectively (based on data from the 2008 National Health Interview Survey).

Asthma Exacerbation, Wheeze

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

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

Population: asthmatic African-American population ages 8 to 13 = 17.76% of African-American population ages 8 to 13. (Described above.)

G.1.6.3 Pope et al. (1991)

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

treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM₁₀ significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM₁₀ effect. The results from the school-based sample are used here.

Upper Respiratory Symptoms

The coefficient and standard error for a one µg/m³ change in PM₁₀ is reported in Table 5.

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

Population: asthmatic population ages 9 to 11 = 10.70% of population ages 9 to 11. (The American Lung Association (2010b, Table 7) estimates asthma prevalence for children ages 5 to 17 at 10.70%, based on data from the 2008 National Health Interview Survey.)

G.2 Additional Ozone Health Impact Functions

Section G.2 summarizes the additional health impact functions for Ozone included in BenMAP-CE.

G.2.1 Short-term Mortality

Table G-11 summarizes the additional health impacts functions used to estimate the relationship between ozone and short-term mortality. Below, we present a brief summary of each of the studies and any items that are unique to the study.

Table G-11. Additional Health Impact Functions for Ozone and Mortality*

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Non-Accidental	Bell et al.	2004	95 US cities	0-99		D8HourMax	0.000261	0.000089	Log-linear	Warm season. 8-hour max from 24-hour mean
Non-Accidental	Bell et al.	2004	95 US cities	0-99		D24HourMean	0.000520	0.000128	Log-linear	All year
All Cause	Bell et al.	2005	US & non-US	0-99		D8HourMax	0.000795	0.000212	Log-linear	Warm season. 8-hour max from 24-hour mean
All Cause	Bell et al.	2005	US & non-US	0-99		D24HourMean	0.001500	0.000401	Log-linear	Warm season

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Cardio-pulmonary	Huang et al.	2005	19 US cities	0-99		D8HourMax	0.000813	0.000259	Log-linear	Warm season. 8-hour max from 24-hour mean.
Cardio-pulmonary	Huang et al.	2005	19 US cities	0-99		D24HourMean	0.001250	0.000398	Log-linear	Warm season
Non-Accidental	Ito and Thurston	1996	Chicago, IL	18-99	PM ₁₀	D1HourMax	0.000634	0.000251	Log-linear	
Non-Accidental	Ito et al.	2005		0-99		D8HourMax	0.001173	0.000239	Log-linear	Warm season. 8-hour max from 24-hour mean.
Non-Accidental	Ito et al.	2005		0-99		D1HourMax	0.000400	0.000066	Log-linear	1-hour max
Non-Accidental	Ito et al.	2005		0-99		D24HourMean	0.001750	0.000357	Log-linear	Warm season. 24-hour mean
Non-Accidental	Ito et al.	2005		0-99		D8HourMax	0.000532	0.000088	Log-linear	8-hour max from 1-hour max
Long Term Mortality, Respiratory	Jerrett et al.	2009	86 urban areas	30-99	PM _{2.5}	Annual	0.003922	0.001325	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Northeast	30-99		Annual	-0.001005	0.003853	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Industrial Midwest	30-99		Annual	0.000000	0.004604	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Southeast	30-99		Annual	0.011333	0.003193	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Upper Midwest	30-99		Annual	0.013103	0.026212	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Northwest	30-99		Annual	0.005827	0.003118	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Southwest	30-99		Annual	0.019062	0.007583	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	Southern California	30-99		Annual	0.000995	0.002767	Log-linear	
Long Term Mortality, Respiratory	Jerrett et al.	2009	86 urban areas	30-99	PM _{2.5}	Annual	0.004471	0.001510	Log-linear	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
All Cause	Levy et al.	2005	US and non-US	0-99		D8HourMax	0.001119	0.000179	Log-linear	Warm season. 8-hour max from 1-hour max.
All Cause	Levy et al.	2005	US and non-US	0-99		D1HourMax	0.000841	0.000134	Log-linear	Warm season.
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	0-99		D24HourMean	0.001398	0.000266	Log-linear	Warm season.
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	0-99	TSP, SO ₂	D24HourMean	0.001389	0.000373	Log-linear	Warm season.
Non-Accidental	Moolgavkar et al.	1995	Philadelphia, PA	18-99	TSP, SO ₂	D24HourMean	0.000611	0.000216	Log-linear	
Non-Accidental	Samet et al.	1997	Philadelphia, PA	18-99	CO, NO ₂ , SO ₂ , TSP	D24HourMean	0.000936	0.000312	Log-linear	
Non-Accidental	Schwartz	2005	14 U.S. cities	0-99		D8HourMax	0.000426	0.000150	Logistic	Warm season. 8-hour max from 1-hour max.
Non-Accidental	Schwartz	2005	14 U.S. cities	0-99		D1HourMax	0.000370	0.000130	Logistic	Warm season
Non-Accidental	Smith et al.	2009	98 U.S. cities	0-99		D8HourMax	0.000322	0.000084	Log-linear	Ozone season
Non-Accidental	Smith et al.	2009	98 U.S. cities	0-99	PM ₁₀	D8HourMax	0.000258	0.000167	Log-linear	Ozone season
All-Cause	Zanobetti and Schwartz	2008	48 Cities	0-99		D8HourMax	0.00050	0.00012	Log-linear	0-3 day lag, June-August, 1989-2000

*Unless otherwise stated, mortality is short-term.

G.2.1.1 Bell et al. (2004)

Ozone has been associated with various adverse health effects, including increased rates of hospital admissions and exacerbation of respiratory illnesses. Although numerous time-series studies have estimated associations between day-to-day

variation in ozone levels and mortality counts, results have been inconclusive. The authors investigated whether short-term (daily and weekly) exposure to ambient ozone is associated with mortality in the United States. Using analytical methods and databases developed for the National Morbidity, Mortality, and Air Pollution Study, they estimated a national average relative rate of mortality associated with short-term exposure to ambient ozone for 95 large US urban communities from 1987-2000. The authors used distributed-lag models for estimating community-specific relative rates of mortality adjusted for time-varying confounders (particulate matter, weather, seasonality, and long-term trends) and hierarchical models for combining relative rates across communities to estimate a national average relative rate, taking into account spatial heterogeneity. A 10-ppb increase in the previous week's ozone was associated with a 0.52% increase in daily mortality (95% posterior interval [PI], 0.27%-0.77%) and a 0.64% increase in cardiovascular and respiratory mortality (95% PI, 0.31%-0.98%). Effect estimates for aggregate ozone during the previous week were larger than for models considering only a single day's exposure. Results were robust to adjustment for particulate matter, weather, seasonality, and long-term trends. These results indicate a statistically significant association between short-term changes in ozone and mortality on average for 95 large US urban communities, which include about 40% of the total US population.

Non-Accidental Mortality

The coefficient and standard error are based on the relative risk (1.003908) and 95% confidence interval (1.0013-1.0065) associated with a 10 ppb increase in daily average ozone (Bell et al., 2004, p. 2376).

G.2.1.2 Bell et al. (2005)

Although many time-series studies of ozone and mortality have identified positive associations, others have yielded null or inconclusive results, making the results of these studies difficult to interpret. The authors performed a meta-analysis of 144 effect estimates from 39 time-series studies, and estimated pooled effects by lags, age groups, cause-specific mortality, and concentration metrics. They compared results with pooled estimates from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), a time-series study of 95 large U.S. urban centers from 1987 to 2000. Both meta-analysis and NMMAPS results provided strong evidence of a short-term association between ozone and mortality, with larger effects for cardiovascular and respiratory mortality, the elderly, and current-day ozone exposure. In both analyses, results were insensitive to adjustment for particulate matter and model specifications. In the meta-analysis, a 10-ppb increase in daily ozone at single-day or 2-day average of lags 0, 1, or 2 days was associated with an 0.87% increase in total mortality (95% posterior interval = 0.55% to 1.18%), whereas the lag 0 NMMAPS estimate is 0.25% (0.12% to 0.39%). Several findings indicate possible publication bias: meta-analysis results were consistently larger than those from NMMAPS; meta-analysis pooled estimates at lags 0 or 1 were larger when only a single lag was reported than when estimates for multiple lags were reported; and heterogeneity of city-specific estimates in the meta-analysis were larger than with NMMAPS.

All-Cause Mortality

The coefficient and standard error are based on the relative risk (1.008738) and 95% confidence interval (1.0055-1.0119) associated with a 10 ppb increase in daily average ozone (Bell et al., 2005, Table 6).

G.2.1.3 Huang et al. (2005)

The authors developed Bayesian hierarchical distributed lag models for estimating associations between daily variations in summer ozone levels and daily variations in cardiovascular and respiratory (CVDRESP) mortality counts for 19 large U.S. cities included in the National Morbidity, Mortality and Air Pollution Study (NMMAPS) for the summers of 1987-1994. In the first stage, they defined a semi-parametric distributed lag Poisson regression model to estimate city-specific relative rates of CVDRESP mortality associated with short-term exposure to summer ozone. In the second stage, they specified a class of distributions for the true city-specific relative rates to estimate an overall effect by taking into account the variability within and across cities. They performed the calculations with respect to several random effects distributions (normal, t-student, and mixture of normal), thus relaxing the common assumption of a two-stage normal-normal hierarchical model. They assessed the sensitivity of the results to: (i) lag structure for ozone exposure; (ii) degree of adjustment for long-term trends; (iii) inclusion of other pollutants in the model; (iv) heat waves; (v) random effects distributions; and (vi) prior hyperparameters. On average across cities, the authors found that a 10 ppb increase in summer ozone level over the previous week is associated with a 1.25 per cent increase in CVDRESP mortality (95 per cent posterior regions: 0.47, 2.03). The relative rate estimates are also positive and statistically significant at lags 0, 1 and 2. They found that associations between summer ozone and CVDRESP mortality are sensitive to the confounding adjustment for PM₁₀, but are robust to: (i) the adjustment for long-term trends, other gaseous pollutants (NO₂, SO₂ and CO); (ii) the distributional assumptions at the second stage of the hierarchical model; and (iii) the prior distributions on all unknown parameters.

Cardiopulmonary Mortality

Assuming a 10 ppb change in ozone, Huang et al. (2005, Table 1) reported a 1.25% change in CVDRESP mortality with a 95% confidence interval of 0.47% to 2.03%.

Note that Huang et al. (2005, p. 549) define CVDRESP as including ICD-9 codes: 390-448, 480-487, 490-496, and 507. This differs somewhat from the the definition of “cardiopulmonary” mortality in BenMAP -- defined as ICD-9 codes 401-440 and 460-519.

G.2.1.4 Ito and Thurston (1996)

In this study, race, gender, and cause-specific counts of daily mortality in Cook County, Illinois (which encompasses the city of Chicago) during 1985-1990 were analyzed to determine if there was any heterogeneity in air pollution/weather/mortality associations across these various population subcategories. Seasonal cross-correlations

between mortality and environmental variables first were examined to identify appropriate lag structures. Of the pollution variables considered -- PM₁₀, ozone, CO, SO₂, and visual range-derived extinction coefficient -- both PM₁₀ and ozone showed significant associations with same-day and next-day mortality. The Poisson regression models employed included seasonal cycles (sine/cosine series), square and linear terms of lagged temperature, trend line, day-of-week dummy variables, and the average of the same day's and previous day's PM₁₀ or ozone.

The authors reported a significant relationship for ozone and PM₁₀ with both pollutants in the model; no significant effects were found for SO₂ and CO. In single pollutant models the effects were slightly larger. The health impact function for ozone is based on results from the co-pollutant models.

Non-Accidental Mortality

For a co-pollutant model with PM₁₀, the ozone coefficient (0.000634) and standard error (0.000251) were obtained directly from the author because the published paper reported incorrect information.

G.2.1.5 Ito et al. (2005)

The authors conducted a review and meta-analysis of short-term ozone mortality studies, identified unresolved issues, and conducted an additional time-series analysis for 7 U.S. cities (Chicago, Detroit, Houston, Minneapolis-St. Paul, New York City, Philadelphia, and St. Louis). They found a combined estimate of 0.39% (95% confidence interval = 0.26-0.51%) per 10-ppb increase in 1-hour daily maximum ozone for the all-age nonaccidental cause/single pollutant model (43 studies). Adjusting for the funnel plot asymmetry resulted in a slightly reduced estimate (0.35%; 0.23-0.47%). In a subset for which particulate matter (PM) data were available (15 studies), the corresponding estimates were 0.40% (0.27-0.53%) for ozone alone and 0.37% (0.20-0.54%) with PM in model. The estimates for warm seasons were generally larger than those for cold seasons. The additional time-series analysis found that including PM in the model did not substantially reduce the ozone risk estimates. However, the difference in the weather adjustment model could result in a 2-fold difference in risk estimates (eg, 0.24% to 0.49% in multicity combined estimates across alternative weather models for the ozone-only all-year case). The authors concluded that the results suggest short-term associations between ozone and daily mortality in the majority of the cities, although the estimates appear to be heterogeneous across cities.

Non-Accidental Mortality

Ito et al. (2005) reported results for functions with 1-hour daily maximum, 24-hour daily average metrics, and 8-hour maximum from the 24-hour mean. We include the warm season 8-hour maximum from the 24-hour mean. Assuming a 20 ppb change in the daily 24-hour average, Ito et al. (2005, p. 448) reported a 3.5% change in non-accidental mortality with a 95% confidence interval of 2.1% to 4.9%.

One-hour Max Function

Assuming a 10 ppb change in the daily 1-hour maximum, Ito et al. (2005, p. 446) reported a 0.40% change in non-accidental mortality with a 95% confidence interval of 0.27% to 0.53%.

Daily Average Function

Assuming a 20 ppb change in the daily 24-hour average, Ito et al. (2005, p. 448) reported a 3.5% change in non-accidental mortality with a 95% confidence interval of 2.1% to 4.9%.

G.2.1.6 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 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) - 86 U.S. urban areas

In a two-pollutant model the coefficient and standard error are estimated from the relative risk (1.040) and 95% confidence interval (95% CI: 1.013-1.067) for a 10 ppb increase in ambient ozone concentration measured from April to September during the years from 1977 to 2000 in 86 MSAs (Jerrett, et al., 2009, Table 3).

Mortality, Respiratory (ICD-9 code 460-519) - by region

In single-pollutant models the coefficient and standard error for different regions are estimated from the relative risks and 95% confidence intervals for a 10 ppb increase in ambient ozone concentration measured from April to September during the years from 1977 to 2000 (Jerrett, et al., 2009, Table 4).

Mortality, Respiratory (ICD-9 code 460-519) - adjusted daily metric

Based on the coefficients estimated from the two-pollutant model in the 86 urban areas using daily 1-hour max metric, the coefficients were adjusted for daily 8-hour max metric using a ratio of 1.14 (Anderson and Bell 2010, Table 2).

G.2.1.7 Levy et al. (2005)

The authors conducted an empiric Bayes metaregression to estimate the ozone effect on mortality, and to assess whether this effect varies as a function of hypothesized confounders or effect modifiers. They gathered 71 time-series studies relating ozone to all-cause mortality, and they selected 48 estimates from 28 studies for the metaregression. Metaregression covariates included the relationship between ozone concentrations and concentrations of other air pollutants, proxies for personal exposure-ambient concentration relationships, and the statistical methods used in the studies. For the metaregression, they applied a hierarchical linear model with known level-1 variances. The authors estimated a grand mean of a 0.21% increase (95% confidence interval = 0.16-0.26%) in mortality per 10-microg/m increase of 1-hour maximum ozone (0.41% increase per 10 ppb) without controlling for other air pollutants. In the metaregression, air-conditioning prevalence and lag time were the strongest predictors of between-study variability. Air pollution covariates yielded inconsistent findings in regression models, although correlation analyses indicated a potential influence of summertime PM_{2.5}.

All-Cause Mortality

Levy et al. (2005, Table 1) reported a 0.43% change in all-cause mortality with a 95% confidence interval of 0.29% to 0.56% associated with a 10 µg/m³ change in ozone. We converted µg/m³ to ppb with an assumed relationship of 1.96 µg/m³ per 1.0 ppb.

G.2.1.8 Moolgavkar et al. (1995)

Moolgavkar et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO₂ in a three-pollutant model, and found a significant relationship for ozone and SO₂; TSP was not significant. In season-specific models, ozone was significantly associated with mortality only in the summer months.

Non-Accidental Mortality

The health impact function for ozone is based on the full-year three-pollutant model reported in Table 5 (Moolgavkar et al., 1995, p. 482). The coefficient and standard error are based on the relative risk (1.063) and 95% confidence interval (1.018-1.108) associated with a 100 ppb increase in daily average ozone.

G.2.1.9 Samet et al. (1997)

Samet et al. (1997) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They

examined ozone, TSP, SO₂, NO₂, and CO in a Poisson regression model. In single pollutant models, ozone, SO₂, TSP, and CO were significantly associated with mortality. In a five-pollutant model, they found a positive statistically significant relationship for each pollutant except NO₂.

Non-Accidental Mortality

The health impact function for ozone is based on the five-pollutant model (ozone, CO, NO₂, SO₂ and TSP) reported in Table 9 (Samet et al., 1997, p. 20). The ozone coefficient and standard error are based on the percent increase (1.91) and t-statistic (3) associated with a 20.219 ppb increase in two-day average ozone.

G.2.1.10 Schwartz (2005)

The author used the case-crossover approach, where the control for each person is the same person on a day near in time, when he or she did not die. This method controls for season and individual risk factors by matching. One can also choose the control day to have the same temperature as the event day. The author applied this approach to a study of more than 1 million deaths in 14 U.S. cities. He found that, with matching on temperature, a 10-ppb increase in maximum hourly ozone concentrations was associated with a 0.23% (95% confidence interval [CI] 0.01%, 0.44%) increase in the risk of dying. This finding was indistinguishable from the risk when only matching on season and controlling for temperature with regression splines (0.19%; 95% CI 0.03%, 0.35%). Control for suspended particulate matter with an aerodynamic diameter of 10 µm or less (PM₁₀) did not change this risk. However, the association was restricted to the warm months (0.37% increase; 95% CI 0.11%, 0.62%), with no effect in the cold months. The author concluded that the association between ozone and mortality risk is unlikely to be caused by confounding by temperature.

Non-Accidental Mortality

Assuming a 10 ppb change in the daily 1-hour maximum, Schwartz (2005, Table 2) reported a 0.37% change in non-accidental mortality with a 95% confidence interval of 0.11% to 0.62%.

G.2.1.11 Smith et al. (2009)

Smith et al. (2009) analyzed the relationship between daily mortality and ambient ozone concentrations through re-examination of evidence using the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), which collected daily data on mortality, meteorology, and air pollutant concentrations for 100 U.S. cities from 1987-2000. The authors examined the sensitivity of city-specific ozone-mortality estimates to treatment of meteorology and co-pollutants, dependence on different ozone metrics, use of air conditioning, regional and spatial variability, and non-linear exposure-response relationships.

Non-Accidental Mortality

Assuming a 10 ppb change in daily 8-hour maximum ozone concentration, Smith et al. (2009) reported a 0.40 (0.22)% population-weighted change in non-accidental mortality in a model without PM₁₀, and 0.27 (0.22)% population-weighted change in non-accidental mortality in a model with PM₁₀.

G.2.1.12 Zanobetti and Schwartz (2008)

See full study description under Zanobetti and Schwartz (2008) in Appendix F, Section F.1.3.

Zanobetti and Schwartz (2008) examined the issue of “mortality displacement” (i.e., deaths are occurring in frail individuals and exposure is only moving the day of death to a day slightly earlier) in 48 U.S. cities during the warm season (i.e., June-August) for the years 1989-2000. The authors estimated a series of lagged specifications and found an effect size of 0.53% (95%C.I.: 0.28-0.77) for lag 0-3 days for all-cause mortality.

G.2.2 Hospital Admissions

Table G-12 summarizes the additional 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 G-12. Additional Health Impact Functions for Ozone and Hospital Admissions

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
All Respiratory	Burnett et al.	2001	Toronto, CAN	0-1	PM _{2.5}	D8HourMax	0.008177	0.002377	Log-linear	Warm season. 8-hour max from 1-hour max.
All Respiratory	Katsouyanni et al.	2009	14 U.S. cities	65-99		D8HourMax	0.000637	0.000400	Log-linear	Summer, penalized splines, 8df
All Respiratory	Katsouyanni et al.	2009	14 U.S. cities	65-99		D8HourMax	0.000614	0.000406	Log-linear	Summer, natural splines, 8df
Chronic Lung	Moolgavkar et al.	1997	Minneapolis, MN	65-99	PM ₁₀ , CO	D24HourMean	0.002800	0.001769	Log-linear	
Chronic Lung	Moolgavkar et al.	1997	Minneapolis, MN	65-99	PM ₁₀ , CO	D8HourMax	0.001960	0.001238	Log-linear	All year. 8-hour max from 24-hour mean
Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	65-99	PM ₁₀ , SO ₂ , NO ₂	D24HourMean	0.003800	0.001088	Log-linear	
Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	65-99	PM ₁₀ , SO ₂ , NO ₂	D8HourMax	0.002660	0.000762	Log-linear	All year. 8-hour max from 24-hour mean

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Chronic Lung (less Asthma)	Schwartz	1994	Detroit, MI	65-99	PM ₁₀	D24HourMean	0.005523	0.002085	Log-linear	All year
Chronic Lung (less Asthma)	Schwartz	1994	Detroit, MI	65-99	PM ₁₀	D8HourMax	0.003424	0.001293	Log-linear	All year. 8-hour max from 24-hour mean.
Pneumonia	Schwartz	1994	Detroit, MI	65-99	PM ₁₀	D24HourMean	0.005210	0.001300	Log-linear	All year.
Pneumonia	Schwartz	1994	Minneapolis, MN	65-99	PM ₁₀	D24HourMean	0.003977	0.001865	Log-linear	All year.
Pneumonia	Schwartz	1994	Detroit, MI	65-99	PM ₁₀	D8HourMax	0.003230	0.000806	Log-linear	All year. 8-hour max from 24-hour mean
Pneumonia	Schwartz	1994	Minneapolis, MN	65-99	PM ₁₀	D8HourMax	0.002784	0.001305	Log-linear	All year. 8-hour max from 24-hour mean.
All Respiratory	Schwartz	1995	New Haven, CT	65-99	PM ₁₀	D24HourMean	0.002652	0.001398	Log-linear	Warm season
All Respiratory	Schwartz	1995	Tacoma, WA	65-99	PM ₁₀	D24HourMean	0.007147	0.002565	Log-linear	Warm season
All Respiratory	Schwartz	1995	New Haven, CT	65-99	PM ₁₀	D8HourMax	0.001777	0.000936	Log-linear	Warm season. 8-hour max from 24-hour mean
All Respiratory	Schwartz	1995	Tacoma, WA	65-99	PM ₁₀	D8HourMax	0.004931	0.001770	Log-linear	Warm season. 8-hour max from 24-hour mean

G.2.2.1 Burnett et al. (2001)

Burnett et al. (2001) studied the association between air pollution and acute respiratory hospital admissions (ICD codes 493, 466, 464.4, 480-486) in Toronto from 1980-1994, among children less than 2 years of age. They collected hourly concentrations of the gaseous pollutants, CO, NO₂, SO₂, and ozone. Daily measures of particulate matter were estimated for the May to August period of 1992-1994 using TSP, sulfates, and coefficient of haze data. The authors report a positive association between ozone in the May through August months and respiratory hospital admissions, for several single days after elevated ozone levels.

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

Hospital Admissions, All Respiratory (ICD-9 codes 464, 466, 480-487, 493)

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

G.2.2.2 Katsouyanni et al. (2009)

Katsouyanni et al. (2009) assessed the relationship between air pollution and hospital admissions from 1985 to 1994. Specifically, the authors examined hospitalizations due to respiratory diseases, conditions, or infections (ICD-9 codes 460-519) among U.S. citizens age 65 or older. The U.S. dataset included 14 cities with each city having data for 4 to 10 years from 1985-1994 and 7 cities having only summer ozone data. The investigators used a three-stage hierarchical model to account for within-city, within region, and between region variability. In the U.S. in adults 65 and older, authors found a .26% (.01, .51) increase in respiratory hospital admissions with 10 µg/m³ increase in ozone concentration using penalized splines with 8 df/year using lag 1. Authors found a .33% (.08, .58) increase in respiratory hospital admissions with 10 µg/m³ increase in ozone concentration using natural splines with 8 df/year using distributed lags.

G.2.2.3 Moolgavkar et al. (1997)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions (ICD-9 codes 490-496) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a Poisson regression, they found no significant effect for any of the pollutants (PM₁₀, ozone, or CO). The effect for ozone was marginally significant. The model with a 100 df smoother was reported to be optimal (p. 368). The health impact function for chronic lung disease is based on the results from a three-pollutant model (ozone, CO, PM₁₀) using the 100 df smoother; the function for Pneumonia uses the 130 df smoother.

Hospital Admissions, Chronic Lung Disease (ICD-9 codes 490-496)

In a model with CO and PM₁₀, the estimated coefficient and standard error are based on the percent increase (4.2) and 95% confidence interval of the percent increase (-1.0-9.4) associated with a change in daily average ozone levels of 15 ppb (Moolgavkar et al., 1997, Table 4).

Hospital Admissions, Pneumonia (ICD-9 codes 480-487)

In a model with NO₂, PM₁₀, and SO₂, the estimated coefficient and standard error are based on the percent increase (5.7) and 95% confidence interval of the percent increase (2.5-8.9) associated with an increase in daily average ozone levels of 15 ppb (Moolgavkar et al., 1997, Table 4).

G.2.2.4 Schwartz (1994a)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In single-pollutant Poisson regression models, both ozone and PM₁₀ were significantly associated with pneumonia admissions. In a two-pollutant model, Schwartz found PM₁₀ significantly related to pneumonia; ozone was weakly linked to pneumonia. The results were not sensitive to the methods used to control for seasonal patterns and weather. The ozone C-R functions are based on the results of the single pollutant model and the two-pollutant model (PM₁₀ and ozone) with spline smoothing for temporal patterns and weather.

Hospital Admissions, Pneumonia (ICD-9 codes 480-487)

In a model with PM₁₀ and spline functions to adjust for time and weather, the coefficient and standard error are based on the relative risk (1.22) and 95% confidence interval (1.02, 1.47) for a 50 ppb increase in daily average ozone levels (Schwartz, 1994a, Table 4).

G.2.2.5 Schwartz (1994b)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions (ICD codes 491-492, 494-496) for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant Poisson regression model, Schwartz found both PM₁₀ and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The C-R function for chronic lung disease incidence is based on the results of the “basic” co-pollutant model (ozone and PM₁₀) presented in Table 4 (p. 651). The study also reports results using generalized additive models to fit time and temperature variables, however no standard error or confidence intervals were reported.

Hospital Admissions, Chronic Lung Disease less Asthma (ICD-9 codes 490-492, 494-496)

The coefficient and standard error for the “basic” model are reported in Table 4 (Schwartz, 1994b, p.651) for a one ppb change in daily average ozone.

Hospital Admissions, Pneumonia (ICD-9 codes 480-487)

The ozone C-R function for pneumonia incidence is based on the coefficient and standard error for the “basic” co-pollutant model presented in Table 4 (Schwartz, 1994b, p. 651).

G.2.2.6 Schwartz (1995)

Studies have reported associations between short term changes in air pollution and respiratory hospital admissions. This relationship was examined in two cities with substantially different levels of sulphur dioxide (SO₂) but similar levels of airborne particles in an attempt to separate the effects of the two pollutants. Significant differences in weather between the two cities allowed the evaluation of that potential confounder also. Daily counts of admissions to all hospitals for respiratory disease (ICD 9 460-519) were constructed for persons aged 65 years and older in two cities - New Haven, Connecticut and Tacoma, Washington.

Each city was analysed separately. Average daily concentrations of SO₂, inhalable particles (PM₁₀), and ozone were computed from all monitors in each city, and daily average temperature and humidity were obtained from the US weather service. Daily respiratory admission counts were regressed on temperature, humidity, day of the week indicators, and air pollution. A 19-day weighted moving regression filter was used to remove all seasonal and subseasonal patterns from the data. Possible U- shaped dependence of admissions on temperature was dealt with using indicator variables for eight categories each of temperature and humidity. Each pollutant was first examined individually and then multiple pollutant models were fitted. All three pollutants were associated with respiratory hospital admissions of the elderly. The PM₁₀ associations were little changed by control for either ozone or SO₂. The ozone association was likewise independent of the other pollutants. The SO₂ association was substantially attenuated by control for ozone in both cities, and by control for PM₁₀ in Tacoma. The magnitude of the effect was small (relative risk 1.06 in New Haven and 1.10 in Tacoma for a 50 micrograms/m³ increase in PM₁₀, for example) but, given the ubiquitous exposure, this has some public health significance. The authors concluded that air pollution concentrations within current guidelines were associated with increased respiratory hospital admissions of the elderly. The strongest evidence for an independent association was for PM₁₀, followed by ozone.

Hospital Admissions, All Respiratory (ICD-9 codes 460-519) -- Tacoma

In a model with PM₁₀, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.06-1.37) for a 50 µg/m³ increase in average daily ozone levels (Schwartz, 1995, Table 6, p. 535). To calculate the coefficient, a conversion of 1.96 µg/m³ per ppb was used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius).

Hospital Admissions, All Respiratory (ICD-9 codes 460-519) -- New Haven

In a model with PM₁₀, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.15) for a 50 µg/m³ increase in average daily ozone levels (Schwartz, 1995, Table 3, p. 534). To calculate the coefficient, a conversion of 1.96 µg/m³ per ppb was used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius).

G.2.3 Emergency Room Visits

Table G-13 summarizes the additional 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 G-13. Additional Health Impact Functions for Ozone and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form
Asthma	Glad et al.	2012	Pittsburgh, PA	0-99		D8HourMax	0.003057	0.0011709	Logistic
Asthma	Ito et al.	2007	New York, NY	0-99		D8HourMax	0.005213	0.000909	Log-linear
Asthma	Ito et al.	2007	New York, NY	0-99	PM _{2.5}	D8HourMax	0.003976	0.000979	Log-linear
Asthma	Mar and Koenig	2009	Seattle, WA	18-99		D8HourMax	0.007696	0.0028374	Log-linear
Asthma	Mar and Koenig	2009	Seattle, WA	0-17		D8HourMax	0.010436	0.0043576	Log-linear
Asthma	Peel et al.	2005	Atlanta, GA	0-99		D8HourMax	0.000870	0.000529	Log-linear
Asthma	Sarnat et al.	2013	Atlanta, GA	0-99		D8HourMax	0.001113	0.0002828	Log-linear
Asthma	Wilson et al.	2005	Portland, ME	0-99		D8HourMax	0.003000	0.001000	Log-linear
Asthma	Wilson et al.	2005	Manchester, NH	0-99		D8HourMax	-0.001000	0.002000	Log-linear

G.2.3.1 Glad et al. (2012)

Glad et al. (2012) is summarized in section G.1.4.1.

Emergency Room Visits, Asthma

Using a case-crossover methodology, which controls for the effects of subject-specific covariates such as gender and race, a 2.5% increase was observed in asthma ED visits for each 10 ppb increase in the 1-hour maximum ozone level on day 2 (odds ratio [OR] = 1.025, p < 0.05).

G.2.3.2 Ito et al. (2007)

The authors assessed the effect of multi-collinearity among gaseous co-pollutants of PM_{2.5} and weather variables. The authors compiled daily meteorological and pollutant data in New York City for the years 1999 to 2002 and analyzed the effect of ozone on

asthma emergency department visits for all year, warm months and cold months (all excluding September and October due to fall peaks in asthma emergency department visits). Data on ED visits were obtained from the 11 New York City Health and Hospitals Corporation medical centers with emergency receiving facilities.

G.2.3.3 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 2 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.

G.2.3.4 Peel et al. (2005)

A number of emergency department studies have corroborated findings from mortality and hospital admission studies regarding an association of ambient air pollution and respiratory outcomes. More refined assessment has been limited by study size and available air quality data. Measurements of 5 pollutants: PM₁₀, ozone, NO₂, CO, and SO₂ were available for the entire study period (1 January 1993 to 31 August 2000); detailed measurements of particulate matter were available for 25 months. The authors obtained data on 4 million emergency department visits from 31 hospitals in Atlanta. Visits for asthma, chronic obstructive pulmonary disease, upper respiratory infection, and pneumonia were assessed in relation to air pollutants using Poisson generalized estimating equations. In single-pollutant models examining 3-day moving averages of pollutants (lags 0, 1, and 2): standard deviation increases of ozone, NO₂, CO, and PM₁₀ were associated with 1-3% increases in URI visits; a 2 microg/m increase of PM_{2.5} organic carbon was associated with a 3% increase in pneumonia visits; and standard deviation increases of NO₂ and CO were associated with 2-3% increases in chronic obstructive pulmonary disease visits. Positive associations persisted beyond 3 days for several of the outcomes, and over a week for asthma. The results of this study contribute to the evidence of an association of several correlated gaseous and particulate pollutants, including ozone, NO₂, CO, PM, and organic carbon, with specific respiratory conditions.

Emergency Room Visits, Asthma

The ozone coefficient and standard error are reported per 25 ppb increment of the maximum daily 8-hour average ozone level (Peel et al., 2003, Table 4). We used the results from the three cities combined. The relative risk is 1.022, with a 95 percent confidence interval of 0.996 to 1.049.

G.2.3.5 Sarnat et al. (2013)

The authors examined the influence of indoor air exchange rates as an effect modifier of associations between air pollutants and emergency department visits for asthma. The study analyzed associations between urban air pollutants (CO, NO_x, O₃, and PM_{2.5}) and asthma ED visits (ICD-9 codes 493 and 786.07) over a four-year period between 1999 and 2002 for residents of 186 zip codes in metropolitan Atlanta. A spatial surface of daily ambient air concentrations of pollutants was generated using AERMOD air quality modeling of 2002 NEI emissions combined with spatially interpolated background concentrations. AER was estimated at the zip code level based on an empirical model of direct and indirect predictors of AER collected from field surveys of building and meteorological characteristics. Effects of air pollution on asthma ED visits were estimated using a spatially resolved time series approach with Poisson generalized linear models, including stratified analyses for low or high AER areas. The authors found significant or near-significant positive effect modification of AER on the asthma ED visit effects of CO, NO_x, and PM_{2.5}. Ozone was a strong predictor of asthma ED visits but appeared unaffected by the AER. Overall, the study provides the first indication of short-term effect modification of air pollution risks with changes in AER. The HIF in BenMAP-CE is based on the association between ozone exposures and asthma ED visits for all AERs and all poverty levels (RR = 1.050, 95%CI 1.024-1.075) for a 26 ppb change in daily average ozone (3-day moving average of 0, 1, and 2 day lags). The beta value was adjusted to use the daily 8-hour max metric as described below.

G.2.3.6 Wilson et al. (2005)

Daily emergency room (ER) visits for all respiratory (ICD-9 460-519) and asthma (ICD-9 493) were compared with daily SO₂, ozone, and weather variables over the period 1998-2000 in Portland, Maine (population 248,000), and 1996-2000 in Manchester, New Hampshire (population 176,000). Seasonal variability was removed from all variables using nonparametric smoothed function (LOESS) of day of study. Generalized additive models were used to estimate the effect of elevated levels of pollutants on ER visits. Relative risks of pollutants were reported over their interquartile range (IQR, the 75th -25th percentile pollutant values). In Portland, an IQR increase in SO₂ was associated with a 5% (95% CI 2-7%) increase in all respiratory ER visits and a 6% (95% CI 1-12%) increase in asthma visits. An IQR increase in O₃ was associated with a 5% (95% CI 1-10%) increase in Portland asthmatic ER visits. No significant associations were found in Manchester, New Hampshire, possibly due to statistical limitations of analyzing a smaller population. The absence of statistical evidence for a relationship should not be used as evidence of no relationship. This analysis reveals that, on a daily basis, elevated SO₂ and O₃ have a significant impact on public health in Portland, Maine.

Emergency Room Visits, Asthma

The coefficient and standard error are taken from Wilson et al. (2005, Table 5).

G.2.4 Minor Effects

Table G-14 summarizes the additional 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 G-14. Additional 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	Chen et al.	2000	Washoe Co, NV	5-17	PM ₁₀ , CO	D8HourMax	0.015763	0.004985	Linear	All year, 8-hour max from 1-hour max.
School Loss Days, All Cause	Chen et al.	2000	Washoe Co, NV	5-17	PM ₁₀ , CO	D1HourMax	0.013247	0.004985	Linear	
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}	D8HourMax	0.002596	0.000776	Log-linear	8-hour max from 1-hour max.
Minor Restricted Activity Days	Ostro and Rothschild	1989	Nationwide	18-64	PM _{2.5}	D1HourMax	0.002200	0.000658	Log-linear	

G.2.4.1 Chen et al. (2000)

Chen et al. (2000) studied the association between air pollution and elementary school absenteeism (grades 1-6) in Washoe County, Nevada. Assuming that most children start kindergarten at age 5, the corresponding ages for grades 1 through 6 would be 6 through 11. Daily absence data were available for all elementary schools in the Washoe County School District. The authors regressed daily total absence rate on the three air pollutants, meteorological variables, and indicators for day of the week, month, and holidays. They reported statistically significant associations between both ozone and CO and daily total absence rate for grades one through six. PM₁₀ was negatively associated with absence rate, after adjustment for ozone, CO, and meteorological and temporal variables. The C-R function for ozone is based on the results from a multiple linear regression model with CO, ozone, and PM₁₀.

School Loss Days, All Cause

The coefficient and standard error are presented in Table 3 (Chen et al., 2000, p. 1008) for a unit ppm increase in the two-week average of daily one-hour maximum ozone concentration. This is converted to unit ppb increase by dividing by 1,000. The reported coefficient represents an absolute increase in absenteeism rate for a unit increase in ozone. If we apply this study to other locations, we assume that the same absolute increase will occur for a unit increase in ozone, regardless of the baseline rate. If the study location has a particularly high baseline rate, we may be overestimating decreases in absenteeism nationally, and vice-versa. As an example, consider if the baseline absenteeism rate were 10% in the study and 5% nationally. An absolute increase in absence rate of 2% associated with a given increase in ozone reflects a relative increase in absence rate of 20% for the study population. However, in the national estimate, we would assume the same absolute increase of 2%, but this would reflect a relative increase in the absenteeism rate of 40%.

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

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

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

Population: population of children ages 6-11

Scaling Factor 1: Ratio of national school absence rate to study-specific school absence rate = 1.081. (National school absence rate of 5.5% obtained from the U.S. Department of Education (1996, Table 42-1). Study-specific school absence rate of 5.09% obtained from Chen et al. (2000, Table 1).)

Scaling Factor 2: Convert beta in percentage terms to a proportion = 0.01

Scaling Factor 3: Proportion of days that are school days in the ozone season = 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 ($2.75/5 \times 5/7$.)

G.2.4.2 Gilliland et al. (2001)

See full study description under Gilliland et al. (2001) in Appendix F, Section F.4.1.

G.2.4.3 Ostro and Rothschild (1989)

See full study description under Ostro and Rothschild (1989) in Appendix F, Section F.4.2.

G.2.5 Asthma-Related Effects

Table G-15 summarizes the additional 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 G-15. Additional Health Impact Functions for Ozone and Asthma-Related Effects

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Asthma Exacerbation, One or More Symptoms	Mortimer et al.	2002	Eight U.S. urban areas	6-18		D8HourMax	0.009288	0.00387	Logistic	Uses incidence rate of 0.116
Asthma Exacerbation, One or More Symptoms	O'Connor et al.	2008	Seven U.S. urban communities	6-18	PM _{2.5} , NO ₂	D8HourMax	0.0009661	0.002991	Log-linear	Uses incidence rate of 0.207
Asthma Exacerbation, One or More Symptoms	Schildcrout et al.	2006	Eight U.S. urban areas	6-18		D8HourMax	0.00222	0.002822	Logistic	Uses incidence rate of 0.52

G.2.5.1 Mortimer et al. (2002)

Mortimer et al. (2002) examined the effects of daily levels of PM₁₀, SO₂, NO₂ and ozone on peak expiratory flow rate and asthma symptoms among 846 asthmatic children aged four to nine living in eight urban areas in the Northeast and Midwest. Exposures were estimated using EPA's AIRS database and averaging pollutant concentrations at all monitors in each subject's county. Effects were analyzed using mixed linear models and generalized estimating equation models. The authors found an association in single-pollutant models between each pollutant and self-reported morning asthma symptoms.

The health impact function in BenMAP is based on the odds ratio for a 15 ppb increase in 4-day average 8-hour mean ozone between June and August (OR=1.16, 95%CI 1.02-1.30), adjusted to 8-hour max exposure metric as described below.

G.2.5.2 O'Connor et al. (2008)

O'Connor et al. (2008) studied the association between asthma symptoms and daily outdoor air pollutant concentrations (NO₂, SO₂, CO, ozone, and PM_{2.5}) among 937 asthmatic children aged five to 12 in seven inner-city areas in the U.S. Exposure data were estimated from monitor data from EPA's AIRS database for the years 1998 to 2001. Outcome data were collected via telephone interview every two weeks. The effect of pollutant levels on frequency of asthma symptoms was assessed using generalized estimating equation models. The health impact function in BenMAP is based on the relative risk for a 26.7 ppb increase in 19-day average 8-hour mean ozone between June and August (OR=1.04, 95%CI 0.82-1.32), adjusted to the 8-hour max exposure metric as described below.

Incidence Rate: Daily incidence rate for days with wheeze, tightness in chest, cough 21% (Table 1).

G.2.5.3 Schildcrout et al. (2006)

Schildcrout et al. (2006) investigated the relation between ambient concentrations of the five criteria pollutants (PM₁₀, O₃, NO₂, SO₂, and CO) and asthma exacerbations (daily symptoms and use of rescue inhalers) among 990 children in eight North American cities during the 22-month prerandomization phase (November 1993-September 1995) of the Childhood Asthma Management Program. Short-term effects of CO, NO₂, PM₁₀, SO₂, and warm-season O₃ were examined in both one-pollutant and two-pollutant models, using lags of up to 2 days in logistic and Poisson regressions. When modeling ozone, the authors limited the study period to warm months (May through September).

Asthma Exacerbation, One or More Symptoms

In a single-pollutant model, Schildcrout et al. (2006, Figure 1) reported an odds ratio of 1.06 (95% CI: 0.92, 1.23) for daily asthma symptoms associated with 30 ppb change in 24-hr mean of ozone at lag 0.

Incidence Rate: Daily incidence rate for one or more symptoms (symptom score>0) = 52% (Schildcrout, et al., 2006, Table 1).

G.2.6 Converting Functions to 8-Hour Daily Maximum Metric

A number of health impact functions were converted from 1-hour maximum and 24-hour average to the 8-hour maximum metric. To convert, say, a 1-hour maximum function, we multiplied the 1-hour maximum coefficient with the ratio of the typical 1-hour maximum value to the typical 8-hour maximum value from Anderson and Bell (2010), Table 2. We approximated 8-hour average metric as equivalent to the 8-hour maximum metric due to high correlation between the two metrics.

Appendix H. Core Health Valuation Functions in U.S. Setup

This appendix presents the core unit values that are available in BenMAP for each of the health endpoints 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 endpoint. 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 endpoint. The definitions of the distributions and their parameters are given in Table H-1.

Table H-1. 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 ***	α	β

*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 $Y = \log_e X$, then X is lognormally distributed. Equivalently, X is lognormally distributed if $X = e^Y$, where Y is normally distributed.

*** The Weibull distribution has the following probability density function:

$$\left(\frac{\beta}{\alpha}\right)\left(\frac{x}{\alpha}\right)^{\beta-1} e^{-(x/\alpha)^\beta}$$

This appendix also presents EPA methods for developing income growth adjustment factors that allow BenMAP-CE users 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 endpoint while the undiscounted rate represents an additional valuation function.

Table H-2. 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 endpoints 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 endpoints.

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 endpoints 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-3. Core Unit Values Available for Hospital Admissions

Endpoint	ICD Codes	Age Range		Mean Hospital Charge (2015 \$)	Mean Length of Stay (days)	Total Cost of Illness (Unit Value in 2015\$)*
		Min	Max			
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 endpoints, Alzheimer’s and Parkinson’s Disease, we calculated the lifetime cost of illness 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-4. 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-5. 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

Year	3% Discount Rate	7% Discount Rate
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 endpoint. 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 endpoint in NIS to the same set of ICD-10 codes in NEDS.

For each health endpoint, 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-6. Core Unit Values Available for Emergency Department Visits

Endpoint	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-4.

Table H-4. 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 Endpoint Occurrence

Monetary valuation estimates for health endpoint 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-5).

Table H-5. Core Unit Values Available for non-fatal Lung Cancer

Health Endpoint	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-6. 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-6. 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-7 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-7. 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

Age Group	Percent New Cases per Year by Age*	Percent of New Cases Occurring at or After Age 55 ¹
55-99	92.2	100

*May not sum to 100% due to rounding

¹Calculated 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-8).

Table H-8. Valuation Estimate for Cardiac Arrests (2015\$)

Costs	Cumulative Costs	Annual Costs		
		Undiscounted	3% Discount Rate	7% Discount Rate
<i>Month of Event*</i>	\$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 endpoints 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-9 and H-10 summarize the values used by EPA in their regulatory impact analyses.

Table H-9. 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-10. Core Unit Values Available for Acute Symptoms and Illnesses

Health Endpoint	Basis for Estimate	Age Range		Unit Value (2015\$)	Distribution of Unit Value	Parameters of Distribution	
		Min	Max			P1	P2
Minor Restricted Activity Days	WTP: 1 day, CV studies	18	99	\$70	Triangular	28.51	110.62
New Onset	COI: lifetime med, lifetime	0	17	\$17,232	Normal	441.62	0

Health Endpoint	Basis for Estimate	Age Range		Unit Value (2015\$)	Distribution of Unit Value	Parameters of Distribution	
		Min	Max			P1	P2
Asthma	productivity, 3% DR	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
Asthma Symptoms (Albuterol Use), Albuterol Use	COI: use of inhaler	0	99	\$0.35	None	0	0
Allergic Rhinitis	COI: 1 yr med costs	0	17	\$600	None	0	0
Work Loss Days *	Median daily wage, county-specific	18	65	\$173	None	N/A	N/A
School Loss Days	Described in text, 3% DR	0	17	\$1000	None	N/A	N/A
	Described in text, 7% DR	0	17	\$610	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 endpoint itself is not precisely defined. Many different combinations of symptoms could presumably result in some minor or less minor restriction in activity. Krupnick and Kopp (1988) argued that mild symptoms will not be sufficient to result in a MRRAD, so that WTP to avoid a MRRAD should exceed WTP to avoid any single mild symptom. A single severe symptom or a combination of symptoms could, however, be sufficient to restrict activity. Therefore, WTP to avoid a MRRAD should, these authors argue, not necessarily exceed WTP to avoid a single severe symptom or a combination of symptoms. The “severity” of a symptom, however, is similarly not precisely defined; moreover, one level of severity of a symptom could induce restriction of activity for one individual

while not doing so for another. The same is true for any particular combination of symptoms.

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-CE 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 endpoint.

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 rhinitis (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 endpoint 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 $\$195 \times 0.93$ 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.

H.5 Developing Income Growth Adjustment Factors

Chapter 4 of the BenMAP-CE 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 7: Aggregating, Pooling, and Valuing.

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-CE 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-CE

includes income elasticity estimates specific to the type of health endpoint associated with the WTP estimate. BenMAP-CE 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 endpoint. Table H-11 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 endpoint.

Table H-11. Income Elasticity Estimates

Health Endpoint	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 endpoint. The adjustment formula follows four steps:

$$1) \quad \varepsilon = \frac{\frac{\Delta WTP}{WTP}}{\frac{\Delta I}{I}} = \frac{(WTP_2 - WTP_1) \times (I_2 + I_1)}{(I_2 - I_1) \times (WTP_2 + WTP_1)}$$

$$2) \quad \begin{aligned} \varepsilon I_2 WTP_2 + \varepsilon I_2 WTP_1 - \varepsilon I_1 WTP_2 - \varepsilon I_1 WTP_1 \\ = I_2 WTP_2 + I_1 WTP_2 - I_2 WTP_1 - I_1 WTP_1 \end{aligned}$$

$$3) \quad WTP_2 \times (\varepsilon I_2 - \varepsilon I_1 - I_2 - I_1) = WTP_1 \times (\varepsilon I_1 - \varepsilon I_2 - I_1 - I_2)$$

$$4) \quad WTP_2 = WTP_1 \times \frac{\varepsilon I_1 - \varepsilon I_2 - I_1 - I_2}{\varepsilon I_2 - \varepsilon I_1 - I_2 - I_1}$$

Table H-12 summarizes the income-based WTP adjustments used within BenMAP-CE for minor health endpoints, severe health endpoints, and premature mortality. BenMAP-CE applies the “mid” income growth adjustment to the WTP for each corresponding health endpoint. The “low” and “upper” are provided for bounding the “mid” estimate.

Table H-12. 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	1.213747	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	1.306133	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-CE 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-CE includes inflation factors for three different types of values. The source for these values is included in Table H-13. These values were re-indexed to \$2015 prior to import in BenMAP-CE.

Table H-13. Inflation Factors

Name	Description	Years	Source
All Goods Index	Value of generic goods and services	1980-2020	BLS, Data Series CUUR0000SA0 at http://data.bls.gov/cgi-bin/surveymost?cu
Medical Cost Index	Value of medical expenses		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 U.S. Setup

In this Appendix, we present additional health valuation functions. Unlike those in Appendix H, these functions are included in the U.S. Setup but are not currently used by the U.S. EPA in regulatory impact analyses. For the health valuation functions currently used by EPA, see the following page: <https://www.epa.gov/benmap/benmap-community-edition>. For Ozone Health Valuation Functions, click the “U.S. EPA approach for quantifying and valuing ozone effects” link. For PM_{2.5} Health Valuation Functions, click the “U.S. EPA approach for quantifying and valuing PM effects” link.

I.1 Mortality

I.1.1 Value of a Statistical Life Based on Selected Studies

In addition to the value of a statistical life based on the results of 26 studies in Appendix H, section H.1.1, we have included three alternatives based loosely on the results of work by Mrozek and Taylor (2002) and Viscusi and Aldy (2003). Each of these three alternatives has a mean value of \$7.6 million (2015\$), but with a different distribution: normal, uniform, triangular, and beta. Table H-10 presents the distribution parameters for these additional valuations in BenMAP.

Table I-1. Additional Unit Values for VSL for additional 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 2015\$ range from \$1.38 million to \$13.76 million – 95% CI of assumed normal distribution	0	99	7,570,229	Normal	3,160,172	–
VSL based on 2015\$ range from \$1.38 million to \$13.76 million – assumed uniform distribution	0	99	7,570,229	Uniform	1,376,405	13,764,053
VSL based on 2015\$ range from \$1.38 million to \$13.76 million – assumed triangular distribution	0	99	7,570,229	Triangular	1,376,405	13,764,053

* 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.

I.2 Hospital Admissions

This sub-section presents the unit values for hospital admissions that are not used by the EPA in regulatory impact analyses but are included in BenMAP. See Appendix H, Section H.2 for more information about these unit values. Table I-2 includes the unit

values for hospital admissions for endpoints included in BenMAP but not used by the EPA in their regulatory impact analyses.

Table I-2. Additional Unit Values Available for Hospital Admissions

Endpoint	ICD Codes	Age Range		Mean Hospital Charge (2015 \$)	Mean Length of Stay (days)	Total Cost of Illness (Unit Value in 2015\$)*
		Min	Max			
HA, All Cardiovascular	390-429	0	99	\$33,063	4.59	\$33,856
HA, All Cardiovascular	390-429	0	64	\$45,659	4.12	\$46,371
HA, All Cardiovascular	390-429	65	99	42,642	4.88	43,485
HA, Asthma	493	0	64	\$16,655	3.00	\$17,174
HA, Chronic Lung Disease	490-496	18	64	\$21,989	3.90	\$22,663
HA, Congestive Heart Failure	428	65	99	\$33,734	5.32	\$34,654
HA, Dysrhythmia	427	0	99	\$33,063	3.72	\$33,706
HA, Ischemic Heart Disease	410-414	65	99	\$55,591	4.61	\$56,388
HA, All Respiratory	460-519	0	1	\$16,929	3.19	\$17,480
HA, All Respiratory	460-519	0	99	\$32,563	5.35	\$33,488
HA, Asthma	493	65	99	\$26,153	4.79	\$26,981
HA, Asthma	493	0	99	\$18,590	3.37	\$19,172
HA, Chronic Lung Disease	490-496	65	99	\$25,413	4.79	\$26,241
HA, Chronic Lung Disease	490-496	0	99	\$22,312	4.10	\$23,021
HA, Chronic Lung Disease (less Asthma)	490-492, 494-496	18	64	\$23,980	4.23	\$24,711
HA, Chronic Lung Disease (less Asthma)	490-492, 494-496	65	99	\$25,254	4.79	\$26,082
HA, Chronic Lung Disease (Less Asthma)	490-492, 494-496	0	99	\$24,834	4.59	\$25,627
HA, Pneumonia	480-487	65	99	\$30,229	5.77	\$31,226
HA, Pneumonia	480-487	0	99	\$29,046	5.25	\$29,953

* 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."

I.3 Chronic Illness

This sub-section presents the unit values developed for chronic bronchitis, chronic asthma, and non-fatal myocardial infarctions.

I.3.1 Chronic Bronchitis

PM-related chronic bronchitis is expected to last from the initial onset of the illness throughout the rest of the individual's life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) as well as WTP to avoid the pain and suffering associated with the illness. Both WTP and COI estimates are currently available in BenMAP.

I.3.1.1 Unit Value Based on Two Studies of WTP

Two contingent valuation studies, Viscusi et al. (1991) and Krupnick and Cropper (1992), provide estimates of WTP to avoid a case of chronic bronchitis. Viscusi et al. (1991) and Krupnick and Cropper (1992) were experimental studies intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, they can be used to provide reasonable estimates of WTP to avoid a case of chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values. The Viscusi et al. and the Krupnick and Cropper studies are broadly consistent with current contingent valuation practices, although specific attributes of the studies may not be.

The study by Viscusi et al. (1991) uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (1992), which selects people who have a relative with the disease. However, the chronic bronchitis described to study subjects in the Viscusi study is severe, whereas a pollution-related case may be less severe.

The relationship between the severity of a case of chronic bronchitis and WTP to avoid it was estimated by Krupnick and Cropper (1992). We used that estimated relationship to derive a relationship between WTP to avoid a severe case of chronic bronchitis, as described in the Viscusi study, and WTP to avoid a less severe case. The estimated relationship (see Table 4 in Krupnick and Cropper) can be written as:

$$\ln(WTP) = \alpha + \beta \times sev$$

where α denotes all the other variables in the regression model and their coefficients, β is the coefficient of sev , estimated to be 0.18, and sev denotes the severity level (a number from 1 to 13). Let x (< 13) denote the severity level of a pollution-related case of chronic bronchitis, and 13 denote the highest severity level (as described in Viscusi et al., 1991). Then

$$\ln(WTP_{13}) = \alpha + \beta \times 13$$

and

$$\ln(WTP_x) = \alpha + \beta \times x$$

Subtracting one equation from the other,

$$\ln(WTP_{13}) - \ln(WTP_x) = \beta \times (13 - x)$$

or

$$\ln\left(\frac{WTP_{13}}{WTP_x}\right) = \beta \times (13 - x)$$

Exponentiating and rearranging terms,

$$WTP_x = WTP_{13} \times e^{-\beta \cdot (13-x)}$$

There is uncertainty surrounding the exact values of WTP13; x, and β , and this uncertainty can be incorporated in the equation, if you request that the analysis be carried out in “uncertainty mode.” The distribution of WTP to avoid a severe case of chronic bronchitis, WTP13, is based on the distribution of WTP responses in the Viscusi et al. (1991) study. The distribution of x, the severity level of an average case of pollution-related chronic bronchitis, is modeled as a triangular distribution centered at 6.5, with endpoints at 1.0 and 12.0. And the distribution of β is normal with mean = 0.18 and std. dev.= 0.0669 (the estimate of b and standard error reported in Krupnick and Cropper, 1992).

In uncertainty mode, BenMAP uses a Monte Carlo approach. On each Monte Carlo iteration, random draws for these three variables are made, and the resulting WTPx is calculated from the equation above. Because this function is non-linear, the expected value of WTP for a pollution-related case of CB cannot be obtained by using the expected values of the three uncertain inputs in the function (doing that will substantially understate mean WTP). A Monte Carlo analysis suggests, however, that the mean WTP to avoid a case of pollution-related chronic bronchitis is about \$470,000 (2015\$), but not adjusted for the growth of income). Therefore, if you request that the analysis be carried out in “point estimate” mode, that is the unit value that is used.

I.3.1.2 Alternative Cost of Illness Estimates

Cost of illness estimates for chronic bronchitis were derived from estimates of annual medical costs and annual lost earnings by Cropper and Krupnick (1999). This study estimated annual lost earnings resulting from chronic bronchitis as a function of age at onset of the illness, for the following age categories: 25-43, 35-44, 45-54, and 55-65 (see Cropper and Krupnick, Table 8). Annual medical expenses were estimated for 10-years age groups (0-9, 10-19, 20-29, ..., 80-89). We derived estimates of the present discounted value of the stream of medical and opportunity costs for people whose age of onset is 30, 40, 50, 60, 70, and 80. Medical costs (which are in 1977\$ in the Cropper and Krupnick study) were inflated to 2015\$ using the CPI-U for medical care; lost earnings (opportunity costs) were inflated to 2015\$ using the Employment Cost Index for Wages and Salaries. Life expectancies were assumed to be unaffected by the illness. For example, an individual at age 70 has a life expectancy of 14.3 more years, and we assumed that someone whose age of onset of chronic bronchitis is 70 will also live for 14.3 more years. (Source of life expectancies: National Center for Health Statistics, 1999, Table 5.) We also assumed that opportunity costs at ages 66 and over were zero. Present discounted values were calculated using three and seven percent discount rates.

For each of the two discount rates, there are three cost of illness unit values for chronic bronchitis available in BenMAP, for the following age categories: 27-44, 45-64, and 65+. These are the age categories that were used in the epidemiological study that estimated a concentration-response function for chronic bronchitis (Abbey et al., 1995b). The estimate for the 27-44 age group is an average of the present discounted values calculated for ages 30 and 40; the estimate for the 45-64 age category is an average of the present discounted values calculated for ages 50 and 60; and the estimate for the 65+ age category is an average of the present discounted values calculated for ages 70 and 80. The suite of unit values available for use in BenMAP is shown in Table I-3.

Table I-3. Additional Unit Values Available for Chronic Bronchitis

Basis for Estimate	Age of Onset		Present Discounted Value of Medical Costs	Present Discounted Value of Opportunity Costs	Unit Value	Distribution
	Min	Max				
WTP: average severity	30	99	N/A	N/A	\$468,641	Custom
COI: med costs + wage loss, 3% DR	27	44	\$32,478	\$199,479	\$231,947	None
	45	64	\$40,699	\$111,959	\$152,658	None
	65	99	\$18,993	\$0	\$18,993	None
COI: med costs + wage loss, 7% DR	27	44	\$13,509	\$118,460	\$131,969	None
	45	64	\$24,651	\$87,732	\$112,383	None
	65	99	\$15,468	\$0	\$15,468	None

I.3.2 Chronic Bronchitis Reversals

The unit value for chronic bronchitis reversals assumes that this is chronic bronchitis with a severity level of 1. The method for generating a distribution of unit values in BenMAP is therefore the same as the WTP-based unit value method for chronic bronchitis (see above), with $x = 1$. The mean of this distribution is \$206,765.

I.3.3 Chronic Asthma

Two studies have estimated WTP to avoid chronic asthma in adults. Blumenschein and Johannesson (1998) used two different contingent valuation (CV) methods, the dichotomous choice method and a bidding game, to estimate mean willingness to pay for a cure for asthma. The mean WTP elicited from the bidding game was \$189 per month, or \$2,268 per year (in 1996\$). The mean WTP elicited from the dichotomous choice approach was \$343 per month, or \$4,116 per year (in 1996\$). Using \$2,268 per year, a three percent discount rate, and 1997 life expectancies for males in the United States (National Center for Health Statistics, 1999, Table 5), the present discounted value of the stream of annual WTPs is \$65,568 (in 2015\$).

O'Connor and Blomquist (1997) estimated WTP to avoid chronic asthma from estimates of risk-risk tradeoffs. Combining the risk-risk tradeoffs with a statistical value of life, the annual value of avoiding asthma can be derived. Assuming a value of a statistical life of

\$6 million, they derived an annual WTP to avoid asthma of \$1500 (O'Connor and Blomquist, 1997, p. 677). For a value of a statistical life of \$5,894,400 (in 1997 \$), the corresponding implied annual value of avoiding chronic asthma, based on O'Connor and Blomquist would be \$1,474. Assuming a three percent discount rate and 1997 life expectancies for males in the United States, the present discounted value of the stream of annual WTPs would be \$41,646 (in 2015\$). A unit value, based on a three percent discount rate, is the average of the two estimates, or \$53,607. Following the method used for the §812 Prospective analysis, the uncertainty surrounding the WTP to avoid a case of chronic asthma among adult males was characterized by a triangular distribution on the range determined by the two study-specific WTP estimates. A second unit value, using a seven percent discount rate, is also available for use in BenMAP. The method used to derive this unit value is the same as that described above for the three percent discount rate unit value. The unit values available for use in BenMAP are summarized in Table I-4 below.

Table I-4. Additional Unit Values Available for Chronic Asthma

Basis for Estimate	Age Range		Unit Value	Distribution of Unit Value	Parameters of Distribution	
	Min	Max			P1	P2
WTP: 3% DR (Discount Rate)	27	99	\$53,607	Triangular	\$41,646	\$65,568
WTP: 7% DR	27	99	\$34,901	Triangular	\$27,114	\$42,689

I.4 Acute Symptoms and Illness Not Requiring Hospitalization

See Appendix H, Section H.3 for a general explanation of acute symptoms and illness not requiring hospitalization. Table I-5 summarizes unit values for acute bronchitis in children, acute respiratory symptoms (minor restricted activity days), any of 19 respiratory symptoms, upper respiratory symptoms, lower respiratory symptoms, and work loss days (WLDs)

Table I-5. Additional Unit Values Available for Acute Symptoms and Illnesses (in 2015 \$)

Health Endpoint	Basis for Estimate *	Age Range		Unit Value	Distribution of Unit Value	Parameters of Distribution	
		Min	Max			P1	P2
Acute Bronchitis	WTP: 1 day illness, CV studies	0	17	\$82	Uniform	24.10	139.16
	WTP: 6 day illness, CV studies	0	17	\$490	Uniform	144.60	834.98
	WTP: 28 symptom-days, Dickie and Ulery	0	17	\$529	Lognormal	6.27	0.0907
Minor Restricted Activity Days	WTP: 3 symptoms 1 day, Dickie and Ulery (2002)	18	99	\$138	Lognormal	4.93	0.0649
Lower Respiratory Symptoms	WTP: 1 day, CV studies	0	17	\$21	Uniform	9.56	33.68
	WTP: 2 symptoms 1 day, Dickie and Ulery (2002)	0	17	\$264	Lognormal	5.58	0.07048

Health Endpoint	Basis for Estimate *	Age Range		Unit Value	Distribution of Unit Value	Parameters of Distribution	
		Min	Max			P1	P2
	WTP: 2x1 day, CV studies	0	17	\$42	Uniform	19.12	67.36
Upper Respiratory Symptoms	WTP: 1 day, CV studies	0	17	\$34	Uniform	12.29	59.34
	WTP: 2 symptoms 1 day, Dickie and Ulery (2002)	0	17	\$264	Lognormal	5.58	0.07048
	WTP: 2x1 day, CV studies	0	17	\$68	Uniform	24.58	118.68
Any of 19 Respiratory Symptoms	WTP: 1 day illness, CV studies	1	65	\$33	Uniform	0	66.41

* All unit values pulled from a lognormal distribution from Model 1, Table III in Dickie and Ulery are multiplied by 0.973811 to adjust for a difference in mean household income between the study participants and the general population. The unit values shown here have already been adjusted.

I.4.1 Non-Fatal Myocardial Infarctions (Heart Attacks)

See Appendix H, Section H.3.1 for an explanation of this valuation function.

Using age-specific annual lost earnings estimated by Cropper and Krupnick (1999), and a three percent discount rate, we estimated the following present discounted values in lost earnings over 5 years due to a heart attack (2015\$): \$13,301 for someone between the ages of 25 and 44, \$19,604 for someone between the ages of 45 and 54, and \$113,316 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings using a seven percent discount rate are \$11,908, \$17,552, and \$101,451, respectively. Cropper and Krupnick do not provide lost earnings estimates for populations under 25 or over 65. As such we do not include lost earnings in the cost estimates for these age groups.

We have found three additional sources of estimates of the direct medical costs of a myocardial infarction (MI) in the literature:

- Wittels et al. (1990) estimated expected total medical costs of MI over 5 years to be \$51,211 (in 1986\$) for people who were admitted to the hospital and survived hospitalization. (There does not appear to be any discounting used.) Wittels et al. was used to value coronary heart disease in the 812 Retrospective Analysis of the Clean Air Act. Using the CPI-U for medical care, the Wittels estimate is \$187,530 in year 2015\$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes and prices (using “knowledgeable cardiologists” as consultants). The model used medical data and medical decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The authors note that the average length of hospitalization for acute MI has decreased over time (from an average of 12.9 days in 1980 to an average of 11 days in 1983). Wittels et al. used 10 days as the average in their study. It is unclear how much further the length of stay (LOS) for MI may have decreased from 1983 to the present. The average LOS for ICD code 410 (MI) in the year-2000 AHQR HCUP database is 5.5 days. However, this may include patients who died in the hospital (not included among our non-fatal MI

cases), whose LOS was therefore substantially shorter than it would be if they hadn't died.

- Eisenstein et al. (2001) estimated 10-year costs of \$44,663, in 1997\$ (using a three percent discount rate), or \$85,052 in 2015\$ for MI patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included. Estimates from Eisenstein et al. are included in Appendix I.4 as they are not used in EPA impact analyses.
- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal MI of \$15,540 (in 1995\$), and \$1,051 annually thereafter. Converting to year 2015\$, that would be \$38,253 for a 5-year period, using a three percent discount rate, or \$36,167, using a seven percent discount rate.

The age group-specific estimates of opportunity cost over a five-year period are combined with the medical cost estimates from each of the three studies listed above. Because opportunity costs are derived for each of five age groups, there are $3 \times 5 = 15$ unit values for each of 2 discount rates, or 30 unit values available for use in BenMAP. These are given in Table I-6 below.

Note that we were unable to achieve complete consistency, unfortunately, because of limitations in the input studies. For example, although we calculated opportunity costs over a five-year period using a 3 percent and a 7 percent discount rate, we were not able to do the same for medical costs, except for the medical costs estimated by Russell et al. (in which they estimate an annual cost). Wittels et al. appear to have used no discounting in their estimate; Eisenstein et al. used a 3 percent discount rate. Similarly, although almost all cost estimates (opportunity costs and medical costs) are for a 5-year period, the medical cost estimate reported by Eisenstein et al. is for a 10-year period. There was no reasonable method for inferring from that study what costs over a 5-year period would be.

Table I-6. Additional Unit Values Available for Myocardial Infarction

Basis of Estimate	Age Range		Medical Cost *	Opportunity Cost **	Total Cost
	Min	Max			
COI: 5 yrs med, 5 yrs wages, 3% DR, Wittels (1990)	0	24	\$187,530	\$0	\$187,530
	25	44	\$187,530	\$13,301	\$200,831
	45	54	\$187,530	\$19,604	\$207,134
	55	65	\$187,530	\$113,316	\$300,846
	66	99	\$187,530	\$0	\$187,530
COI: 5 yrs med, 5 yrs wages, 3% DR, Russell (1998)	0	24	\$38,253	\$0	\$38,253
	25	44	\$38,253	\$13,301	\$51,554
	45	54	\$38,253	\$19,604	\$57,857
	55	65	\$38,253	\$113,316	\$151,569
	66	99	\$38,253	\$0	\$38,253
COI: 10 yrs med, 5 yrs wages,	0	24	\$85,052	\$0	\$85,052

Basis of Estimate	Age Range		Medical Cost *	Opportunity Cost **	Total Cost
	Min	Max			
3% DR, Eisenstein (2001)	25	44	\$85,052	\$13,301	\$98,353
	45	54	\$85,052	\$19,604	\$104,656
	55	65	\$85,052	\$113,316	\$198,368
	66	99	\$85,052	\$0	\$85,052
COI: 5 yrs med, 5 yrs wages, 7% DR, Wittels (1990)	0	24	\$187,530	\$0	\$187,530
	25	44	\$187,530	\$11,908	\$199,438
	45	54	\$187,530	\$17,552	\$205,082
	55	65	\$187,530	\$101,451	\$288,981
	66	99	\$187,530	\$0	\$187,530
COI: 5 yrs med, 5 yrs wages, 7% DR, Russell (1998)	0	24	\$36,167	\$0	\$36,167
	25	44	\$36,167	\$11,908	\$48,075
	45	54	\$36,167	\$17,552	\$53,719
	55	65	\$36,167	\$101,451	\$137,618
	66	99	\$36,167	\$0	\$36,167
COI: 10 yrs med, 5 yrs wages, 7% DR, Eisenstein (2001)	0	24	\$85,052	\$0	\$85,052
	25	44	\$85,052	\$11,908	\$96,960
	45	54	\$85,052	\$17,552	\$102,604
	55	65	\$85,052	\$101,451	\$186,503
	66	99	\$85,052	\$0	\$85,052

* An average of the 5-year costs estimated by Wittels et al. (1990) and Russell et al. (1998). Note that Wittels et al. appears not to have used discounting in deriving a 5-year cost of \$187,530; Russell et al. estimated first-year direct medical costs and annual costs thereafter. The resulting 5-year cost is \$38,253, using a 3% discount rate, and \$36,167, using a 7% discount rate. Medical costs were inflated to 2015\$ using CPI for medical care.

** From Cropper and Krupnick (1999). Present discounted value of 5 yrs of lost earnings, at 3% and 7% discount rate, adjusted from 1977\$ to 2015\$ using CPI-U "all items".

I.4.2 Acute Bronchitis in Children

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore may be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

Three unit values are available in BenMAP for acute bronchitis in children. In previous benefit analyses, EPA used a unit value of \$81.63 (adjusted to 2015\$). This is the midpoint between a low estimate and a high estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. For a more complete description of the derivation of this estimate, see Abt Associates (2000, p. 4-

30). The above unit value assumes that an episode of acute bronchitis lasts only one day. However, this is generally not the case, as acute bronchitis typically lasts 6 or 7 days. To generate a unit value representative of an entire acute bronchitis episode, the original unit value of \$81.63 was multiplied by 6 or 7. A unit value of \$490 ($=\81.63×6) was therefore derived.

As discussed the epidemiological study relating air pollution to the incidence of acute bronchitis referred to children specifically. The value of an avoided case should therefore be WTP to avoid a case in a child, which may be different from WTP to avoid a case in an adult. Recent work by Dickie and Ulery (2002) suggests, in fact, that parents are generally willing to pay about twice as much to avoid sickness in their children as in themselves. In one of several models they estimated, the natural logarithm of parents' WTP was related both to the number of symptom-days avoided and to whether it was their child or themselves at issue. Dickie and Ulery noted that "experiencing all of the symptoms [considered in their study - cough and phlegm, shortness of breath/wheezing, chest pain, and fever] for 7 days, or 28 symptom-days altogether, is roughly equivalent to a case of acute bronchitis ..." Using this model, and assuming that a case of acute bronchitis can be reasonably modeled as consisting of 28 symptom-days, we estimated parents' WTP to avoid a case of acute bronchitis in a child to be \$529. This is the third unit value available in BenMAP.

The mean household income among participants in the Dickie and Ulery CV survey was slightly higher than the national average. We therefore adjusted all WTP estimates that resulted from their models downward slightly, using an income elasticity of WTP of 0.147, the average of the income elasticities estimated in the four models in the study. The adjustment factor thus derived was 0.9738. Estimates for Acute Bronchitis are available in Table I-5.

I.4.3 Minor Restricted Activity Days (MRADs)

See Appendix H, Section H.3.3 for more information.

In addition to the estimate of WTP to avoid a MRRAD used in EPA benefits analyses, a second unit value is based on Model 1, Table III in Dickie and Ulery (2002). This model estimates the natural logarithm of parents' WTP to avoid symptoms as a linear function of the natural logarithm of the number of symptom-days avoided and whether or not the person avoiding the symptoms is the parent or the child. The unit value derived from this model, assuming that an MRAD consists of one day of 3 symptoms in an adult, is \$138.

The estimate for the MRADs that is not used in EPA benefits analyses can be found in Table I-5.

I.4.4 Asthma Exacerbation

Table I-7 below describes the unit values for Asthma-related Acute Symptoms and Illnesses included in BenMAP but not used by the EPA for their regulatory impact analyses. All unit values for this section are summarized in Table I-7.

Several respiratory symptoms in asthmatics or characterizations of an asthma episode have been associated with exposure to air pollutants. All of these can generally be taken as indications of an asthma exacerbation (“asthma attack”) when they occur in an asthmatic. BenMAP therefore uses the same set of unit values for all of the variations of “asthma exacerbation” that appear in the epidemiological literature.

In Rowe and Chestnut (1986), the mean of the four average WTPs is \$32 (1990 \$), or \$59 in 2015\$. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [\$12, \$54] in 1990\$, or [\$21, \$98] in 2015\$. In previous benefit analyses, the EPA only used the unit value for asthma exacerbation in children from Rowe and Chestnut (1986) for avoiding a “bad asthma day”. There are two other unit values for children and two unit values for adults included in BenMAP discussed further below.

The first unit value for adult is based on willingness to pay to avoid an asthma exacerbation from four WTP estimates from Rowe and Chestnut (1986) for avoiding a “bad asthma day.” The second unit value for adults was derived by using Model 1, Table III in Dickie and Ulery (2002) -- the same model used for acute bronchitis, LRS, and URS -- assuming that an asthma exacerbation consists of 1 symptom-day. As noted above, this model relates parental WTP to the number of symptom-days avoided and to whether it is the parent or the child at issue. The unit value derived from this model for adults is \$74.

Two additional unit values are available for children. One of these is twice the original unit value, or \$104, based on the evidence from Dickie and Ulery (2002) that parents are willing to pay about twice as much to avoid symptoms and illness in their children as in themselves. The third unit value is based on Model 1, Table III in Dickie and Ulery (the same model used for asthma exacerbation in adults, only now with the “adult or child” variable set to 1 rather than 0). The unit value derived from this model is \$221.

Table I-7. Additional Unit Values Available for Asthma-related Acute Symptoms and Illnesses

Health Endpoint	Basis for Estimate *	Age Range		Unit Value	Unit Value Distribution	Parameters of Distribution	
		Min	Max			P1	P2
Asthma Attacks; Cough; Moderate or Worse; One or more symptoms; Shortness of Breath; Wheeze	Bad asthma day, Rowe and Chestnut (1986)	18	99	\$59	Uniform	21.42	97.56
	1 symptom-day, Dickie and Ulery (2002)	18	99	\$104	Lognormal	4.64	0.0957
	2 x bad asthma day, Rowe and Chestnut (1986)	0	17	\$118	Uniform	42.84	195.12
	1 symptom-day, Dickie and Ulery (2002)	0	17	\$221	Lognormal	5.39	0.0925
Cough; Moderate or	Bad asthma day, Rowe and Chestnut (1986)	0	17	\$59	Uniform	21.42	97.56

Health Endpoint	Basis for Estimate *	Age Range		Unit Value	Unit Value Distribution	Parameters of Distribution	
		Min	Max			P1	P2
Worse; One or more symptoms; Shortness of Breath; Wheeze							

* All unit values pulled from a lognormal distribution from Model 1, Table III in Dickie and Ulery, 2002, are multiplied by 0.973811 to adjust for a difference in mean household income between the study participants and the general population. The unit values shown here have already been adjusted.

I.4.5 Upper Respiratory Symptoms (URS) in Children

In past benefit analyses, EPA based willingness to pay to avoid a day of URS on symptom-specific WTPs to avoid those symptoms identified as part of the URS complex of symptoms. Pope et al. (1991) defined a day of URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. The three contingent valuation (CV) studies shown in Table I-8 have estimated WTP to avoid various morbidity symptoms that are either within the URS symptom complex defined by Pope et al., or are similar to those symptoms.

Table I-8. Median WTP Estimates and Derived Midrange Estimates (in 2015\$)

Symptom*	Dickie et al.	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	6.84	29.65	-	18.14
Head/sinus congestion	7.98	31.94	14.87	18.14
Coughing	2.29	25.11	9.03	12.70
Eye irritation	-	28.50	-	28.50
Headache	2.29	45.63	-	18.14
Shortness of breath	0.00	-	19.16	9.06
Pain upon deep inhalation (PDI)	8.01	-	-	8.01
Wheeze	4.57	-	-	4.57
Coughing up phlegm	4.99	-	-	4.99
Chest tightness	11.42	-	-	11.42

* All estimates are WTP to avoid one day of symptom. Midrange estimates were derived by IEC (1993).

** 10% trimmed mean.

The three individual symptoms that were identified as most closely matching those listed by Pope et al. for URS are cough, head/sinus congestion, and eye irritation, corresponding to “wet cough,” “runny or stuffy nose,” and “burning, aching or red eyes,” respectively. A day of URS could consist of any one of the seven possible “symptom complexes” consisting of at least one of these three symptoms. The original unit value for URS was based on the assumption that each of these seven URS complexes is equally likely. This unit value for URS, \$33.91, is just an average of the seven estimates of mean WTP for the different URS complexes. This unit value can be found in Table I-5. In

addition to the one unit value that EPA used in past benefit analyses, two other unit values can be found in BenMAP. Recent research by Dickie and Ulery (2002) suggests that parental WTP to avoid symptoms and illnesses in their children is about twice what it is to avoid those symptoms and illnesses in themselves. This second unit value of \$67.82 (=2 x \$33.91) is derived from the unit value found in Appendix H, Section H.3.4.

Another unit value was derived by using Model 1, Table III in Dickie and Ulery (2002) (the same model used for acute bronchitis), assuming that a day of URS consists of 2 symptoms. As noted above, this model relates parental WTP to the number of symptom-days avoided and to whether it is the parent or the child at issue. The unit value derived from this model is \$264.

A WTP estimate elicited from parents concerning their WTP to avoid symptoms in their children may well include some calculation of lost earnings resulting from having to lose a day of work. Estimates from the Dickie and Ulery model therefore (appropriately) probably include not only their WTP to have their children avoid the pain and suffering associated with their illness, but also the opportunity cost of a parent having to stay home with a sick child. Unit values can be found in Table I-5.

I.4.6 Lower Respiratory Symptoms (LRS) in Children

The three unit values for LRS in children currently available in BenMAP follow the same pattern as those for URS in children. In past benefit analyses, EPA based willingness to pay to avoid a day of LRS on symptom-specific WTPs to avoid those symptoms identified as part of the LRS complex of symptoms. Schwartz et al. (1994) defined a day of LRS as consisting of at least two of the following symptoms: cough, chest tightness, coughing up phlegm, and wheeze. Of the symptoms for which WTP estimates are available (listed in Table H-8), those that most closely match the symptoms listed by Schwartz et al. are coughing, chest tightness, coughing up phlegm, and wheeze. A day of LRS, as defined by Schwartz et al., could consist of any one of 11 possible combinations of at least two of these four symptoms. In the absence of any further information, each of the 11 possible "symptom clusters" was considered equally likely. The unit value for LRS, \$21.43, is just an average of the eleven estimates of mean WTP for the different LRS symptom clusters. In addition to the original value used by the EPA in past benefits analyses, BenMAP contains a second unit value is twice the original unit value, or \$42.86. This value is based on the evidence from Dickie and Ulery (2002) that parents are willing to pay about twice as much to avoid symptoms and illness in their children as in themselves. The third unit value is based on Model 1, Table III in Dickie and Ulery, assuming that, as for URS, a day of LRS consists of 2 symptoms. As noted above, this model relates parental WTP to the number of symptom-days avoided and to whether it is the parent or the child at issue. The unit value derived from this model is \$264. These two additional unit values can be found in Table I-5.

I.4.7 Any of 19 Respiratory Symptoms

The presence of “any of 19 acute respiratory symptoms” is a somewhat subjective health effect used by Krupnick et al. (1990). Moreover, not all 19 symptoms are listed in the Krupnick et al. study. It is therefore not clear exactly what symptoms were included in the study. Even if all 19 symptoms were known, it is unlikely that WTP estimates could be obtained for all of the symptoms. Finally, even if all 19 symptoms were known and WTP estimates could be obtained for all 19 symptoms, the assumption of additivity of WTPs becomes tenuous with such a large number of symptoms. The likelihood that all 19 symptoms would occur simultaneously, moreover, is very small.

Acute respiratory symptoms must be either upper respiratory symptoms or lower respiratory symptoms. In the absence of further knowledge about which of the two types of symptoms is more likely to occur among the “any of 19 acute respiratory symptoms,” we assumed that they occur with equal probability. Because this health endpoint may also consist of combinations of symptoms, it was also assumed that there is some (smaller) probability that upper and lower respiratory symptoms occur together. To value avoidance of a day of “the presence of any of 19 acute respiratory symptoms” we therefore assumed that this health endpoint consists either of URS, or LRS, or both. We also assumed that it is as likely to be URS as LRS and that it is half as likely to be both together. That is, it was assumed that “the presence of any of 19 acute respiratory symptoms” is a day of URS with 40 percent probability, a day of LRS with 40 percent probability, and a day of both URS and LRS with 20 percent probability. Using the point estimates of WTP to avoid a day of URS and LRS derived above, the point estimate of WTP to avoid a day of “the presence of any of 19 acute respiratory symptoms” is:

$$(0.40)(\$33.91) + (0.40)(\$21.43) + (0.20)(\$33.91 + \$21.43) = \$33.20.$$

Because this health endpoint is only vaguely defined, and because of the lack of information on the relative frequencies of the different combinations of acute respiratory symptoms that might qualify as “any of 19 acute respiratory symptoms,” the unit dollar value derived for this health endpoint must be considered only a rough approximation. The value for any of 19 respiratory symptoms can be found in Table I-5.

Appendix J. Population & Other Data in U.S. Setup

This section describes the population, monitor, and demographic data in the United States setup. 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.
- **Monitor Data.** The default United States setup has ozone, PM_{2.5}, PM₁₀, lead, NO₂, and SO₂ monitor data for the years 2000-2007. Data for CO are available at the BenMAP website: <http://www.epa.gov/air/benmap/>.
- **Demographic Datasets.** This subsection describes the various datasets in the U.S. setup related to demography: household size, poverty rates, and educational attainment.

J.1 Population Data in U.S. Setup

The U.S. setup in 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 population level 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-CE 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-1. 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 population data in the U.S. setup, 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:

$$age_{3-12} = \frac{1}{2} \times age_{1-4} + age_{5-9} + \frac{3}{5} \times age_{10-14}$$

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 until 2050 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:

$$age_{4-9,g,2020} = age_{4-9,g,2010} \times \frac{age_{4-9,county,2020}}{age_{4-9,county,2010}}$$

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:

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

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:

$$age_{4-9,g\ in\ county_c,2010} = age_{4-9,g,2010} \times fraction\ age_{4-9,g\ in\ county_c}$$

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:

$$age_{4-9,g \text{ in county}_c, 2020} = age_{4-9,g \text{ in county}_c, 2010} \times \frac{age_{4-9, \text{county}_c, 2020}}{age_{4-9, \text{county}_c, 2010}}$$

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:

$$age_{4-9,g, 2020} = \sum_{c=1}^n age_{4-9,g, 2010} \times \frac{total \ pop_{g \text{ in county}_c}}{total \ pop_g} \times \frac{age_{4-9, \text{county}_c, 2020}}{age_{4-9, \text{county}_c, 2010}}$$

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:

$$age_{4-9,g, 2020} = \sum_{c=1}^n age_{4-9,g, 2010} \times \frac{total \ pop_{g \text{ in county}_c}}{total \ pop_g} \times \frac{age_{4-9, \text{county}_c, 2020}}{age_{4-9, \text{county}_c, 2010}}$$

$$age_{10-14,g, 2020} = \sum_{c=1}^n age_{10-14,g, 2010} \times \frac{total \ pop_{g \text{ in county}_c}}{total \ pop_g} \times \frac{age_{10-14, \text{county}_c, 2020}}{age_{10-14, \text{county}_c, 2010}}$$

$$age_{4-12,g, 2020} = age_{4-9,g, 2020} + \frac{3}{5} \times age_{10-14,g, 2020}$$

To estimate population for 2055, we extrapolate Woods and Poole projections from the 2045 to 2050 period:

$$W_{2055,i} = W_{2050,i} * \frac{W_{2050,i}}{W_{2045,i}}$$

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 in the U.S. setup, and is not something that the user needs to load herself. These data are simply county-level ratios of a year (2009, 2011-2055) 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-2. 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-level SF1 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		Census 2000	
	Number	Percent	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.⁴²

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.

Calculating Growth Ratios with Zero Population in 2010

There are a small number of cases where 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.1.3 PopGrid

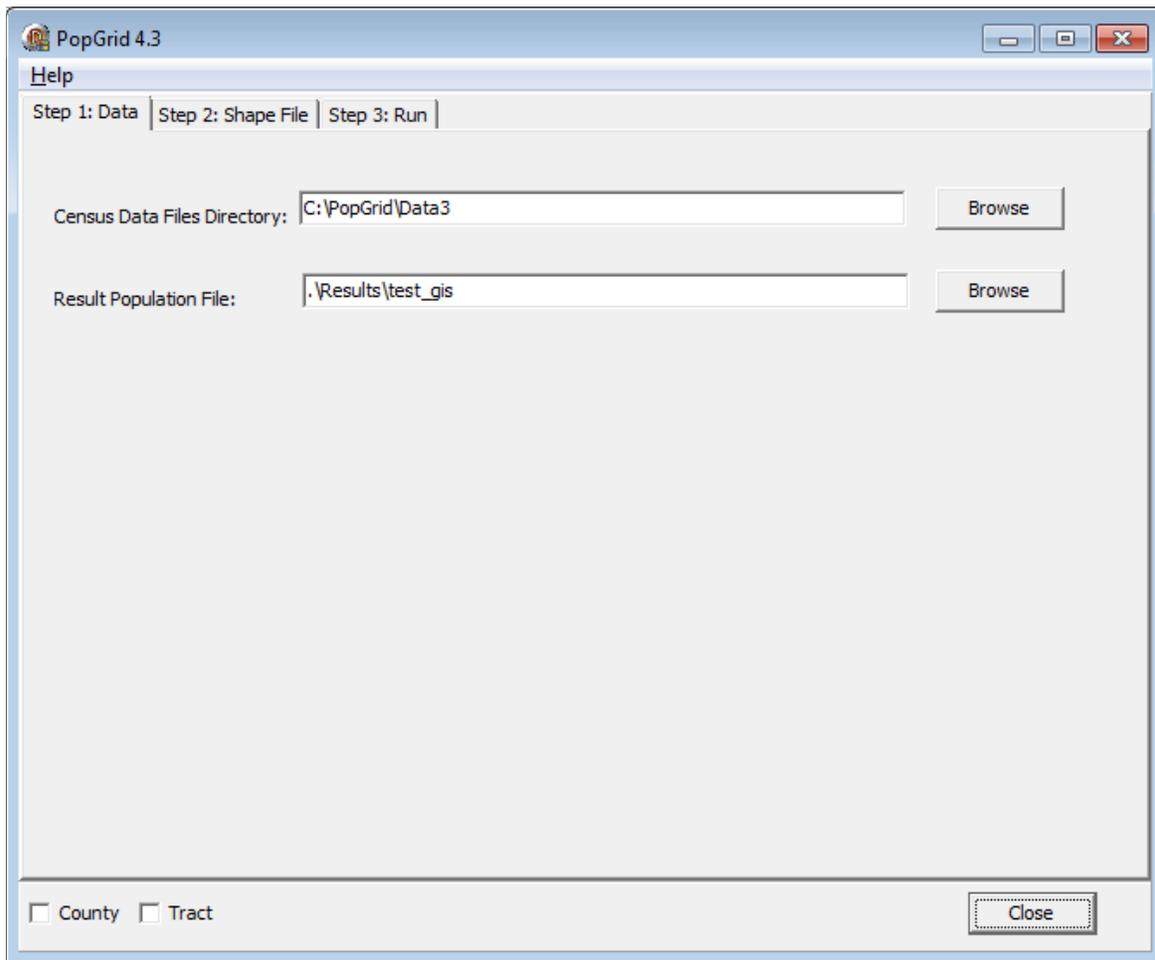
If the geographic center of a Census block falls within a population grid-cell, PopGrid assigns the block population to this particular population grid-cell. Note that the grid-cells in an air quality model, such as CMAQ, may cross multiple county boundaries. PopGrid keeps track of the total number of people in each race-ethnic group by county within a particular population grid-cell. Of course, when the population grid-cell is for U.S. counties, then there is only a single county associated with the population grid-cell. However, with air quality models, there can clearly be multiple counties in a population grid-cell.

⁴² Previous versions of the BenMAP-CE 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.

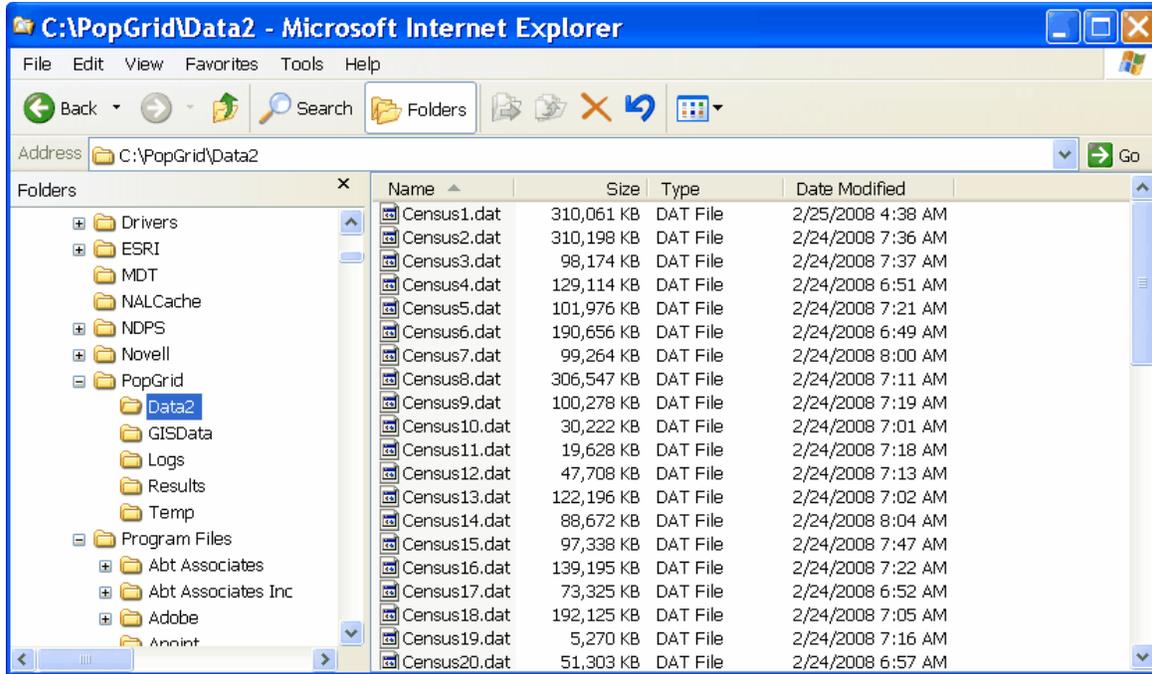
Keeping track of the total number of people in a county is necessary when forecasting population, as the population forecast for a given grid cell is equal to the year 2010 population estimate from the Census Bureau multiplied by the ratio of future-year to year 2010 county population estimates from Woods & Poole. BenMAP assumes that all age-gender groups within a given race-ethnic group have the same geographic distribution.

J.1.3.1 How to Use PopGrid

After installing PopGrid, double-click on the PopGrid executable “PopGrid4.exe.” The following screen will appear:

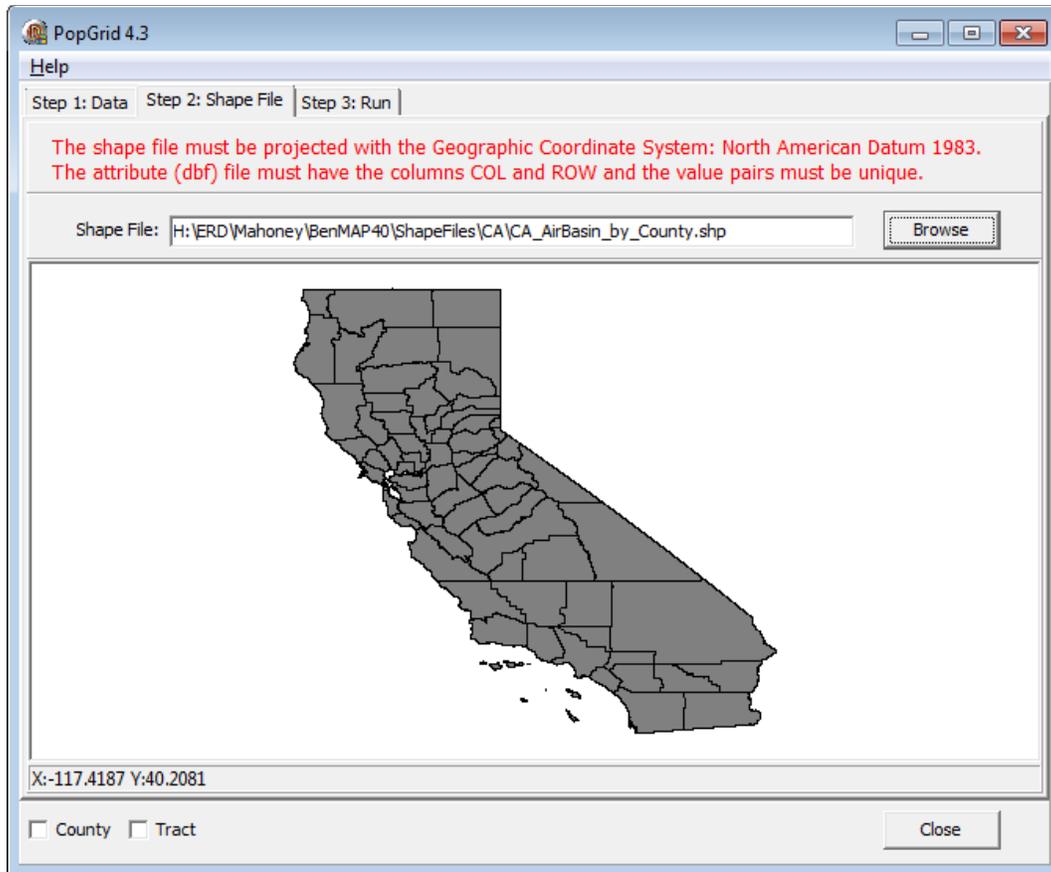


The **Census Data Files Directory** box points PopGrid to where the block data are located that PopGrid uses. Make sure that the files in this directory are unzipped. This data folder should look something like the following:

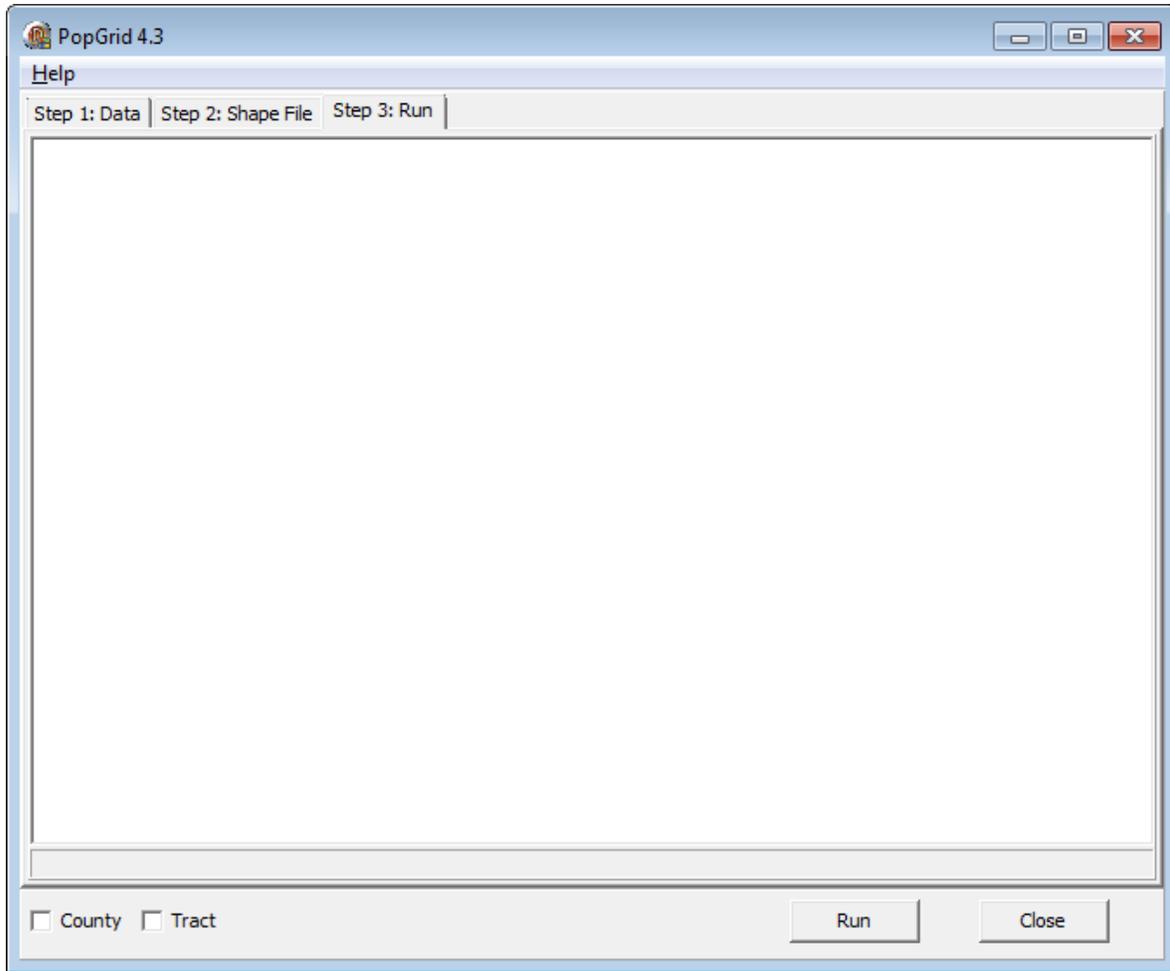


The **Result Population File** box provides the path and the name of the file that you want to create. In the example above, PopGrid is being used to estimate population for the intersection of air basins and counties in California (CA_AirBasin_by_County).

Click on the **Step 2: Shape File** tab. Choose the shapefile that you want to use. The example for air basins and counties in California looks as follows:



After choosing your shapefile, which must contain a column/row index, go to the **Step 3: Run** tab, which should look as follows:



Click **Run**. PopGrid will now begin processing. It can take a very long time to run. When PopGrid has finished running, check the log file. The log file notes the start time, the files that PopGrid used, and the end time. Also, at the very end of the log file, PopGrid notes the number of people that PopGrid assigned to your grid definition (“Population covered by grid”) and the number of people that PopGrid determined are outside of your grid definition (“Population outside grid”).

```

6_18_2008_10_39_42_PM.txt - Notepad
File Edit Format View Help
Records processed : 5110000
Records processed : 5120000
Records processed : 5130000
Records processed : 5140000
Records processed : 5150000
Records processed : 5160000
Records processed : 5170000
Records processed : 5180000
Records processed : 5190000
Records processed : 5200000
Records processed : 5210000
Records processed : 5220000
Reading Census file CENSUS48.dat
Records processed : 5230000
Records processed : 5240000
Records processed : 5250000
Records processed : 5260000
Reading Census file CENSUS49.dat
Records processed : 5270000
Records processed : 5280000
MEMORYTABLE size : 13771108
Saving main table.
Finished @ 6/18/2008 10:39:42 PM
2000 CENSUS :
Population covered by grid : 13749883
Population outside grid : 265833554

```

J.1.3.2 PopGrid Output

PopGrid generates two files. One file has the number of people in each grid cell for each of the 304 race-ethnicity-gender-age demographic groups available in PopGrid. Table J-5 presents an example of what the population file looks like from PopGrid. The Row and Column uniquely identify each grid cell. Note that the Race, Ethnicity, Gender and AgeRange variables are precisely defined (see section on loading population data LoadData_Setups_Population).

Table J-5. Population File Fragment from PopGrid

Row	Column	Year	Population	Race	Ethnicity	Gender	AgeRange
58	81	2000	1.54	WHITE	HISPANIC	MALE	0T00
58	81	2000	0.03	BLACK	HISPANIC	MALE	0T00
58	81	2000	0.01	NATAMER	HISPANIC	MALE	0T00
58	81	2000	0.01	ASIAN	HISPANIC	MALE	0T00
58	81	2000	4.86	WHITE	HISPANIC	MALE	1T04
58	81	2000	0.12	BLACK	HISPANIC	MALE	1T04
58	81	2000	0.03	NATAMER	HISPANIC	MALE	1T04
58	81	2000	0.03	ASIAN	HISPANIC	MALE	1T04
58	81	2000	6.79	WHITE	HISPANIC	MALE	5T09
58	81	2000	0.21	BLACK	HISPANIC	MALE	5T09
58	81	2000	0.05	NATAMER	HISPANIC	MALE	5T09
58	81	2000	0.05	ASIAN	HISPANIC	MALE	5T09
58	81	2000	0.90	WHITE	HISPANIC	MALE	10T014
58	81	2000	0.04	BLACK	HISPANIC	MALE	10T014

Row	Column	Year	Population	Race	Ethnicity	Gender	AgeRange
58	81	2000	0.01	NATAMER	HISPANIC	MALE	10T014
58	81	2000	0.01	ASIAN	HISPANIC	MALE	10T014
58	81	2000	3.44	WHITE	HISPANIC	MALE	15T019
58	81	2000	0.15	BLACK	HISPANIC	MALE	15T019
58	81	2000	0.04	NATAMER	HISPANIC	MALE	15T019
58	81	2000	0.04	ASIAN	HISPANIC	MALE	15T019
58	81	2000	1.49	WHITE	HISPANIC	MALE	20T024
58	81	2000	0.06	BLACK	HISPANIC	MALE	20T024
58	81	2000	0.02	NATAMER	HISPANIC	MALE	20T024
58	81	2000	0.03	ASIAN	HISPANIC	MALE	20T024
58	81	2000	1.93	WHITE	HISPANIC	MALE	25T029
58	81	2000	0.04	BLACK	HISPANIC	MALE	25T029
58	81	2000	0.01	NATAMER	HISPANIC	MALE	25T029
58	81	2000	0.01	ASIAN	HISPANIC	MALE	25T029
58	81	2000	1.87	WHITE	HISPANIC	MALE	30T034
58	81	2000	0.08	BLACK	HISPANIC	MALE	30T034

PopGrid generates a second file that keeps track of the fraction of the total population in each of the eight race-ethnic groups that comes from each county in the United States. Table J-5 presents a sample. The SourceCol and SourceRow uniquely identify each county, and the TargetCol and TargetRow uniquely identify each grid cell. The Value variable gives the fraction of the total population in the grid cell for a given race-ethnic group that comes from the “source” county.

When a grid cell lies completely within a county, then the fraction will be 1. When a grid cell is in more than county, then the sum of the fractions across the counties for a given race-ethnic group must sum to one. In Table J-6, you can see that for grid cell (TargetCol=123, TargetRow=18) that the fraction of Asian Non-Hispanic coming from county (SourceCol=16, SourceRow=71) is 0.49 and for county (SourceCol=49, SourceRow=3) the fraction is 0.51. In this case, about half the population of Asian Non-Hispanics comes from each of the two counties. In the case of Black Hispanics, the fraction from county (SourceCol=16, SourceRow=71) is only 0.12, with most Black Hispanics in this grid cell coming from county (SourceCol=49, SourceRow=3).

Table J-6. Population-Weight File Fragment from PopGrid

SourceCol	SourceRow	TargetCol	TargetRow	Race	Ethnicity	Value	Year
16	71	123	18	ASIAN	NON-HISPANIC	0.49	2000
16	71	123	18	ASIAN	HISPANIC	0.21	2000
16	71	123	18	BLACK	NON-HISPANIC	0.49	2000
16	71	123	18	BLACK	HISPANIC	0.12	2000

SourceCol	SourceRow	TargetCol	TargetRow	Race	Ethnicity	Value	Year
16	71	123	18	NATAMER	NON-HISPANIC	0.98	2000
16	71	123	18	NATAMER	HISPANIC	0.43	2000
16	71	123	18	WHITE	NON-HISPANIC	0.23	2000
16	71	123	18	WHITE	HISPANIC	0.06	2000
49	3	123	18	ASIAN	NON-HISPANIC	0.51	2000
49	3	123	18	ASIAN	HISPANIC	0.79	2000
49	3	123	18	BLACK	NON-HISPANIC	0.51	2000
49	3	123	18	BLACK	HISPANIC	0.88	2000
49	3	123	18	NATAMER	NON-HISPANIC	0.02	2000
49	3	123	18	NATAMER	HISPANIC	0.57	2000
49	3	123	18	WHITE	NON-HISPANIC	0.77	2000
49	3	123	18	WHITE	HISPANIC	0.94	2000
6	23	45	1	ASIAN	NON-HISPANIC	0.00	2000
6	23	45	1	ASIAN	HISPANIC	0.00	2000
6	23	45	1	BLACK	NON-HISPANIC	0.00	2000
6	23	45	1	BLACK	HISPANIC	0.00	2000
6	23	45	1	NATAMER	NON-HISPANIC	1.00	2000
6	23	45	1	NATAMER	HISPANIC	1.00	2000
6	23	45	1	WHITE	NON-HISPANIC	1.00	2000
6	23	45	1	WHITE	HISPANIC	1.00	2000
6	23	45	2	ASIAN	NON-HISPANIC	1.00	2000
6	23	45	2	ASIAN	HISPANIC	1.00	2000
6	23	45	2	BLACK	NON-HISPANIC	1.00	2000
6	23	45	2	BLACK	HISPANIC	1.00	2000
6	23	45	2	NATAMER	NON-HISPANIC	1.00	2000
6	23	45	2	NATAMER	HISPANIC	1.00	2000
6	23	45	2	WHITE	NON-HISPANIC	1.00	2000
6	23	45	2	WHITE	HISPANIC	1.00	2000

J.2 Monitor Data in U.S. Setup

BenMAP-ready data files were created from 2000 through 2018, as reported to the U.S. Environmental Protection Agency’s (EPA) Air Quality System (AQS) for PM_{2.5} and ozone. Table J-7 summarizes the data sources and vintage of the processed data.

Table J-7. Underlying Data Sources for BenMAP Air Quality Data File

Pollutant	AQS Parameter Code	Year	Data Source
PM _{2.5}	88101	2000-2018	https://www.epa.gov/aqs
Ozone	44201	2000-2018	

J.2.1 Data Processing

IEc developed an R script to process the raw AQS data into formatted BenMAP-CE monitor files. The R script completes three major tasks:

1. Padding the raw data to generate a full, daily time series for each pollutant (daily 24 hour average values for PM_{2.5} and daily 8-hour maximum average values for ozone),
2. Converting the data from a long to a wide format, and
3. Adjusting and reformatting the data fields to match BenMAP-CE’s expected format for monitor datasets.

For daily data, BenMAP-CE expects the monitor data input file to contain a comma-separated string of 365 air quality values—one value for each day in the calendar year. Within this string, any missing values are represented by a period (“.”). In contrast, the AQS data only contain records for dates with measured air quality values or calculated pollutant metrics (24 hour average concentrations of PM_{2.5} or 8-hour maximum average concentrations for ozone). The raw AQS data are padded so that there is ultimately a row for every day in the year for each monitor, with periods inserted by the R code for every missing value. During this process, the R script also cleans up the raw data by removing any monitors located in Canada, removing any daily values with an Observation Percent less than 75%, and limiting values in the “Event Type” field to “None”.⁴³ In addition, PM_{2.5} data are limited to values with a “Sample Duration” of “24 HOUR” or “24-HR BLK AVG”.⁴⁴

⁴³ The Observation Percent field describes the volume of hourly data used to calculate each daily metric in the pre-generated AQS data files. Based on guidance provided by AQS staff, IEC limited values to those calculated with at least 75% of the underlying measurements. The “Event Type” field identifies days that may have been impacted by exceptional events, such as wildfires. Limiting the data to “Event Type” values of “None” limits the data included in BenMAP-CE to those days representing ambient air quality.

⁴⁴ For PM_{2.5} these values are taken from the “Arithmetic Mean” field of raw AQS data, while for O₃ they are taken from the “1st Max” field, based on guidance from AQS staff. The “1st Max” field is converted from ppm to ppb; BenMAP uses ppb as the unit for ozone concentrations.

Once the dataset has been expanded to the correct volume, the R script then casts it from a long format to a wide format. This means that the R script takes the 365 rows associated with a monitor and converts them to a single comma-separated string needed for the BenMAP-CE “Values” field.

- Data are reported in $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$, and ppb for ozone.

J.2.2 Output Files

Table H-8 lists the number of monitors by pollutant and year, represented in the resulting BenMAP-ready data files. The AQS Parameter Code for $\text{PM}_{2.5}$ is 88101 and for Ozone is 44201.

Table J-8. Number of Monitors by Pollutant and Year Included in BenMAP

Year	Number of Monitors by Year	
	$\text{PM}_{2.5}$	Ozone
2000	1,316	1,144
2001	1,355	1,190
2002	1,397	1,195
2003	1,421	1,213
2004	1,253	1,208
2005	1,285	1,198
2006	1,230	1,205
2007	1,191	1,212
2008	1,196	1,215
2009	1,273	1,214
2010	1,227	1,228
2011	1,137	1,283
2012	1,158	1,285
2013	1,190	1,293
2014	1,315	1,287
2015	1,373	1,276
2016	1,559	1,292
2017	1,463	1,287
2018	1,494	1,275

J.3 Demographic Datasets in U.S. Setup

The U.S. setup includes data on household size, educational attainment, poverty status, unemployment, health insurance coverage, occupational status, linguistic isolation, and

redlining. These datasets are included in the *EPA Standard Variables* dataset under *Variable Datasets*. We describe the data sources and processing methodology for each dataset below. County-level datasets were generated for 3,109 counties in the contiguous United States.⁴⁵ Census tract level datasets were generated for 72,538 census tracts in the contiguous United States.

J.3.1 Household Size

County-Level

To generate average household size for each county (termed “averagehhsz”), 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.⁴⁶ For these counties, we applied the state level average household size.

J.3.2 Educational Attainment

County-Level

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 and span two broad education categories: no high school diploma (termed “no_hs_degree”) and high school diploma 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.

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.

All estimates were generated at the county level for 3,109 counties in the contiguous United States. 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, 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).

Tract-Level

⁴⁵In 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>

⁴⁶ The two counties without data were Shannon County, South Dakota (FIPS Code 46113) and Bedford, Virginia (FIPS Code 51515).

We use data from the ACS to provide tract-level summaries of educational attainment. These data represent 5-year average ACS estimates from 2015 to 2019 and span the same two education categories as county-level demographic variables: no high school diploma (termed “no_hs_degree_tract”) and high school diploma (or equivalency) and above (termed “hs_degree_plus_tract”).

For both education groups (with/without HS diploma), we estimate the fraction of the total tract population (ages 25 years and above) in each education group. Thus, the two estimates sum to one for each tract.

All estimates were generated at the tract level for 72,538 tracts in the contiguous United States. For each estimate, we generate a coefficient of variation (CV) equal to the ratio of the standard error to the point estimate. For tracts with a CV greater than 0.3, we impute the tract-level estimate with a county-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). In cases of counties with a CV greater than 0.3, we further impute to the state-level.

J.3.3 Poverty Status

County-Level

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 representing the fraction of the county-level population below and above 200% of the poverty line (termed “below_2x_poverty_line” and “above_2x_poverty_line”).

We followed the same imputation procedures described in section J.3.2 to process poverty variables.

Tract-Level

To determine the poverty status at the tract level, we utilize ACS 5-year estimates from 2015 to 2019. The resulting datasets represent the fraction of the total population in the tract that falls below the federal poverty line (termed “below_poverty_line_tract”) and the fraction of the population that falls above the poverty line (termed “above_poverty_line_tract”). The EPA Standard Variables dataset also includes two variables representing the fraction of the tract-level population below and above 200% of the poverty line (termed “below_2x_poverty_line_tract” and “above_2x_poverty_line_tract”).

We followed the same imputation procedures described in section J.3.2 to process poverty variables.

J.3.4 Unemployment Rates

County-Level

BenMAP includes county-level employment variables representing average rates from 2017 to 2021 (termed “adj_employment_rate” and “adj_unemployment_rate”). Importantly, the employment variables are adjusted for use in BenMAP to use *total population* as the denominator rather than *labor force*. This allows users 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 (termed “not_in_labor_force_rate”) was calculated by subtracting the labor force from the total population and then dividing the remainder by the total population.

BenMAP also includes a previous version of the unemployment variable termed “unemployment_rate_2017_18”. In this dataset we use monthly county-level unemployment rates from the Bureau of Labor Statistics for the period from February 2017 to February 2018. The value calculated in BenMAP-CE represents the average employment rate across all months within this time period. The unemployment rate was calculated by dividing the number of unemployed workers in each county by the county’s total labor force. We note that the labor force does not include discouraged workers, people who have permanently left the labor force (e.g., retirees), and individuals who have not yet entered the labor force. Thus, the rates do **not** reflect the fraction of the population in BenMAP-CE that is not employed. We impute values for two counties using state unemployment rates, calculated by taking the total number of unemployed individuals within the state for each month, dividing by the total labor force within the state for the month, and averaging over all months.

J.3.5 Health Insurance

County-Level

We use 2016 data from the Small Area Health Insurance Estimates (SAHIE) collected by the U.S. Census Bureau to calculate the percentage of individuals without health insurance in each county. Total numbers of individuals with and without health insurance by county were downloaded for the year 2016. The SAHIE data provides the number of individuals with and without health insurance by county, disaggregated into three age groups: 18 to 65, 40 to 65, and under 65. Using these data, we calculated the percent of people uninsured by county for the age groups 1 to 17, 18-39, 40-64, and under 65 (termed “pct_uninsured1_17”, “pct_uninsured18_39”, “pct_uninsured40_64”, and “pct_uninsuredUnder_65”). As an example of this calculation, we subtracted the 18 to 65 uninsured population from the under 65 uninsured population to generate the 1

to 17 uninsured population. This value is then divided by the total 1 to 17 population (derived in the same manner) to estimate the uninsured rate.

There were two identical rows of data for the Washington, D.C. area (FIPS Code 11000 and 11001); we use FIPS code 11001.

We also use data from SAHIE collected by the U.S. Census Bureau from 2015 to 2019 to calculate the percentage of all individuals with and without health insurance in each county (termed “insured_rate” and “uninsured_rate”). The SAHIE data 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.3.6 Blue Collar Workers

County-Level

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-9.

Table J-9. 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 (termed “pct_BlueCollar”), with the blue collar and white collar worker fractions in each county adding up to 100%.

J.3.7 Linguistic Isolation

County-Level

We use 5-year ACS estimates from 2015-2019 to estimate linguistic isolation at the county level. The resulting datasets represent the fraction of the total population age 5 and over in the county that are linguistically isolated (defined by speaking English “less than very well”, termed “english_less_than_verywell”) and the fraction of the population age 5 and over that speak English “very well or better” (termed

“english_verywell_or_better”). This definition follows as closely as possible the definition of linguistic isolation used in EJ Screen and in other regulatory analyses.⁴⁷ Additionally, we produced an alternative definition in which the linguistically isolated population is more restrictive, i.e., those who speak English less than “well” (termed “english_less_than_well”), and inversely, those who speak English “well or better” (termed “english_well_or_better”).

Instead of imputing in cases of uncertainty, we generate county-level point estimates of linguistic isolation in an effort to prioritize geographic specificity in these estimates.

Tract-Level

We use 5-year ACS estimates from 2015-2019 to estimate linguistic isolation at the tract level. The resulting datasets represent the fraction of the total population age 5 and over in the tract that are linguistically isolated (defined by speaking English “less than very well”, termed “english_less_than_verywell_tract”) and the fraction of the population age 5 and over that speak English “very well or better” (termed “english_verywell_or_better_tract”). This definition follows as closely as possible the definition of linguistic isolation used in EJ Screen and in other regulatory analyses.⁴⁸ Additionally, we produced an alternative definition in which the linguistically isolated population is more restrictive, i.e., those who speak English less than “well” (termed “english_less_than_well_tract”), and inversely, those who speak English “well or better” (termed “english_well_or_better_tract”).

Instead of imputing in cases of uncertainty, we generate tract-level point estimates of linguistic isolation in an effort to prioritize geographic specificity in these estimates.

J.3.8 Redlining

Tract-Level

We use [graded census tracts](#) developed by Noelke et al., 2022 from digitized Home Owner’s Loan Corporation (HOLC) residential security maps overlaid onto 2010 Census tracts. Each census tract is classified as being covered by “Mainly A,” “Mainly B,” “Mainly C,” and “Mainly D” grading, corresponding to coverage of different hazard ratings from original HOLC maps. The dataset covers 14,818 census tracts, since HOLC maps only covered certain urban areas. This dataset was adapted to cover 72,538 census tracts for use in BenMAP, with the remaining census tracts categorized as “redlined_na” since they were not covered by HOLC grading. Census tracts labeled as “Mainly D” were categorized as “redlined” and census tracts that were mainly A-C were categorized as “not_redlined.”

⁴⁷ EJ screen defines linguistic isolation by households in which all members age 14 years and over speak a non-English language and also speak English less than “very well” (have difficulty with English).

⁴⁸ EJ screen defines linguistic isolation by households in which all members age 14 years and over speak a non-English language and also speak English less than “very well” (have difficulty with English).

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. Finally, the mechanics of the various Pooling Methods available in BenMAP are discussed in detail, including Subjective Weight based pooling, Fixed Effects pooling, Random / Fixed Effects pooling, and independent and dependent Sum and Subtraction.

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 endpoint group depends on how much the endpoint group 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:

$$(n - 1) \times \frac{100}{N} + \frac{100}{2N}$$

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 β . 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 endpoint 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.

K.2 Pooling

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

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

It is also possible to pool the study-specific estimates of incidence change derived from the health impact functions, instead of pooling the underlying β 's themselves. For a variety of reasons, this is often possible when it is not feasible to pool the underlying β 's. For example, if one study is log-linear and another is linear, we could not pool the β 's because they are not different estimates of a coefficient in the same health impact function, but are instead estimates of coefficients in different health impact functions. We can, however, calculate the incidence

change predicted by each health impact function (for a given change in pollutant concentration and, for the log-linear function, a given baseline incidence rate), and pool these incidence changes. BenMAP allows the pooling of incidence changes predicted by several studies for the same pollutant-health endpoint group combination. It also allows the pooling of the corresponding study-specific estimates of monetary benefits.

As with estimates based on only a single study, BenMAP allows you to characterize the uncertainty surrounding pooled estimates of incidence change and/or monetary benefit. To do this, BenMAP pools the study-specific distributions of incidence changes (or monetary benefit) to derive a pooled distribution. This pooled distribution incorporates information from all the studies used in the pooling procedure.

K.2.1 Weights Used for Pooling

The relative contribution of any one study in the pooling process depends on the weight assigned to that study. A key component of the pooling process, then, is the determination of the weight given to each study. There are various methods that can be used to assign weights to studies. Below we discuss the possible weighting schemes that are available in BenMAP.

K.2.1.1 Subjective Weights

BenMAP allows you the option of specifying the weights to be used. Suppose, for example, you want to simply average all study-specific results. You would then assign a weight of $1/N$ to each of the N study-specific distributions that are to be pooled. Note that subjective weights are limited to two decimal places, and are normalized to sum to one, if they do not already sum to one.

K.2.1.2 Automatically Generated Weights

A simple average has the advantage of simplicity but the disadvantage of not taking into account the uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty. A common method for weighting estimates involves using their variances. Variance takes into account both the consistency of data and the sample size used to obtain the estimate, two key factors that influence the reliability of results. BenMAP has two methods of automatically generating pooling weights using the variances of the input distributions - Fixed Effects Pooling and Random / Fixed Effects Pooling.

The discussion of these two weighting schemes is first presented in terms of pooling the pollutant coefficients (the β 's), because that most closely matches the discussion of the method for pooling study results as it was originally presented by DerSimonian and Laird. We then give an overview of the analogous weighting process used within BenMAP to generate weights for incidence changes rather than β 's.

K.2.1.3 Fixed-Effect Weights

The fixed effects model assumes that there is a single true concentration-response relationship and therefore a single true value for the parameter β that applies everywhere. Differences

among β 's reported by different studies are therefore simply the result of sampling error. That is, each reported β is an estimate of the same underlying parameter. The certainty of an estimate is reflected in its variance (the larger the variance, the less certain the estimate). Fixed effects pooling therefore weights each estimate under consideration in proportion to the inverse of its variance.

Suppose there are n studies, with the i th study providing an estimate β_i with variance v_i ($i = 1, \dots, n$). Let

$$S = \sum \frac{1}{v_i},$$

denote the sum of the inverse variances. Then the weight, w_i , given to the i th estimate, β_i , is:

$$w_i = \frac{1/v_i}{S}.$$

This means that estimates with small variances (i.e., estimates with relatively little uncertainty surrounding them) receive large weights, and those with large variances receive small weights. The estimate produced by pooling based on a fixed effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above.

That is:

$$\beta_{fe} = \sum w_i \times \beta_i$$

The variance associated with this pooled estimate is the inverse of the sum of the inverse variances:

$$v_{fe} = \frac{1}{\sum 1/v_i}$$

Table K-1 shows the relevant calculations for this pooling for three sample studies.

Table K-1. Example of Fixed Effects Model Calculations

Study	B_i	V_i	$1/v_i$	W_i	$W_i * \beta_i$
1	0.75	0.1225	8.16	0.016	0.012
2	1.25	0.0025	400	0.787	0.984
3	1.00	0.0100	100	0.197	0.197
Sum			?=508.16	?=1.000	?=1.193

The sum of weighted contributions in the last column is the pooled estimate of β based on the fixed effects model. This estimate (1.193) is considerably closer to the estimate from study 2 (1.25) than is the estimate (1.0) that simply averages the study estimates. This reflects the fact that the estimate from study 2 has a much smaller variance than the estimates from the other two studies and is therefore more heavily weighted in the pooling.

The variance of the pooled estimate, v_{fe} , is the inverse of the sum of the variances, or 0.00197. (The sums of the β_i and v_i are not shown, since they are of no importance. The sum of the $1/v_i$ is S , used to calculate the weights. The sum of the weights, w_i , $i=1, \dots, n$, is 1.0, as expected.)

K.2.1.4 Random- / Fixed-Effect Weights

An alternative to the fixed effects model is the random effects model, which allows the possibility that the estimates β_i from the different studies may in fact be estimates of different parameters, rather than just different estimates of a single underlying parameter. In studies of the effects of PM_{10} on mortality, for example, if the composition of PM_{10} varies among study locations the underlying relationship between mortality and PM_{10} may be different from one study location to another. For example, fine particles make up a greater fraction of PM_{10} in Philadelphia than in El Paso. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β in Philadelphia to be greater than the true value of β in El Paso. This would violate the assumption of the fixed effects model.

The following procedure can test whether it is appropriate to base the pooling on the random effects model (vs. the fixed effects model):

A test statistic, Q_w , the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model, is calculated as:

$$Q_w = \sum_i \frac{1}{v_i} (\beta_{fe} - \beta_i)^2$$

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

The weights used in a pooling based on the random effects model must take into account not only the within-study variances (used in a meta-analysis based on the fixed effects model) but the between-study variance as well. These weights are calculated as follows:

Using Q_w , the between-study variance, η^2 , is:

$$\eta^2 = \frac{Q_w - (n-1)}{\sum 1/v_i - \frac{\sum 1/v_i^2}{\sum 1/v_i}}$$

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

Given a value for η^2 , the random effects estimate is calculated in almost the same way as the fixed effects estimate. However, the weights now incorporate both the within-study variance (v_i) and the between-study variance (η^2). Whereas the weights implied by the fixed effects model used only v_i , the within-study variance, the weights implied by the random effects model use $v_i + \eta^2$.

Let $v_i^* = v_i + \eta^2$. Then:

$$S^* = \sum \frac{1}{v_i^*},$$

$$w_i^* = \frac{1/v_i^*}{S^*}$$

The estimate produced by pooling based on the random effects model, then, is just a weighted average of the estimates from the studies being considered, with the weights as defined above. That is:

$$\beta_{rand} = \sum w_i^* \times \beta_i$$

The variance associated with this random effects pooled estimate is, as it was for the fixed effects pooled estimate, the inverse of the sum of the inverse variances:

$$v_{rand} = \frac{1}{\sum 1/v_i^*}$$

The weighting scheme used in a pooling based on the random effects model is basically the same as that used if a fixed effects model is assumed, but the variances used in the calculations are different. This is because a fixed effects model assumes that the variability among the estimates from different studies is due only to sampling error (i.e.,

each study is thought of as representing just another sample from the same underlying population), while the random effects model assumes that there is not only sampling error associated with each study, but that there is also between-study variability -- each study is estimating a different underlying β . Therefore, the sum of the within-study variance and the between-study variance yields an overall variance estimate.

Fixed Effects and Random / Fixed Effects Weighting to Pool Incidence Change Distributions and Dollar Benefit Distributions

Weights can be derived for pooling incidence changes predicted by different studies, using either the fixed effects or the fixed / random effects model, in a way that is analogous to the derivation of weights for pooling the β 's in the C-R functions. As described above, BenMAP generates a Latin Hypercube representation of the distribution of incidence change corresponding to each health impact function selected. The means of those study-specific Latin Hypercube distributions of incidence change are used in exactly the same way as the reported β 's are used in the calculation of fixed effects and random effects weights described above. The variances of incidence change are used in the same way as the variances of the β 's. The formulas above for calculating fixed effects weights, for testing the fixed effects hypothesis, and for calculating random effects weights can all be used by substituting the mean incidence change for the i th health impact function for β_i and the variance of incidence change for the i th health impact function for v_i .

Similarly, weights can be derived for dollar benefit distributions. As described above, BenMAP generates a Latin Hypercube representation of the distribution of dollar benefits. The means of those Latin Hypercube distributions are used in exactly the same way as the reported β 's are used in the calculation of fixed effects and random effects weights described above. The variances of dollar benefits are used in the same way as the variances of the β 's. The formulas above for calculating fixed effects weights, for testing the fixed effects hypothesis, and for calculating random effects weights can all be used by substituting the mean dollar benefit change for the i th valuation for β_i and the variance of dollar benefits for the i th valuation for v_i .

BenMAP always derives Fixed Effects and Random / Fixed Effects weights using nationally aggregated results, and uses those weights for pooling at each grid cell (or county, etc. if you choose to aggregate results prior to pooling). This is done because BenMAP does not include any regionally based uncertainty - that is, all uncertainty is at the national level in BenMAP, and all regional differences (population, for example) are treated as certain.

K.2.2 Mechanics of Pooling in BenMAP

Once weights are generated for each input distribution, BenMAP has three options for using these weights to combine the input distributions into a single new distribution. These options are referred to as Advanced Pooling Methods.

Round Weights to Two Digits

This is BenMAP's default Advanced Pooling Method, and is always the method used when Subjective Weights are used. The first step is converting the weights to two digit integers by multiplying them by 100 and rounding to the nearest integer. If all the integral weights thus generated are divisible by the smallest weight, they are each divided by that smallest weight. For example, if the original weights were 0.1, 0.2, 0.3, and 0.4, the resulting integral weights would be 10/10, 20/10, 30/10, and 40/10 (or 1, 2, 3, and 4).

BenMAP then creates a new distribution by sampling each entire input distribution according to its weight. That is, in the above example the first distribution would be sampled once, the second distribution twice, and so forth. The advantage of sampling whole distributions is that it preserves the characteristics (i.e., the moments - the mean, the variance, etc.) of the underlying distributions. Assuming n latin hypercube points, the resulting distribution will contain a maximum of $100 * n$ values, which are then sorted low to high and binned down to n values, which will represent the new, pooled distribution.

Round Weights to Three Digits

This Advanced Pooling Method is essentially the same as rounding weights to two digits, except that the weights are converted to three digit integers, and so forth. That is, the weights are multiplied by 1000 and rounded to the nearest integer. Again, if all the integral weights thus generated are divisible by the smallest weight, they are each divided by that smallest weight. Assuming n Latin Hypercube points, the resulting distribution with this Advanced Pooling Method can contain a maximum of $1000 * n$ values, which are sorted low to high and binned down to n values, which represent the new, pooled distribution.

Exact Weights for Monte Carlo

This Advanced Pooling Method uses a Monte Carlo method to combine the input distributions. Using this method, on each of many iterations, (1) an input distribution is selected (with the probability of selection equal to the weight assigned to the distribution), and (2) a value is randomly drawn from that distribution. Values chosen in this way are placed into a temporary pooled distribution, which will have one point per iteration of the Monte Carlo method. The number of iterations is specified by the user, and defaults to 5,000. After the temporary distribution is fully generated, it is sorted low to high and binned down to n values (where n is the number of Latin Hypercube Points chosen for the analysis).

K.2.3 Summing Distributions

Sometimes rather than pooling distributions we want to add them. For example, some studies have estimated a health impact function for hospital admissions for COPD and another health impact function for hospital admissions for pneumonia. From each of these health impact functions, BenMAP can derive the corresponding distributions for

incidence change. Hospital admissions for COPD and pneumonia are two of the most important components of respiratory hospital admissions, and we may want to estimate the number of cases of “respiratory hospital admissions,” as characterized by being either COPD or pneumonia. To do this we would add the two distributions.

Summing across distributions can be done in one of two ways: We can assume the two distributions are independent of each other or dependent. Which is the more reasonable assumption depends on the particulars of the distributions being summed.

Assuming Independence

This is the Sum (Independent) Pooling Method. To sum two distributions that are independent, on each of many iterations of a Monte Carlo procedure, BenMAP (1) randomly selects a value from the first input distribution, (2) randomly selects a value from the second input distribution, and (3) adds the two values together. To sum N distributions that are independent, BenMAP follows an analogous procedure in which, on each iteration it makes a random selection from each of the input distributions and then adds the results together. When the Monte Carlo procedure is completed, all such generated results are sorted low to high and binned down to the appropriate number of latin hypercube points. The number of iterations is determined by the Monte Carlo Iterations setting.

Assuming Dependence

This is the Sum (Dependent) Pooling Method. Recall that the uncertainty distributions in BenMAP are latin hypercube representations, consisting of N percentile points. To sum two distributions assumed to be dependent, BenMAP simply generates a new N point latin hypercube where each point is the sum of the corresponding points from the input latin hypercubes. That is, the first point in the new latin hypercube is the sum of the first points in the two input latin hypercubes, and so forth. To sum n distributions that are assumed to be dependent, BenMAP follows an analogous procedure in which each point in the new latin hypercube is the sum of the corresponding points from each of the input latin hypercubes.

K.2.4 Subtracting Distributions

In some cases, you may want to subtract one or more distribution(s) from another. For example, one study may have estimated a health impact function for minor restricted activity days (MRADs), and another study may have estimated a health impact function for asthma “episodes.” You may want to subtract the change in incidence of asthma episodes from the change in incidence from MRADs before estimating the monetary value of the MRADs, so that the monetary value of asthma episodes avoided will not be included.

Subtracting across distributions can be done in one of two ways: we can assume the two distributions are independent of each other or dependent. Which is the more

reasonable assumption depends on the particulars of the distributions being subtracted.

Assuming Independence

This is the Subtraction (Independent) Pooling Method. To subtract one distribution from another, assuming independence, on each of many iterations of a Monte Carlo procedure, BenMAP (1) randomly selects a value from the first input distribution, (2) randomly selects a value from the second input distribution, and (3) subtracts the second value from the first. To subtract N distributions from another distribution, assuming independence, BenMAP follows an analogous procedure in which, on each iteration it makes a random selection from each of the input distributions and then subtracts the second through the Nth from the first. When the Monte Carlo procedure is completed, all such generated results are sorted low to high and binned down to the appropriate number of Latin Hypercube points. The number of iterations is determined by the Monte Carlo Iterations setting.

Assuming Dependence

This is the Subtraction (Dependent) Pooling Method (see Chapter 6 for details). Recall that the uncertainty distributions in BenMAP are Latin Hypercube representations, consisting of N percentile points. To subtract one distribution from another, assuming them to be dependent, BenMAP simply generates a new N point Latin Hypercube where each point is the result of subtracting the corresponding point of the second input Latin Hypercube from the corresponding point of the first input Latin Hypercube. That is, the first point in the new Latin Hypercube is the result of subtracting the first point in the second Latin Hypercube from the first point of the first Latin Hypercube, and so forth. To subtract n distributions from another distribution, assuming dependence, BenMAP follows an analogous procedure in which each point in the new Latin Hypercube is the result of subtracting the corresponding points of the second through the Nth input Latin Hypercubes from the corresponding point of the first.

Appendix L. Command Line BenMAP

The command line version of BenMAP is capable of performing all of the functions of the GUI-based version. It is most useful for large, complex analyses that require generation of a substantial number of files. This appendix describes the syntax and use of the command line version.

L.1 Overview

The overall format of the file is a variable definitions section followed by a commands section. Comment statements are supported at any point in the file. Lines beginning with a pound character (#) are considered comment lines and will be ignored during file parsing.

Additionally, LOAD <filename> statements are supported at any point in the file. These work as string replacements - the contents of the file specified by <filename> are simply inserted into the main file. Multi-level LOAD statements are supported, but no attempt is made to detect cycles (two files referencing each other with LOAD statements, for example).

The control file is, in general, not case sensitive. In the case of user-defined strings, (variable values, etc.), it is preserved.

L.2 Variables

The variable definitions section is optional, and if present will consist of a single line with the word “Variables” on it, followed by one or more lines that define variables. A variable definition consists of a variable name and a variable value. When parsing lines in the commands section of the control file, all occurrences of the variable name will be replaced by the variable value.

All variable names must begin and end with the percent character (%).

Variable Name/Value replacement will be done in multiple passes (until no variable names remain), so variable values may contain other variable names. No attempt will be made to detect cycles, however, so be careful not to introduce them. For example, avoid variable definitions like the following:

```
%BENMAPDIR%           %AQGDIR%\
%AQGDIR%               %BENMAPDIR%\Air Quality Grids
```

Variable values must be contained in a single line, and will consist of the first non-whitespace character after the variable name through the newline character. Watch out for undesired trailing whitespaces!

L.3 Commands

The commands section is required, and will consist of one or more command sections. There are five types of command sections:

SETACTIVESETUP

CREATE AQG

RUN CFG

RUN APV

GENERATE REPORT

This section will discuss each one in turn.

In general, in command sections, there must be at least one white space between each token (where a token is either a command, a parameter name, or a parameter value).

L.3.1 Set Active Setup

For the US version of the BenMAP command line executable the only valid value is United States. The SETACTIVESETUP section is required.

Example

```
-ActiveSetup United States
```

L.3.2 Create AQG

This section initiates the creation of one or more air quality grids (normally one, potentially two in the case of monitor rollback grid creation - see below). It always starts with the words CREATE AQG. It must then include the following options, in any order:

```
-Filename <filename>
```

```
-Gridtype <gridtype>
```

```
-Pollutant <pollutant>
```

The **Filename** value is the name of the air quality grid that will be created.

The **GridType** value must be one found in the BenMAP database. The actual values for this parameter are found on the Modify Setup screen in the Grid Definitions list box.

BenMAP supports pollutant values present in the active setup. By default, these include Ozone and PM2.5 (no subscripts needed) for the United States setup. BenMAP Command Line will support additional pollutants as well, so long as they are present in

the active setup selected at the start of the Command Line Script. These values can be found in the Modify Setup screen in the Pollutants list box.

After these required options, the type of grid creation must be identified, and then the parameters for that grid creation type must be specified. There are four air quality grid creation types:

- ModelDirect
- MonitorDirect
- MonitorModelRelative
- MonitorRollback

L.3.2.1 Model Direct

This section initiates the creation of a model direct air quality grid.

This creation type has two required parameters:

- ModelFilename <filename>
- DSNName <ODBC DSN name>

and one optional parameter:

- TableName <tablename>

Supported DSNName values are:

- | | |
|--------------------|------------------------------|
| Excel Files | Excel Spreadsheet (.xls) |
| Text Files | Comma-delimited (.csv) files |
| MS Access Database | Access Database (.mdb) |

If the DSNName is “Excel Files” and there is more than one worksheet in the workbook or “MS Access Database” and there is more than one table in the database then the TableName parameter must indicate the worksheet or table name.

L.3.2.2 Monitor Direct

This section initiates the creation of a monitor direct air quality grid.

The required parameters are:

- MonitorDataType <DataSource descriptor>
- InterpolationMethod <Interpolation Method>

Valid values for MonitorDataType are:

- Library
- DatabaseRows
- DatabaseColumns
- TextFile

Valid values for Interpolation method are:

- ClosestMonitor
- V N A

If MonitorDataType is Library then the following parameters are required:

- MonitorDataSet <Monitor Dataset Name>

MonitorDataSet is the Dataset name of Monitor data stored in the BenMAP database. These values can be found on the Modify Setup screen in Monitor Datasets list box.

- MonitorYear <Year>

MonitorYear specifies the year of interest in the monitor library.

If MonitorDataType is DatabaseRows then the following parameters are required:

- MonitorFile <filename>
- DSNName <ODBC DSN name>

and one optional parameter:

- TableName <tablename>

Supported DSNName values are:

- Excel Files Excel Spreadsheet (.xls)
- Text Files Comma-delimited (.csv) files
- MS Access Database Access Database (.mdb)

If the DSNName is “Excel Files” and there is more than one worksheet in the workbook or “MS Access Database” and there is more than one table in the database then the TableName parameter must indicate the worksheet or table name.

If MonitorDataType is DatabaseColumns then the same parameters for MonitorDataType DatabaseRows are required along with the following:

- MonitorDefFilename
- DefDSNName
- DefTableName

These parameters behave the same as the corresponding DatabaseRows parameters.

If MonitorDataType is TextFile the following parameter is required:

- MonitorFile <filename>

MonitorFile specifies a comma separated values (*.csv, generally) file containing monitor data.

Optional Parameters:

- MaxDistance <real>

Specifies the maximum distance (in kilometers) to be used in ClosestMonitor interpolation or VNA interpolation. Monitors outside this distance will not be considered in the interpolation procedure.

- MaxRelativeDistance <real>

Specifies the maximum relative distance to be used in VNA interpolation, where relative distance is the multiple of the distance to the closest monitor used in the interpolation procedure.

- WeightingMethod <method>

Specifies the weighting procedure used for monitors in VNA interpolation. Supported values are InverseDistance and InverseDistanceSquared. If this parameter is not specified, InverseDistance weighting is used.

L.3.2.3 Monitor Rollback

[NOTE: Monitor Rollback is currently disabled in BenMAP Command Line. We are aware of this issue and working to include the functionality in upcoming BenMAP releases.]

```
// MonitorRollback
```

```
BaselineFilename      = -BaselineFilename;

// RollbackOptions

Percentage            = -Percentage;

Increment             = -Increment;

// RollbackToStandardOptions

Standard              = -Standard;

Metric                = -Metric;

Ordinality            = -Ordinality;

InterdayRollbackMethod = -InterdayRollbackMethod;

IntradayRollbackMethod = -IntradayRollbackMethod;
```

L.3.3 Run CFG

The command line version of BenMAP does not support creation of new .cfgx files, both because this would be quite cumbersome to do in plain text, and because it probably is not needed. Slight modifications of existing .cfgx files are supported, and it is thought that at this point this should be enough.

As such, the only required parameter to run a configuration is the configuration filename. Optional parameters allow the slight modifications mentioned above.

Required Parameters

-CFGFilename <filename>

Specifies the .cfgx file to run.

-ResultsFilename <filename>

Specifies the .cfgrx file to save the results in.

Optional Parameters

-BaselineAQG <filename>

Specifies the baseline air quality grid file to use when running the configuration - overrides whatever value is present in the .cfgx file.

-ControlAQG <filename>

Specifies the control air quality grid file to use when running the configuration - overrides whatever value is present in the .cfgx file.

-Year <Integer>

Year in which to run the configuration (this will affect the population numbers used) - overrides whatever value is present in the .cfgx file. Supported values are 1990 and up.

-LatinHypercubePoints <integer>

Number of latin hypercube points to generate when running the configuration (zero means run in point mode), overrides whatever value is present in the .cfgx file.

-Threshold <real>

Threshold to use when running the configuration - overrides whatever value is present in the .cfgx file.

L.3.4 Run APV

The command line version of BenMAP does not support creation of new .apvx files, both because this would be quite cumbersome to do in plain text, and because it probably is not needed. Slight modifications of existing .apvx files are supported, and it is thought that at this point this should be enough.

As such, the only required parameter to run an APV configuration is the APV configuration filename. Optional parameters allow the slight modifications mentioned above.

Required Parameters

-APVFilename <filename>

Specifies the .apvx file to run.

-ResultsFilename <filename>

Specifies the .apvrx file to save the results in.

Optional Parameters

-CFGRFilename <filename>

Specifies the .cfg file to use when running the APV configuration - note that this file must contain the same set of results which the .cfg file originally used to generate the .apv file contained. Overrides whatever value is present in the .apv file.

-IncidenceAggregation <aggregation level>

Level to aggregate incidence results to before pooling them. Supported values are None, County, State, and Nation. Overrides whatever value is present in the .apv file.

-ValuationAggregation <aggregation level>

Level to aggregate valuation results to before pooling them. Supported values are None, County, State, and Nation (though the value must be greater than or equal to IncidenceAggregation). Overrides whatever value is present in the .apv file.

-RandomSeed <integer>

Random seed to use for all procedures requiring pseudo-random numbers (e.g. monte carlo procedures). Overrides the default behavior, which is to generate a new random seed each time the APV configuration is run.

-DollarYear <integer>

L.3.5 Generate Report

Reports come in three main varieties - Audit Trail Reports, which can be generated from any BenMAP file; Configuration Results Reports, which can be generated from .cfg files; and APV Configuration Results Reports, which can be generated from .apv files. All these report types need an input filename and an output filename. CFGR reports and APVR reports additionally take many optional parameters.

The format for each report type is:

GENERATE REPORT <ReportType>

-InputFile <filename>

-ReportFile <filename>

<optional parameters>

Supported ReportType values are: AuditTrail, CFGR, and APVR.

L.3.5.1 Audit Trail

Audit trail reports require only the parameters described in the “Generate Report” section.

L.3.5.2 CFGR Report

A CFGR report may be generating using only the parameters described in the “Generate Report” section. However, there are also a number of additional options, described below.

Optional Parameters

-GridFields <comma separated field names>

Specifies the set of grid fields to include in the report. Grid fields include Column and Row. If this parameter is not present, all fields will be included in the report.

-CustomFields <comma separated field names>

Specifies the set of custom fields (C-R Function identifiers, in this case) to include in the report. Custom fields include DataSet, Endpoint Group, Endpoint, Pollutant, Metric, Seasonal Metric, Metric Statistic, Author, Year, Location, Other Pollutants, Qualifier, Reference, Race, Ethnicity, Gender, Start Age, End Age, Function, Incidence Dataset, Prevalence Dataset, Beta, Disbeta, P1Beta, P2Beta, A, NameA, B, NameB, C, NameC, and Geographic Area. If this parameter is not present, all fields will be included in the report.

-ResultFields <comma separated field names>

Specifies the set of result fields to include in the report. Result fields include Point Estimate, Population, Delta, Mean, Baseline, Percent of Baseline, Standard Deviation, Variance, and All Percentiles. If this parameter is not present, all fields will be included in the report. If All Percentiles is not chosen, BenMAP will only export the 2.5th and 97.5th percentiles.

L.3.5.3 APVR Report

Required Parameters

APVR Reports require one additional parameter beyond those required for Audit Trail or CFGR Reports.

-ResultType <result type>

Specifies the result type for which a report should be created. Supported result types are: IncidenceResults, PooledIncidence, PooledValuation.

Optional Parameters

All of the CFGR report parameters are supported for APVR reports as well, except that Population and Delta are not supported ResultField elements.

-Totals <total type>

Specifies the type of totals which should be included in the report. Supported types are Dependent and Independent. Totals can only be generated for valuation results (Valuation, AggregatedValuation, and PooledValuation result types).

-ResultFields <comma separated field names>

Specifies the set of result fields to include in the report. For IncidenceResults reports, all fields will be included by default. For PooledIncidence reports, supported fields are: Point Estimate, Population, Delta, Mean, Baseline, Percent of Baseline, Standard Deviation, Variance, and All Percentiles. For PooledValuation reports, supported fields are: Point Estimate, Mean, Standard Deviation, Variance, Percentiles. If All Percentiles is not chosen, BenMAP will only export the 2.5th and 97.5th percentiles.

L.4 Example 1

VARIABLES

%CFG%	C:\BenMAP\CommandLine\Configurations\PM25 Wizard.cfgx
%APV%	C:\BenMAP\CommandLine\Configurations\PM25 Wizard.apvx
%RESULTSDIR%	C:\BenMAP\Temp
%REPORTDIR%	C:\BenMAP\Temp
%AQG%	C:\BenMAP\CommandLine\Air Quality Grids

COMMANDS

SETACTIVESETUP

-ActiveSetup United States

CREATE AQG

-Filename	%AQG%\PM25_2002Baseline_50km.aqgx
-GridType	CMAQ 12km
-Pollutant	PM2.5

MonitorDirect

-InterpolationMethod	VNA
-MonitorDataType	Library
-MonitorDataSet	EPA Standard Monitors

-MonitorYear 2002
-MaxDistance 50

CREATE AQG

-Filename %AQG%\PM25_2002Control_50km.aqgx
-GridType CMAQ 12km
-Pollutant PM2.5

MonitorRollback

-InterpolationMethod VNA
-MonitorDataType Library
-MonitorDataSet EPA Standard Monitors
-MonitorYear 2002
-RollbackGridType State
-MaxDistance 50

RollbackToStandardOptions

-Stanard 65
-Metric D24HourMean
-InterdayRollbackMethodIncremental

RUN CFG

-CFGFilename %CFG%
-ResultsFilename %RESULTSDIR%\PM25_2002_50km.aqgx
-BaselineAQG %AQG%\PM25_2002Baseline_50km.aqgx
-ControlAQG %AQG%\PM25_2002Control_50km.aqgx

RUN APV

-APVFilename %APV%
-ResultsFilename %RESULTSDIR%\PM25_2002_50km.apvrx
-CFGRFilename %RESULTSDIR%\PM25_2002_50km.cfgrx
-IncidenceAggregation Nation
-ValuationAggregation Nation

GENERATE REPORT APVR

-InputFile %RESULTSDIR%\PM25_2002_50km.apvrx
-ReportFile %REPORTDIR%\PM25_2002_50km_IncidenceNation.csv
-ResultType PooledIncidence
-CustomFields Endpoint Group,Author,Start Age,Endpoint,Qualifier,
Pooling Window

-ResultFields Mean,Standard Deviation,Latin Hypercube Points
 -DecimalDigits 0

GENERATE REPORT APVR

-InputFile %RESULTSDIR%\PM25_2002_50km.apvrx
 -ReportFile %REPORTDIR%\PM25_2002_50km_ValuationNation.csv
 -ResultType PooledValuation
 -CustomFields Endpoint Group, Author,Start Age, Endpoint, Qualifier,
 Pooling Window
 -ResultFields Mean,Standard Deviation,Latin Hypercube Points
 -DecimalDigits 0

L.5 Example 2

VARIABLES

%CFG% C:\BenMAP\CommandLine\Configurations\PM25
 Wizard.cfgx
 %APV% C:\BenMAP\CommandLine\Configurations\PM25
 Wizard.apvx
 %RESULTSDIR% C:\BenMAP\Temp
 %REPORTDIR% C:\BenMAP\Temp
 %AQG% C:\BenMAP\CommandLine\Air Quality Grids

COMMANDS

SETACTIVESETUP

-ActiveSetup United States

CREATE AQG

-Filename %AQG%\PM25_2004Baseline.aqgx
 -GridType County
 -Pollutant PM2.5

MonitorDirect

-InterpolationMethod VNA
 -MonitorData Type Library
 -MonitorDataSet EPA Standard Monitors
 -MonitorYear 2004

CREATE AQG

-Filename %AQG%\PM25_2004_Control.aqgx
 -GridType County
 -Pollutant PM2.5

MonitorRollback

-InterpolationMethod VNA
 -MonitorDataType Library
 -MonitorDataSet EPA Standard Monitors
 -MonitorYear 2004
 -RollbackGridType State
 -MaxDistance 50

RollbackToStandardOptions

-Standard 35
 -Metric D24HourMean
 -InterdayRollbackMethod Incremental

RUN CFG

-CFGFilename %CFG%
 -ResultsFilename %RESULTSDIR%\PM25_2004.cfgrx
 -BaselineAQG %AQG%\PM25_2004Baseline.aqgx
 -ControlAQG %AQG%\PM25_2004Control.aqgx

RUN APV

-APVFilename %APV%
 -ResultsFilename %RESULTSDIR%\PM25_2004.apvrx
 -CFGRFilename %RESULTSDIR%\PM25_2004.cfgrx
 -IncidenceAggregaton Nation
 -IncidenceAggregation Nation

GENERATE REPORT APVR

-InputFile %RESULTSDIR%\PM25_2004.apvrx
 -ReportFile %REPORTDIR%\pm25_2004_IncidenceNation.csv
 -ResultType PooledIncidence
 -CustomFields Endpoint Group, Author,Start Age, Endpoint,Qualifier,
 Pooling Window
 -ResultFields Mean,Standard Deviation, Latin Hypercube Points
 -DecimalDigits 0

GENERATE REPORT APVR

-InputFile	%RESULTSDIR%\PM25_2004.apvrx
-ReportFile	%REPORTDIR%\PM25_2004_ValuationNation.csv
-ResultType	PooledValuation
-CustomFields	Endpoint Group, Author, Start Age, Endpoint, Qualifier, Pooling Window
-ResultFields	Mean,Standard Deviation, Latin Hypercube Points
-DecimalDigits	0

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.

M.1 User Defined Variables

In addition to pre-defined variables that you can select from the Available Variables list, you can create your own variables in the C-R Function Editor.

A variable is an identifier whose value can change at runtime. Put differently, a variable is a name for a location in memory; you can use the name to read or write to the memory location. Variables are like containers for data, and, because they are typed, they tell the compiler how to interpret the data they hold.

The basic syntax for a variable declaration is

```
var identifierList: type;
```

where identifierList is a comma-delimited list of valid identifiers and type is any valid type. For example,

```
var I: Integer;
```

declares a variable I of type Integer, while

```
var X, Y: Real;
```

declares two variables--X and Y--of type Real.

Consecutive variable declarations do not have to repeat the reserved word var:

```
var
```

```
X, Y, Z: Double;
```

```
I, J, K: Integer;
```

```
Digit: 0..9;
```

```
IndicatorName: String;
```

```
Okay: Boolean;
```

Variables can be initialized at the same time they are declared, using the syntax

```
var identifier: type = constantExpression;
```

where `constantExpression` is any constant expression representing a value of type `type`. Thus the declaration

```
var I: Integer = 7;
```

is equivalent to the declaration and statement

```
var I: Integer;
```

```
...
```

```
I := 7;
```

Multiple variable declarations (such as `var X, Y, Z: Real;`) cannot include initializations, nor can declarations of variant and file-type variables.

M.2 The Script Language

In the C-R Function Editor, you can evaluate complex block of statements.

You can use constructions like:

```
If...then...else;
```

```
for I:= ... to .. do ;
```

```
while... do ;
```

```
repeat .... until...;
```

```
break;
```

```
assignment (...:=...;)
```

```
try...finally...end; try...except...end;
```

Each function you create can be a single statement or a block of statements.

When you specify it as a block of statements, your script must conform to the rules of the script language, as follows:

1. Each single statement must end with a semicolon (;)
2. You can use the following statements:

```
variable := expression;
```

```
If logical expression then statement(s) [else statement(s)];
```

for variable := from_expression **to/downto** to_expression **do** statement(s);

while logical_expression **do** statement(s);

repeat statement(s) **until** logical_expression;

try statement(s) **finally** statement(s) **end; try**

statement(s) **except** statement(s) **end;**

inline comments: // comment... until the end of the line

nested comments: { nested comment }

Statement(s) in the above declarations states that you can specify either a single statement or a block of statements. The block of statements must be enclosed in **begin** ... **end** keywords. It is not necessary to enclose the body of the function in **begin .. end**. Cycle statements can use break keyword to break the cycle (break must also end with semicolon.)

M.3 Operands

Expressions may contain the following constant and variable types:

Integer numbers;

Floating point numbers;

Scientific numbers;

Decimal separator for all floating point and scientific-format numbers in expressions, is independent of the Regional Settings of Windows and always is a decimal point ('.').

Boolean values - TRUE or FALSE;

Date type values - values of that type must be put in quotes (' '), and also date separator character is independent of the Regional Settings of Windows and always is a slash - /, i.e. - '01/01/2005'

String values - values of that type must be put in double quotes (" "); If a string contains double quotes, you should double them (i.e., "this is a ""string""");

M.4 Operations

Arithmetical

+, -, ×, /;

div - integer division;

mod - modulo;

^ - power of;

-- negate;

Logical

<, <=, >=, >, <>, =;

and, or, xor, not;

Bitwise

and, or, xor;

~ - negate;

M.5 Arithmetic Functions

ABS(X)	Absolute value
SQR(X)	Square = $X^2 = X \times X$
SQRT(X)	Square root
SIGN(X)	Sign of X; =1 for $X > 0$, =0 for $X = 0$, =-1 for $X < 0$
ZERO(X)	=0 for $X = 0$, =1 for $X \neq 0$
TRUNC(X)=INT(X)	Integer part
FRAC(X)	fractional part
ROUND(X)	rounds X to the nearest integer value
CEIL(X)	always returns "ceil" integer value
FLOOR(X)	always returns "floor" integer value
DEC(X)	decrements a value X by 1 and returns a new value
INC(X)	increments a value X by 1 and returns a new value
ARG(X,Y)	argument(phase) of X and Y
RADIUS(X,Y)	= $\sqrt{\text{sqr}(X) + \text{sqr}(Y)}$
POWER(X,Y)	raises X to a power of Y (Y is a floating point value)
IPOWER(X,Y)	raises X to a power of Y (Y is a integer value)
X^Y	raises X to a power of Y (same as above two functions)
EXP(X)	exponent
LN(X)	natural logarithm
LG(X)	decimal logarithm
LOG(X)	base 2 logarithm
SIN(X)	sine

COS(X)	cosine
TAN(X)	Tangent
COTAN(X)	cotangent
ASIN(X)	Arcsine
ACOS(X)	arccosine
ATAN(X)	arctangent
SINH(X)	hyperbolic sine
COSH(X)	hyperbolic cosine
TANH(X)	hyperbolic tangent

M.6 Aggregate Functions

AVG(X1,X2,...)	returns average value of (unlimited number of) arguments.
MAX(X1,X2,...)	maximum of (unlimited number of) arguments.
MIN(X1,X2,...)	minimum of (unlimited number of) arguments.
SUM(X1,X2,...)	sum of (unlimited number of) arguments.
PROD(X1,X2,..)	product of (unlimited number of) arguments.

References

- Abbey, D. E., B. L. Hwang, R. J. Burchette, T. Vancuren and P. K. Mills. 1995a. Estimated long-term ambient concentrations of PM₁₀ and development of respiratory symptoms in a nonsmoking population. *Arch Environ Health*. Vol. 50 (2): 139-52.
- Abbey, D. E., B. E. Ostro, F. Petersen and R. J. Burchette. 1995b. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM_{2.5}) and Other Air Pollutants. *J Expo Anal Environ Epidemiol*. Vol. 5 (2): 137-159.
- Abbey, D. E., F. Petersen, P. K. Mills and W. L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. *Archives of Environmental Health*. Vol. 48 (1): 33- 46.
- Abt Associates Inc. 2000. Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results. Prepared for U. S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC. Bethesda, MD. December.
- Adams, P. F., G. E. Hendershot and M. A. Marano. 1999. Current Estimates from the National Health Interview Survey, 1996. *Vital Health Stat*. Vol. 10 (200): 1-212.
- Agency for Healthcare Research and Quality (AHRQ). 2007. Healthcare Cost and Utilization Project. National Inpatient Sample (NIS) Rockville, Maryland. Available at: <http://www.ahrq.gov/data/hcup/> [accessed February, 2012].
- Agency for Healthcare Research and Quality (AHRQ). 2009. HCUPnet, Healthcare Cost and Utilization Project. Rockville, MD. Available on the Internet at <http://hcupnet.ahrq.gov>
- Alhanti, B.A., Chang, H.H., Winqvist, A., Mulholland, J.A., Darrow, L.A. and Sarnat, S.E., 2016. Ambient air pollution and emergency department visits for asthma: a multi-city assessment of effect modification by age. *Journal of exposure science & environmental epidemiology*, 26(2), pp.180-188.
- Alzheimer's Association. 2020. 2020 Alzheimer's Disease Facts and Figures. Available at: <https://www.alz.org/media/Documents/alzheimers-facts-and-figures.pdf>.
- American Lung Association. 2002a. Trends in Asthma Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.<http://www.lungusa.org/data/asthma/ASTHMAdt.pdf>.
- American Lung Association. 2002b. Trends in Chronic Bronchitis and Emphysema: Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.<http://www.lungusa.org/data/copd/COPD1.pdf>.

- American Lung Association. 2002c. Trends in Morbidity and Mortality: Pneumonia, Influenza, and Acute Respiratory Conditions. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit.http://www.lungusa.org/data/pi/PI_1.pdf.
- American Lung Association. 2010a. Trends in COPD (Chronic Bronchitis and Emphysema): Morbidity and Mortality. American Lung Association Epidemiology and Statistics Unit Research and Program Services Division. February. Table 4. Based on NHIS data (CDC, 2008). Available at <http://www.lungusa.org/finding-cures/our-research/trend-reports/copd-trend-report.pdf>.
- American Lung Association. 2010b. Trends in Asthma Morbidity and Mortality. American Lung Association Epidemiology and Statistics Unit Research and Program Services Division. February. Table 7. Based on NHIS data (CDC, 2008). Available at <http://www.lungusa.org/finding-cures/our-research/trend-reports/asthma-trend-report.pdf>.
- Anderson, G.B. and Bell, M.L. 2010. Does one size fit all? The suitability of standard ozone exposure metric conversion ratios and implications for epidemiology. *J Expo Sci Environ Epidemiol* 20(1): 2-11.
- Babin, S. M., H. S. Burkom, et al. 2007. "Pediatric patient asthma-related emergency department visits and admissions in Washington, DC, from 2001-2004, and associations with air quality, socio-economic status and age group." *Environ Health* 6: 9.
- Baena-Cagnani, C, Rossi, GA and Canonica, GW. 2007. Airway remodelling in children: When does it start? *Curr Opin Allergy Clin Immunol* 7(2): 196-200.
- Barry, V., Klein, M., Winqvist, A., Chang, H.H., Mulholland, J.A., Talbott, E.O., Rager, J.R., Tolbert, P.E., & Sarnat, S.E. 2018. Characterization of the concentration-response curve for ambient ozone and acute respiratory morbidity in 5 US cities. *J Expo Sci Environ Epidemiol*.
- Baxter, L.K., Crooks, J.L. and Sacks, J.D. 2017. Influence of exposure differences on city-to-city heterogeneity in PM2.5-mortality associations in US cities. *Environ Health* 16(1): 1.
- Bell, M. L., K. Ebisu, et al. 2008. "Seasonal and Regional Short-term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999-2005." *American Journal of Epidemiology* 168 (11): 1301-1310.
- Bell, M. L., F. Dominici and J. M. Samet. 2005. A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology*. Vol. 16 (4): 436-45.
- Bell, M. L., A. McDermott, S. L. Zeger, J. M. Samet and F. Dominici. 2004. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *Jama*. Vol. 292 (19): 2372-8.

- Bell, M. L., Son, J. Y., Peng, R. D., Wang, Y., & Dominici, F. 2015. Ambient PM_{2.5} and Risk of Hospital Admissions: Do Risks Differ for Men and Women?. *Epidemiology*, 26(4): 575–579.
- Belova, A., E. Post and D. McCubbin. 2007. Using Expert Distributions in BenMAP. Memorandum prepared for Bryan Hubbell at U.S. EPA, Office of Air Quality Planning and Standards. Prepared by Abt Associates, Bethesda, MD. March 7.
- Belova, A, Fann, N, Haskell, J, Hubbell, B and Narayan, T. 2020. Estimating Lifetime Cost of Illness. An Application to Asthma. *Ann Am Thorac Soc* 17(12): 1558-1569.
- Bhattacharyya, N. 2011. Incremental healthcare utilization and expenditures for allergic rhinitis in the United States. *The Laryngoscope* 121(9): 1830-1833.
- Blumenschein, K. and M. Johannesson. 1998. Relationship between quality of life instruments, health state utilities, and willingness to pay in patients with asthma. *Ann Allergy Asthma Immunol*. Vol. 80 (2): 189-94.
- Burnett, R. T., M. Smith-Doiron, D. Stieb, M. E. Raizenne, J. R. Brook, R. E. Dales, J. A. Leech, S. Cakmak and D. Krewski. 2001. Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age. *Am J Epidemiol*. Vol. 153 (5): 444-52.
- Burnett, R. T., C. Arden Pope III, M. Ezzati, C. Olives, S. S. Lim, S. Mehta, H. H. Shin, G. Singh, B. Hubbell, M. Brauer, H. R. Anderson, K. R. Smith, J. R. Balmes, N. G. Bruce, H. Kan, F. Laden, A. Prüss-Ustün, M. C. Turner, S. M. Gapstur, W. R. Diver, and A. Cohen. 2014. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environmental Health Perspectives*. Vol. 122 (4): 397-403.
- Burnett, R., Chen, H., Szyszkowicz, M., Fann, N., Hubbell, B., Pope, C.A., Spte, J.S., Brauer, M., Cohen, A., Weichenthal, S., Coggin, J., Qian, D., Brunekreef, B., Frostad, J., Lim, S.S., Kan, H., Walker, K.D., Thurston, G.D., Hayes, R.B., Lim, C.C., Turner, M.C., Jerrett, M., Krewski, D., Gapstur, M., Diver, W.R., Ostro, B., Goldberg, D., Crouse, D.L., Martin, R.V., Peters, P., Pinault, L., Tjepkema, M., van Donkelaar, A., Villeneuve, P.J., Miller, A.B., Yin, P., Zhou, M., Wang, L., Janssen, N.A.H., Marra, M., Atkinson, R.W., Tsang, H., Thach, T.Q., Cannon, J.B., Allen, R.T., Hart, J.E., Laden, F., Cesaroni, G., Forastiere, F., Weinmayr, G., Jaensch, A., Nagel, G., Concin, H., Spadaro, J.V. 2018. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. *Proceedings of the National Academy of Sciences*. Vol. 115 (38): 9592-9597.
- Cakmak, S., Dales, R., Judek, S. 2006. Respiratory Health Effects of Air Pollution Gases: Modification by Education and Income. *Archives of Environmental & Occupational Health*, 61(1), 5-10.
- Centers for Disease Control and Prevention (CDC). 2008. National Center for Health Statistics. National Health Interview Survey, 1999-2008.

- Chen, L., B. L. Jennison, W. Yang and S. T. Omaye. 2000. Elementary school absenteeism and air pollution. *Inhal Toxicol.* Vol. 12 (11): 997-1016.
- Chiang, P. L. 1967. Variance and Covariance of Life Table Functions Estimated from a Sample of Deaths. National Center for Health Statistics. Washington, DC. March.
- Cohen, A. J., M. Brauer, R. Burnett, H.R. Anderson, J. Frostad, K. Estep, K. Balakrishnan, B. Brunekreef, L. Dandona, R. Dandona, V. Feigin, G. Freedman, B. Hubbell, A. Jobling, H. Kan, L. Knibbs, Y. Liu, R. Martin, L. Morawska, C. A. Pope III, H. Shin, K. Straif, G. Shaddick, M. Thomas, R. van Dingenen, A. van Donkelaar, T. Vos, C. J. L. Murray, M. H. Forouzanfar. 2017. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Disease Study 2015. *Lancet.* Vol. 389: 1907-1918.
- Collet, D. 1994. *Modelling Survival Data in Medical Research.* Chapman & Hall: New York.
- Cropper, ML and Krupnick, AJ. 1990. Social costs of chronic heart and lung disease. *Quality of the Environment Division, Resources for the Future.*
- Cropper, Maureen R. and Alan J. Krupnick. 1999. The social costs of chronic heart and lung disease, in Maureen Cropper, *Valuing Environmental Benefits: Selected Essays of Maureen Cropper,* Edward Elgar: Northampton, MA.
- Daya, M., R. Schmicker, S. May, and L. Morrison. 2015. Current burden of cardiac arrest in the United States: Report from the Resuscitation Outcomes Consortium. Paper commissioned by the Committee on the Treatment of Cardiac Arrest: Current Status and Future Directions.
- De Pablo-Fernandez, E, Tur, C, Revesz, T, Lees, AJ, Holton, JL and Warner, TT. 2017. Association of Autonomic Dysfunction With Disease Progression and Survival in Parkinson Disease. *JAMA Neurol* 74(8): 970-976.
- Decisioneering. 1996. *Crystal Ball: Forecasting and Risk Analysis for Spreadsheet Users: User Manual.* Version 4.0. Denver, CO. www.decisioneering.com.
- DerSimonian, R. and N. Laird. 1986. Meta-Analysis in Clinical Trials. *Controlled Clinical Trials.* Vol. 7: 177-188.
- Dewanji, A. and S. H. Moolgavkar. 2000. A Poisson process approach for recurrent event data with environmental covariates. *Environmetrics.* Vol. 11: 665-673.
- Dewanji, A. and S. H. Moolgavkar. 2002. Choices of stratification in Poisson process analysis of recurrent event data with environmental covariates. *Stat. Med.* Vol. 21: 3383-3393.
- Di, Q., Wang, Y., Zanobetti, A., Wang, Y., Koutrakis, P., Choirat, C., Dominici, F. and Schwartz, J.D. 2017. Air pollution and mortality in the Medicare population. *New Engl J Med* 376(26): 2513-2522.

- Dickie, M. and S. Gerking. 1987. Reconciling Averting Behavior and Contingent Valuation Benefit Estimates of Reducing Symptoms of Ozone Exposure (draft), as cited in Neumann, J. E., M. Dickie, and R.E. Unsworth. 1994. Prepared by Industrial Economics. Prepared for Jim DeMocker, U.S. EPA, Office of Air and Radiation. March 31.
- Dickie, M and Messman, VL. 2004. Parental altruism and the value of avoiding acute illness: are kids worth more than parents? *J Environ Econ Manage* 48(3): 1146-1174.
- Dickie, M. and V. L. Ulery. 2002. Parental Altruism and the Value of Avoiding Acute Illness: Are Kids Worth More Than Parents? (Paper to be submitted for publication. Presented at Association of Environmental and Resource Economists 2001 Workshop, "Assessing and Managing Environmental and Public Health Risks."). December.
- Dockery, D. W., J. Cunningham, A. I. Damokosh, L. M. Neas, J. D. Spengler, P. Koutrakis, J. H. Ware, M. Raizenne and F. E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. *Environmental Health Perspectives*. Vol. 104 (5): 500-505.
- Dockery, D. W., F. E. Speizer, D. O. Stram, J. H. Ware, J. D. Spengler and B. G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. *Am Rev Respir Dis*. Vol. 139: 587-594.
- Eisenstein, E. L., L. K. Shaw, K. J. Anstrom, C. L. Nelson, Z. Hakim, V. Hasselblad and D. B. Mark. 2001. Assessing the clinical and economic burden of coronary artery disease: 1986- 1998. *Med Care*. Vol. 39 (8): 824-35.
- Ensr, K.B., Raun, L.H., & Persse, D. 2013. A Case-Crossover Analysis of Out-of-Hospital Cardiac Arrest and Air Pollution. *Circulation* 127: 1192-1199.
- ESEERCO. 1994. New York State Environmental Externalities Cost Study. Report 2: Methodology. Empire State Electric Energy Research Corporation.
- Fung, K. Y. K., Daniel; Chen, Yue; Burnett, Rick; Cakmak, Sabit. 2003. Comparison of time series and case-crossover analyses of air pollution and hospital admission data. *International Journal of Epidemiology*. Vol. 32: 1064-1070.
- Garcia, E., Berhane, K.T., Islam, T., McConnell, R., Urman, R., Chen, Z., & Gilliland, F.D. 2019. Association of Changes in Air Quality With Incident Asthma in Children in California, 1993-2014. *JAMA-J Am Med Assoc*, 321(19): 1906-1915.
- GeoLytics Inc. 2001. CensusCD® 1990 + Maps, Release 4.1. CD-ROM. GeoLytics, Inc.
- Gharibvand, L., Shavlik, D., Ghamsary, M., Beeson, W.L., Soret, S., Knutsen, R., & Knutsen, S.F. 2016. The association between ambient fine particulate air pollution and lung cancer incidence: results from the AHSMOG-2 study. *Environ Health Perspect* 125 (3): 378-384.

- Gilliland, F. D., K. Berhane, E. B. Rappaport, D. C. Thomas, E. Avol, W. J. Gauderman, S. J. London, H. G. Margolis, R. McConnell, K. T. Islam and J. M. Peters. 2001. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology*. Vol. 12 (1): 43-54.
- Glad, J.A., L.L. Brink, E.O. Talbott, P.C. Lee, X. Xu, M. Saul, and J. Rager. 2012. The Relationship of Ambient Ozone and PM_{2.5} Levels and Asthma Emergency Department Visits: Possible Influence of Gender and Ethnicity. *Archives of Environmental & Occupational Health*. Vol 62 (2): 103-108.
- Goodwin, N, Smith, J, Davies, A, Perry, C, Rosen, R, Dixon, A, Dixon, J and Ham, C. 2011. A report to the Department of Health and the NHS Future Forum; Integrated Care for Patients and Populations: Improving Outcomes by Working Together.
- Greene, W. H. 1997. *Econometric Analysis*. Prentice Hall: Upper Saddle River, NJ.
- Guerra, S, Wright, AL, Morgan, WJ, Sherrill, DL, Holberg, CJ and Martinez, FD. 2004. Persistence of asthma symptoms during adolescence: role of obesity and age at the onset of puberty. *Am J Respir Crit Care Med* 170(1): 78-85.
- Haase, N. 2002. Phone conversation. American Heart Association, October.
- Huang, Y, F. Dominici and M. L. Bell. 2005. Bayesian hierarchical distributed lag models for summer ozone exposure and cardio-respiratory mortality. *Environmetrics*. Vol. 16: 547-562.
- Industrial Economics Incorporated (IEc). 1993. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. September 30.
- Industrial Economics Incorporated (IEc). 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by J.E. Neumann, M.T. Dickie, and R.E. Unsworth. March 31.
- Industrial Economics Incorporated (IEc). 2006. Expanded Expert Judgment Assessment of the Concentration-Response Relationship Between PM_{2.5} Exposure and Mortality, Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by IEc. Cambridge, MA. September 21.
- Ito, K. 2003. Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Health Effects Institute. Boston, MA. May.
- Ito, K., S. F. De Leon and M. Lippmann. 2005. Associations between ozone and daily mortality: analysis and meta-analysis. *Epidemiology*. Vol. 16 (4): 446-57.

- Ito, K, Ross, Z, Zhou, J, Nádas, A, Lippmann, M. and Thurston, G.D. 2013. National Particle Component Toxicity (NPACT) initiative: Study 3. Time-series analysis of mortality, hospitalizations, and ambient PM_{2.5} and its components. Boston, MA, Health Effects Institute: 95-125.
- Ito, K. and G. D. Thurston. 1996. Daily PM₁₀/mortality associations: an investigations of at-risk subpopulations. *Journal of Exposure Analysis and Environmental Epidemiology*. Vol. 6 (1): 79-95.
- Ito, K, G. D. Thurston and R. A. Silverman. 2007. Characterization of PM_{2.5}, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. *J Expo Sci Environ Epidemiol*. Vol. 17 Suppl 2: S45-60. Jerrett, M, Turner, MC, Beckerman, BS, Pope, CA, van Donkelaar, A, Martin, RV, Serre, M, Crouse, D, Gapstur, SM, Krewski, D, Diver, WR, Coogan, PF, Thurston, GD and Burnett, RT. 2017. Comparing the health effects of ambient particulate matter estimated using ground-based versus remote sensing exposure estimates. *Environ Health Perspect* 125(4): 552-559.
- Jerrett, M., R. Burnett, A. I. Pope, K. Ito, G. Thurston, D. Krewski, Y. Shi, E. Calle and M. Thun. 2009. Long-Term Ozone Exposure and Mortality. *The New England Journal of Medicine*. Vol. 360 (11): 1085-95.
- Jones, R.R., Hogrefe, C., Fitzgerald, E.F., Hwang, S.A., Özkaynak, H., Garcia, V.C., & Lin, S. 2015. Respiratory hospitalizations in association with fine PM and its components in New York State. *J Air Waste Manag Assoc*, 65(5): 559-569.
- Judge, G. G., W. E. Griffiths, R. C. Hill, H. Lutkepohl and T.-C. Lee. 1985. *The Theory and Practice of Econometrics*. John Wiley and Sons: New York. Katsouyanni, K and J.M. Samet. 2009. *Air Pollution and Health: A European and North American Approach (APHENA)*. Health Effects Institute, Research Report 142.
- Jutkowitz, E, Kane, RL, Gaugler, JE, MacLehose, RF, Dowd, B and Kuntz, KM. 2017. Societal and Family Lifetime Cost of Dementia: Implications for Policy. *J Am Geriatr Soc* 65(10): 2169-2175.
- Katsouyanni, K, Samet, J. M., Anderson, H. R., Atkinson, R., Tertre, A. L., Medina, S., et al. 2009. *Air Pollution and Health: A European and North American Approach (APHENA)*: Health Effects Institute.
- Kaye, DR, Min, HS, Herrel, LA, Dupree, JM, Ellimoottil, C and Miller, DC. 2018. Costs of Cancer Care Across the Disease Continuum. *Oncologist* 23(7): 798-805.
- Kennedy, P. 1990. *A Guide to Econometrics*. MIT Press: Cambridge, MA.
- King, K, Starsinic, M., Hernandez Viver, A., Beaghen, M. 2015, "The Reliability of ACS 5-Year Estimates of Race Groups and American Indian and Alaska Native Populations", U.S. Census Bureau.

- Kioumourtzoglou, M.A., Schwartz, J.D., Weisskopf, M.G., Melly, S.J., Wang, Y., Dominici, F., & Zanobetti, A. 2016. Long-term PM_{2.5} exposure and neurological hospital admissions in the northeastern United States. *Environ Health Perspect*, 124(1): 23–29.
- Kloog, I., B.A. Coull, A. Zanobetti, P. Koutrakis, J.D. Schwartz. 2012. Acute and Chronic Effects of Particles on Hospital Admissions in New-England. *PLoS ONE*. Vol 7 (4): 1-8.
- Krall, J. R., Mulholland, J. A., Russell, A. G., Balachandran, S., Winquist, A., Tolbert, P. E., Waller, L.A., & Sarnat, S. E. 2016. Associations between Source-Specific Fine Particulate Matter and Emergency Department Visits for Respiratory Disease in Four U.S. Cities. *Environ Health Perspect*, 125(1), 97–103.
- Krewski, D., M. Jerrett, R. Burnett, R. Ma, E. Hughes, Y. Shi, M. C. Turner, C. A. I. Pope, G. Thurston, E. Calle and M. J. Thun. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. HEI Research Report, 140, Health Effects Institute, Boston, MA.
- Krupnick, A. J. and M. L. Cropper. 1992. The Effect of Information On Health Risk Valuations. *Journal of Risk and Uncertainty*. Vol. 5 (1): 29-48.
- Laden, F., J. Schwartz, F. E. Speizer and D. W. Dockery. 2006. Reduction in Fine Particulate Air Pollution and Mortality: Extended follow-up of the Harvard Six Cities Study. *Am J Respir Crit Care Med*. Vol. 173 (6): 667-72.
- Lee, WC, Christensen, MC, Joshi, AV and Pashos, CL. 2007. Long-term cost of stroke subtypes among Medicare beneficiaries. *Cerebrovascular Diseases* 23(1): 57-65.
- Levy, J. I., S. M. Chemerynski and J. A. Sarnat. 2005. Ozone exposure and mortality: an empiric bayes metaregression analysis. *Epidemiology*. Vol. 16 (4): 458-68.
- Lewis, T.C., Robins, T.G., Mentz, G.B., Zhang, X., Mukherjee, B., Lin, X., Keeler, G.J., Dvonch, J.T., Yip, F.Y., O'Neill, M.S., Parker, E.A., Israel, B.A., Max, P.T., Reyes, A., & Committee, CAAACS. 2013. Air pollution and respiratory symptoms among children with asthma: Vulnerability by corticosteroid use and residence area. *Sci Total Environ* 448: 48-55.
- Lin, M., Stieb, D.M., Chen, Y. 2005. Coarse Particulate Matter and Hospitalization for Respiratory Infections in Children Younger than 15 Years in Toronto: A Case-Crossover Analysis. *American Academy of Pediatrics*, 116, 235-240.
- Lippmann, M., K. Ito, A. Nádas and R. Burnett. 2000. Association of Particulate Matter Components with Daily Mortality and Morbidity in Urban Populations. *Health Effects Institute*. Number 95. August.
- Loehman, E. T., S. V. Berg, A. A. Arroyo, R. A. Hedinger, J. M. Schwartz, M. E. Shaw, R. W. Fahien, V. H. De, R. P. Fische, D. E. Rio, W. F. Rossley and A. E. S. Green. 1979. Distributional Analysis of Regional Benefits and Cost of Air Quality Control. *Journal of Environmental Economics and Management*. Vol. 6: 222-243.

- Luengo-Fernandez, R, Gray, AM and Rothwell, PM. 2012. A population-based study of hospital care costs during 5 years after transient ischemic attack and stroke. *Stroke* 43(12): 3343-3351.
- Mar, T.F., J.Q. Koenig. 2009. Relationship between visits to emergency departments for asthma and ozone exposure in greater Seattle, Washington. *Annals of Allergy, Asthma & Immunology*, Vol. 103(6):474-479.
- Mar, T. F., J. Q. Koenig, et al. 2010. Associations between asthma emergency visits and particulate matter sources, including diesel emissions from stationary generators in Tacoma, Washington. *Inhal Toxicol* 22 (6): 445-8.
- Mar, T. F., T. V. Larson, et al. 2004. An analysis of the association between respiratory symptoms in subjects with asthma and daily air pollution in Spokane, Washington." *Inhal Toxicol* 16 (13): 809-15.
- McConnell, R., K. Berhane, F. Gilliland, S. J. London, H. Vora, E. Avol, W. J. Gauderman, H. G. Margolis, F. Lurmann, D. C. Thomas and J. M. Peters. 1999. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect*. Vol. 107 (9): 757-60.
- McConnell, R., Islam, T., Shankardass, K., Jerrett, M., Lurmann, F., Gilliland, F., Gauderman, J., Avol, E., Künzli, N., Yao, L., Peters, J., & Berhane, K. 2010. Childhood Incident Asthma and Traffic-Related Air Pollution at Home and School. *Environ Health Perspect*, 118(7), 1021–1026.
- Medina-Ramon, M. and Schwartz, J. 2008. Who is more vulnerable to die from ozone air pollution? *Epidemiology*, 19(5), 672-679.
- Moolgavkar, S. H. 2000a. Air Pollution and Hospital Admissions for Chronic Obstructive Pulmonary Disease in Three Metropolitan Areas in the United States. *Inhalation Toxicology*. Vol. 12 (Supplement 4): 75-90.
- Moolgavkar, S. H. 2000b. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. *J Air Waste Manag Assoc*. Vol. 50 (7): 1199-206.
- Moolgavkar, S. H. 2003. Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Health Effects Institute. Boston, MA. May.
- Moolgavkar, S. H., E. G. Luebeck and E. L. Anderson. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis St. Paul and Birmingham. *Epidemiology*. Vol. 8 (4): 364-370.
- Moolgavkar, S. H., E. G. Luebeck, T. A. Hall and E. L. Anderson. 1995. Air Pollution and Daily Mortality in Philadelphia. *Epidemiology*. Vol. 6 (5): 476-484.

- Mortimer, K. M., L. M. Neas, D. W. Dockery, S. Redline and I. B. Tager. 2002. The effect of air pollution on inner-city children with asthma. *Eur Respir J*. Vol. 19 (4): 699-705.
- Mrozek, J. R. and L. O. Taylor. 2002. What Determines the Value of Life? A Meta-Analysis. *Journal of Policy Analysis and Management*. Vol. 21: 253-270.
- Mu, F, Hurley, D, Betts, K, Messali, A, Paschoalin, M, Kelley, C and Wu, E. 2017. Real-world costs of ischemic stroke by discharge status. *Current medical research and opinion* 33(2): 371-378.
- National Center for Health Statistics. 1999. National Vital Statistics Reports. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. Washington, DC. Volume 47, Number 19. June 30.
- NCI. 2015. SEER, Surveillance, Epidemiology, and End Results Program. National Institutes of Health, National Cancer Institute (NCI). Bethesda, MD.
- Nelder, J. A. and R. Mead. 1965. A simplex algorithm for function minimization. *Computer Journal*. Vol. 7: 308-313.
- Nicholson, G, Gandra, SR, Halbert, RJ, Richhariya, A and Nordyke, RJ. 2016. Patient-level costs of major cardiovascular conditions: a review of the international literature. *ClinicoEconomics and outcomes research: CEOR* 8: 495.
- Nishimura, K.K., Galanter, J.M., Roth, L.A., Oh, S.S., Thakur, N., Nguyen, E.A., Thyne, S., Farber, H.J., Serebrisky, D., Kumar, R., Brigino-Buenaventura, E., Davis, A., LeNoir, M.A., Meade, K., Rodriguez-Cintron, W., Avila, P.C., Borrell, L.N., Bibbins-Domingo, K., Rodriguez-Santana, J.R., Sen, S., Lurmann, F., Balmes, J.R., & Burchard, E.G. 2013. Early-Life Air Pollution and Asthma Risk in Minority Children: The GALA II and SAGE II Studies. *Am J Respir Crit Care Med* Vol, 188(3), 309–318.
- Noelke, C., Outric, M., Baek, M., Reece, J., Osypuk, T.L., McArdle, N., Ressler, R.R., Acevedo-Garcia, D. 2022. Connecting past to present: Examining different approaches to linking historical redlining to present day health inequities. *PloS One*, 17(5), e0267606–e0267606. <https://doi.org/10.1371/journal.pone.0267606>
- Norris, G., S. N. YoungPong, J. Q. Koenig, T. V. Larson, L. Sheppard and J. W. Stout. 1999. An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect*. Vol. 107 (6): 489-93.
- Ochs, M, Nyengaard, JR, Jung, A, Knudsen, L, Voigt, M, Wahlers, T, Richter, J and Gundersen, HJ. 2004. The number of alveoli in the human lung. *Am J Respir Crit Care Med* 169(1): 120-124.
- O'Connor, R. M. and G. C. Blomquist. 1997. Measurement of Consumer-Patient Preferences Using a Hybrid Contingent Valuation Method. *Journal of Health Economics*. Vol. 16: 667-683.

- O'Connor, G.T., L. Neas, B. Vaughn, M. Kattan, H. Mitchell, E. F. Crain, R. Evans III, R. Gruchalla, W. Morgan, J. Stout, G. K. Adam, and M. Lippmann. 2008. Acute respiratory health effects of air pollution on children with asthma in US inner cities. *Journal of Allergy and Clinical Immunology*, Vol. 121(5): 1133-1139.
- O'Sullivan, AK, Rubin, J, Nyambose, J, Kuznik, A, Cohen, DJ and Thompson, D. 2011. Cost estimation of cardiovascular disease events in the US. *Pharmacoeconomics* 29(8): 693-704.
- Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens and M. White. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology*. Vol. 12 (2): 200-8.
- Ostro, B. D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. *Journal of Environmental Economics and Management*. Vol. 14: 87-98.
- Ostro, B. D., M. J. Lipsett, M. B. Wiener and J. C. Selner. 1991. Asthmatic Responses to Airborne Acid Aerosols. *Am J Public Health*. Vol. 81 (6): 694-702.
- Ostro, B., Malig, B., Hasheminassab, S., Berger, K., Chang, E. & Sioutas, C. 2016. Associations of Source-Specific Fine Particulate Matter With Emergency Department Visits in California. *Am J Epidemiol* 184(6): 450-459.
- Ostro, B., Roth, L., Malig, M., & Marty, M. 2009. The Effects of Fine Particle Components on Respiratory Hospital Admissions in Children. *Environ Health Perspect*, 117(3): 475-480.
- Ostro, B. D. and S. Rothschild. 1989. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. *Environ Res*. Vol. 50 (2): 238-247. Owings, M. F. and L. Lawrence. 1999. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1997. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 13, No. 145. December.
- Parker, J.D., Akinbami, L.J., & Woodruff, T.J. 2009. Air Pollution and Childhood Respiratory Allergies in the United States. *Environ Health Perspect* 117(1): 140-147.
- Paulu, C. and Smith, A.E. 2008. Tracking Associations Between Ambient Ozone and Asthma-Related Emergency Department Visits Using Case-Crossover Analysis. *Journal of Public Health Management and Practice*, 14(6), 581-591.
- Peel, J. L., P. E. Tolbert, M. Klein, K. B. Metzger, W. D. Flanders, K. Todd, J. A. Mulholland, P. B. Ryan and H. Frumkin. 2005. Ambient air pollution and respiratory emergency department visits. *Epidemiology*. Vol. 16 (2): 164-74.
- Peng, R. D., M. L. Bell, et al. 2009. Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. *Environ Health Perspect*, Vol. 117 (6): 957-63.

- Peng, R. D., H. H. Chang, et al. 2008. "Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients." *Jama*, Vol. 299 (18): 2172-9.
- Peters, A., D. W. Dockery, J. E. Muller and M. A. Mittleman. 2001. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. Vol. 103 (23): 2810-5. <http://www.circulationaha.org/cgi/content/full/103/23/2810>.
- Pope, C. A., 3rd, J. B. Muhlestein, et al. 2006. "Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution." *Circulation*, Vol. 114 (23): 2443-8.
- Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Jama*. Vol. 287 (9): 1132-41.
- Pope, C. A., D. W. Dockery, J. D. Spengler and M. E. Raizenne. 1991. Respiratory Health and PM₁₀ Pollution - a Daily Time Series Analysis. *American Review of Respiratory Disease*. Vol. 144 (3): 668-674.
- Pope, C.A., Turner, M.C., Burnett, R., Jerrett, M., Gapstur, S.M., Diver, W.R., Krewski, D. and Brook, R.D. 2015. Relationships between fine particulate air pollution, cardiometabolic disorders and cardiovascular mortality. *Circ Res* 116(1): 108-U258.
- Popovic, J. R. 2001. 1999 National Hospital Discharge Survey: annual summary with detailed diagnosis and procedure data. *Vital Health Stat* 13. (151): i-v, 1-206.
- Rabinovitch, N., Strand, M., & Gelfand, E.W. 2006. Particulate Levels Are Associated with Early Asthma Worsening in Children with Persistent Disease. *Am J Respir Crit Care Med*, 173: 1098-1105.
- Ransom, M. R. and C. A. Pope. 1992. Elementary school absences and PM₁₀ pollution in Utah Valley. *Environ Res*. Vol. 58 (2): 204-19.
- Roger V.L., A.S. Go, D.M. Lloyd-Jones, E.J. Benjamin, J.D. Berry, W.B. Borden, D.M. Bravata, S. Dai, E.S. Ford, C.S. Fox, H.J. Fullerton, C. Gillespie, S.M. Hailpern, J.A. Heit, V.J. Howard, B.M. Kissel, S.J. Kittner, D.T. Lackland, J.H. Lichtman, L.D. Lisabeth, D.M. Makuc, G.M. Marcus, A. Marelli, D.B. Matchar, C.S. Moy, D. Mozaffarian, M.E. Mussolino, G. Nichol, N.P. Paynter, E.Z. Soliman, P.D. Sorlie, N. Sotoodehnia, T.N. Turan, S.S. Virani, N.D. Wong, D. Woo and M.B. Turner; on behalf of the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2012 update: a report from the American Heart Association. *Circulation*. 2012;125:e2-e220.
- Rosamond, W., G. Broda, E. Kawalec, S. Rywik, A. Pajak, L. Cooper and L. Chambless. 1999. Comparison of medical care and survival of hospitalized patients with acute myocardial infarction in Poland and the United States. *Am J Cardiol*. Vol. 83 (8): 1180-5.

- Rosenthal, F.S., Carney, J.P., & Olinger, M.L. 2008. Out-of-Hospital Cardiac Arrest and Airborne Fine Particulate Matter: A Case–Crossover Analysis of Emergency Medical Services Data in Indianapolis, Indiana. *Environ Health Perspect* 116(5): 631-636.
- Rowe, R. D. and L. G. Chestnut. 1986. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis -- Executive Summary. Prepared for U.S. Environmental Protection Agency, Office of Policy Analysis. Prepared by Energy and Resource Consultants, Inc. Washington, DC. EPA-230-09-86-018. March.
- Russell, M. W., D. M. Huse, S. Drowns, E. C. Hamel and S. C. Hartz. 1998. Direct medical costs of coronary artery disease in the United States. *Am J Cardiol*. Vol. 81 (9): 1110-5.
- Sarnat, J. A., S. E. Sarnat, W. D. Flanders, H. H. Chang, J. Mulholland, L. Baxter, V. Isakob, and H. Özkaynak. 2013. Spatiotemporally resolved air exchange rate as a modifier of acute air pollution-related morbidity in Atlanta. *Journal of Exposure Science and Environmental Epidemiology* 23, 606-615.
- Samet, J. M., S. L. Zeger, J. E. Kelsall, J. Xu and L. S. Kalkstein. 1997. Air Pollution, Weather, and Mortality in Philadelphia 1973-1988. Health Effects Institute. Cambridge, MA. March.
- Schildcrout, J. S., L. Sheppard, T. Lumley, J. C. Slaughter, J. Q. Koenig and G. G. Shapiro. 2006. Ambient air pollution and asthma exacerbations in children: an eight-city analysis. *Am J Epidemiol*. Vol. 164 (6): 505-17.
- Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. *American Journal of Respiratory and Critical Care Medicine*. Vol. 150 (3): 648-655.
- Schwartz, J. 1994b. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. *Archives of Environmental Health*. Vol. 49 (5): 366-374.
- Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. *Thorax*. Vol. 50 (5): 531-538.
- Schwartz, J. 2005. How sensitive is the association between ozone and daily deaths to control for temperature? *Am J Respir Crit Care Med*. Vol. 171 (6): 627-31.
- Schwartz, J., C. Spix, G. Touloumi, L. Bacharova, T. Barumamdzadeh, A. le Tertre, T. Piekarksi, A. Ponce de Leon, A. Ponka, G. Rossi, M. Saez and J.P. Schouten. 1996. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J. Epidemiol. Community Health*. Vol. 50 Suppl. 1: S3-11.
- Schwartz, J., D. W. Dockery, L. M. Neas, D. Wypij, J. H. Ware, J. D. Spengler, P. Koutrakis, F. E. Speizer and B. G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. *Am J Respir Crit Care Med*. Vol. 150 (5): 1234-1242.

- Schwartz, J. and L. M. Neas. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology*. Vol. 11 (1): 6- 10.
- Sheppard, L. 2003. Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Health Effects Institute. Boston, MA. May.
- Sheppard, L., D. Levy, G. Norris, T. V. Larson and J. Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology*. Vol. 10 (1): 23-30.
- Siegel, RL, Miller, KD and Jemal, A. 2019. Cancer statistics, 2019. *CA Cancer J Clin* 69(1): 7-34.
- Slaughter, J. C., E. Kim, et al. 2005. "Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington." *J Expo Anal Environ Epidemiol* 15(2): 153-9.
- Samet, J. M., S. L. Zeger, J. E. Kelsall, J. Xu and L. S. Kalkstein. 1997. *Air Pollution, Weather, and Mortality in Philadelphia 1973-1988*. Health Effects Institute. Cambridge, MA. March.
- Schildcrout, J. S., L. Sheppard, T. Lumley, J. C. Slaughter, J. Q. Koenig and G. G. Shapiro. 2006. Ambient air pollution and asthma exacerbations in children: an eight-city analysis. *Am J Epidemiol*. Vol. 164 (6): 505-17.
- Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. *American Journal of Respiratory and Critical Care Medicine*. Vol. 150 (3): 648-655.
- Schwartz, J. 1994b. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. *Archives of Environmental Health*. Vol. 49 (5): 366-374.
- Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. *Thorax*. Vol. 50 (5): 531-538.
- Schwartz, J. 2005. How sensitive is the association between ozone and daily deaths to control for temperature? *Am J Respir Crit Care Med*. Vol. 171 (6): 627-31.
- Schwartz, J., C. Spix, G. Touloumi, L. Bacharova, T. Barumamdzadeh, A. le Tertre, T. Piekarksi, A. Ponce de Leon, A. Ponka, G. Rossi, M. Saez and J.P. Schouten. 1996. Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. *J. Epidemiol. Community Health*. Vol. 50 Suppl. 1: S3-11.
- Schwartz, J., D. W. Dockery, L. M. Neas, D. Wypij, J. H. Ware, J. D. Spengler, P. Koutrakis, F. E. Speizer and B. G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. *Am J Respir Crit Care Med*. Vol. 150 (5): 1234-1242.

- Schwartz, J. and L. M. Neas. 2000. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology*. Vol. 11 (1): 6- 10.
- Sheppard, L. 2003. Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Health Effects Institute. Boston, MA. May.
- Sheppard, L., D. Levy, G. Norris, T. V. Larson and J. Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. *Epidemiology*. Vol. 10 (1): 23-30.
- Silverman, R.A., Ito, K., Freese, J., Kaufman, B.J., De Claro, D., Braun, J., & Prezant, D.J. 2010. Association of Ambient Fine Particles With Out-of-Hospital Cardiac Arrests in New York City. *Am J Epidemiol*, 172(8): 917-923.
- Slaughter, J. C., E. Kim, et al. 2005. "Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington." *J Expo Anal Environ Epidemiol* 15(2): 153-9.
- Smith, D. H., D. C. Malone, K. A. Lawson, L. J. Okamoto, C. Battista and W. B. Saunders. 1997. A national estimate of the economic costs of asthma. *Am J Respir Crit Care Med*. Vol. 156 (3 Pt 1): 787-93.
- Soni, A. 2008. Allergic Rhinitis: Trends in use and expenditures, 2000 and 2005. Medical Expenditure Panel Survey, Agency for Healthcare Research and Quality.
- Sparrow, D, O'Connor, GT, Rosner, B, Segal, MR and Weiss, ST. 1991. The influence of age and level of pulmonary function on nonspecific airway responsiveness. The Normative Aging Study. *Am Rev Respir Dis* 143(5 Pt 1): 978-982.
- Stanford, R., T. McLaughlin and L. J. Okamoto. 1999. The cost of asthma in the emergency department and hospital. *Am J Respir Crit Care Med*. Vol. 160 (1): 211-5.
- Sullivan, J., L. Sheppard, et al. 2005. "Relation between short-term fine-particulate matter exposure and onset of myocardial infarction." *Epidemiology* 16 (1): 41-8.
- Talbott, E.O., Rager, J.R., Benson, S., Brink, L.A., Bilonick, R.A., & Wu, C. 2014. A case-crossover analysis of the impact of PM_{2.5} on cardiovascular disease hospitalizations for selected CDC tracking states. *Environ Res* 143: 455-465.
- Tétreault, L.F., Doucet, M., Gamache, P., Fournier, M., Brand, A., Kosatsky, T., & Smargiassi, A. 2016. Childhood exposure to ambient air pollutants and the onset of asthma: an administrative cohort study in Québec. *Environ Health Perspect* 124(8):1276-1282.

- Thurston, GD, Ahn, J, Cromar, KR, Shao, Y, Reynolds, HR, Jerrett, M, Lim, CC, Shanley, R, Park, Y and Hayes, RB. 2016. Ambient particulate matter air pollution exposure and mortality in the NIH-AARP Diet and Health Cohort. *Environ Health Perspect* 124(4): 484-490.
- Tolley, G. S. and et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. Prepared for U.S. Environmental Protection Agency. January.
- Trivedi, M. and Denton, E. 2019. Asthma in Children and Adults-What Are the Differences and What Can They Tell us About Asthma? *Front Pediatr* 7: 256.
- Turner, M.C., Jerrett, M., Pope, A., III, Krewski, D., Gapstur, S.M., Diver, W.R., Beckerman, B.S., Marshall, J.D., Su, J., Crouse, D.L. and Burnett, R.T. 2016. Long-term ozone exposure and mortality in a large prospective study. *Am J Respir Crit Care Med* 193(10): 1134-1142.
- U.S. Bureau of the Census. 1997. *Statistical Abstract of the United States: 1997*. Washington, DC.
- U.S. Bureau of the Census. 2002a. Modified Race Data Summary File 2000 Technical Documentation. Census of Population and Housing: Washington DC. <http://www.census.gov/popest/archives/files/MRSF-01-US1.pdf>.
- U.S. Bureau of the Census. 2002b. *Statistical Abstract of the United States: 2001*. Washington DC.
- US Bureau of Labor Statistics. 2015. Current Population Survey. 2015 Annual Social and Economic Supplement. Available at: <https://www.bls.gov/opub/reports/womens-databook/2016/home.htm>.
- U.S. Department of Education. 1996. The Condition of Education 1996, Indicator 42: Student Absenteeism and Tardiness. National Center for Education Statistics. Washington DC.
- U.S. EPA. 1997. The Benefits and Costs of the Clean Air Act: 1970 to 1990. U.S. EPA, Office of Air and Radiation, Office of Policy, Planning and Evaluation. Washington, DC. EPA 410- R-97-002. October. <http://www.epa.gov/airprogm/oar/sect812/index.html>.
- U.S. EPA. 1999a. The Benefits and Costs of the Clean Air Act: 1990 to 2010: EPA Report to Congress. U.S. EPA, Office of Air and Radiation, Office of Policy. Washington, DC. EPA 410-R-99-001. November. <http://www.epa.gov/airprogm/oar/sect812/index.html>.
- U.S. EPA. 1999b. OAQPS Economic Analysis Resource Document. Prepared by U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Durham, NC. April. <http://www.epa.gov/ttn/ecas/analguid.html>.
- U.S. EPA. 2000. Guidelines for Preparing Economic Analyses. Office of the Administrator. Washington, DC. EPA #: 240-R-00-003. September.

[http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/Guidelines.html/\\$file/Guidelines.pdf](http://yosemite.epa.gov/ee/epa/eed.nsf/webpages/Guidelines.html/$file/Guidelines.pdf)

- U.S. EPA. 2004. Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engine. U.S. EPA, Office of Transportation and Air Quality. Washington, DC. EPA 420-R-04-007. April.
- U.S. EPA. 2006. Final Regulatory Impact Analysis: PM_{2.5} NAAQS. Office of Air and Radiation, Office of Air Quality Planning and Standards. Research Triangle Park, NC. <http://www.epa.gov/ttn/ecas/ria.html>.
- U.S. EPA. 2008a. Final Ozone NAAQS Regulatory Impact Analysis. Prepared by Office of Air and Radiation, Office of Air Quality Planning and Standards. Research Triangle Park, NC. <http://www.epa.gov/ttn/ecas/ria.html>.
- U.S. EPA. 2008b. Regulatory Impact Analysis: Control of Emissions of Air Pollution from Locomotive Engines and Marine Compression Ignition Engines Less than 30 Liters Per Cylinder. Office of Transportation and Air Quality, Assessment and Standards Division. Washington, DC. EPA420-R-08-001. March. <http://www.epa.gov/otaq/locomotv.htm>.
- U.S. EPA. 2012. Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter. EPA-452/R-12-003. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. December. Available at <http://www.epa.gov/pm/2012/finalria.pdf>.
- U.S. EPA Science Advisory Board (U.S. EPA-SAB). 2004. Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act, 1990–2020. EPA-SAB-COUNCIL-ADV-04-002. March. Available at [http://yosemite.epa.gov/sab%5CSABPRODUCT.NSF/08E1155AD24F871C85256E5400433D5D/\\$File/council_adv_04002.pdf](http://yosemite.epa.gov/sab%5CSABPRODUCT.NSF/08E1155AD24F871C85256E5400433D5D/$File/council_adv_04002.pdf).
- U.S. EPA. 2017. Trichloroethylene (TCE); Regulation of Use in Vapor Degreasing Under TSCA Section 6(a). 40 CFR 751. Washington, DC. Available at: <https://www.federalregister.gov/documents/2017/01/19/2017-01229/trichloroethylene-tce-regulation-of-use-in-vapor-degreasing-under-tsca-section-6a>.
- Villeneuve, P.J., Chen, L., Rowe, B.H., Coates, F. 2007. Outdoor air pollution and emergency department visits for asthma among children and adults: A case-crossover study in northern Alberta, Canada. *Environmental Health*, 6(40).
- Viscusi, W. K. 1992. *Fatal Tradeoffs: Public and Private Responsibilities for Risk*. Oxford University Press: New York.

- Viscusi, W. K. and J. E. Aldy. 2003. *The Value of a Statistical Life: A Critical Review of Market Estimates throughout the World*. AEI-Brookings Joint Center for Regulatory Studies. Washington, DC. January.
- Viscusi, W. K., W. A. Magat and J. Huber. 1991. Pricing Environmental Health Risks - Survey Assessments of Risk - Risk and Risk - Dollar Trade-Offs For Chronic Bronchitis. *Journal of Environmental Economics and Management*. Vol. 21 (1): 32-51.
- Wei, W., Di, Q., Choirat, C., Wang, Y., Koutrakis, P., Zanobetti, A., Dominici, F., Schwartz, J.D. 2019. Short term exposure to fine particulate matter and hospital admission risk and costs in the Medicare population: time stratified, case crossover study. *BMJ (Online)*, 367, l6258–l6258. <https://doi.org/10.1136/bmj.l6258>
- Wilson, A. M., C. P. Wake, T. Kelly and J. C. Salloway. 2005. Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study. *Environ Res*. Vol. 97 (3): 312-21.
- 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 asthma call-back survey—United States, 2006–2008. *J Asthma* 49(1): 16-22.
- Wittels, E. H., J. W. Hay and A. M. Gotto, Jr. 1990. Medical costs of coronary artery disease in the United States. *Am J Cardiol*. Vol. 65 (7): 432-40.
- Woodruff, T.J., Darrow, L.A., & Parker, J.D. 2008. Air Pollution and Postneonatal Infant Mortality in the United States, 1999-2002. *Environ Health Perspect* 116(1): 110-115.
- Woodruff, T. J., J. Grillo and K. C. Schoendorf. 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. *Environmental Health Perspectives*. Vol. 105 (6): 608-612.
- Woodruff, T. J., J. D. Parker and K. C. Schoendorf. 2006. Fine particulate matter (PM_{2.5}) air pollution and selected causes of postneonatal infant mortality in California. *Environmental Health Perspectives*. Vol. 114: 786-790.
- Woods & Poole Economics Inc. 2012. *Complete Demographic Database*. Washington, DC. <http://www.woodsandpoole.com/index.php>.
- Wu, X., Braun, D., Schwartz, J., Kioumourtzoglou, M.A., Dominici, F., 2020. Evaluating the impact of long-term exposure to fine particulate matter on mortality among the elderly. *Science Advances* 6(29), <https://doi.org/10.1126/sciadv.aba5692>.
- Yang, W, Hamilton, JL, Kopil, C, Beck, JC, Tanner, CM, Albin, RL, Ray Dorsey, E, Dahodwala, N, Cintina, I, Hogan, P and Thompson, T. 2020. Current and projected future economic burden of Parkinson's disease in the U.S. *NPJ Parkinsons Dis* 6: 15.

- Yao, J., Ghosh, K., Perrailon, M.C., Cutler, D.M, Fang, M.C. 2019. Trends and Racial Differences in First Hospitalization for Stroke and 30-Day Mortality in the US Medicare Population From 1988 to 2013. *Medical Care* 57(4): 262-269.
- Zanobetti, A., M. Franklin, et al. 2009. Fine particulate air pollution and its components in association with cause-specific emergency admissions. *Environmental Health* 8: 58-60.
- Zanobetti, A, Dominici, F, Wang, Y and Schwartz, JD. 2014. A national case-crossover analysis of the short-term effect of PM2.5 on hospitalizations and mortality in subjects with diabetes and neurological disorders. *Environ Health* 13(1): 38.
- Zanobetti A. and Schwartz, J. 2006. Air pollution and emergency admissions in Boston, MA. *J Epidemiol Community Health* 60 (10): 890-5.
- Zanobetti, A. and Schwartz, J., 2008. Mortality displacement in the association of ozone with mortality: an analysis of 48 cities in the United States. *American Journal of Respiratory and Critical Care Medicine* 177(2): 184-189.
- Zanobetti, A. and Schwartz, J. 2008b. Is there adaptation in the ozone mortality relationship: A multi-city case-crossover analysis. *Environmental Health*, 7(22).