Environmental Benefits Mapping and Analysis Program – Community Edition

User's Manual

Appendices

March 2015

Prepared for
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, NC
Neal Fann, Project Manager

Prepared by
RTI International

Table of Contents

Appendix A: Monitor Rollback Algorithms

This Appendix details the rollback procedures that you can perform on monitor data. The rollback procedure is a quick way to determine the monitor levels that would exist under various kinds of changes that you can specify. This includes three basic types of rollbacks: 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 a set of states with an associated set of rollback parameter values. Three rollback **types** are available:

- Percentage Rollback. Monitor values are reduced the same percentage.
- Incremental Rollback. Monitor values are reduced 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

Incremental Rollback

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, they 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_{10}, $PM_{2.5}$) 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 **Interday 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 four supported rollback methods for Interday Rollbacks - Percentage, Incremental, Peak Shaving, and Quadratic. 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. Quadratic rollback is more complicated than these first three, and has its own section.

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 nth 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 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 - 30)

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	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.3.1.3 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 three supported rollback methods for Intraday Rollback - Percentage, Incremental, and Quadratic. 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. Quadratic rollback is more complicated than these first two, and has its own section.

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 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

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 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 15 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.

A.3.4 Interday and Intraday Rollback - Quadratic

Quadratic rollback is based on an algorithm developed by Horst and Duff . The idea behind quadratic rollback is to reduce large values proportionally more than small values while just achieving the standard - that is, the out-of-attainment value should be more or less at the standard after the rollback (some small amount of error is involved).

The original quadratic rollback algorithm is designed to roll back hourly observations given a desired peak value. That is, it assumes that the Attainment Test metric is the one-hour average and the Attainment Test ordinality is one. As such, the algorithm was modified slightly to allow for ordinalities other than one to be used.

The basic formula for quadratic rollback is:

Reduced Observation = [1 - (A + B × Initial Observation)] × Initial Observation

where:

i ranges over the days being reduced.

A = 1- V

V = Min(1, Vi)

Vi = (2 × Maximum Observation Value × Standard) / Xi

Xi = (2 × Maximum Observation Value × Metricsi) - Metricsi2

B = Max(0, [(V × Out of Attainment Value - Standard) / Out of Attainment Value2])

A.3.4.1 Quadratic Rollback - Interday

Because Quadratic Rollback was originally designed to adjust hourly observations to meet a daily metric standard, it is slightly complicated to use it to generate target metric values.

First, Quadratic Rollback calculates the anthropogenic out of attainment value by subtracting the Intraday Background Level from the out of attainment value. Note that this differs from the other interday rollback methods, which subtract the Interday Background Level from the out of attainment value. Similarly, the anthropogenic standard is calculated by subtracting the Intraday Background Level from the standard.

The anthropogenic observations and non-anthropogenic observations are then calculated. For pollutants which have daily observations (PM_{10}, $PM_{2.5}$) the anthropogenic metric values are used (see above for their calculation). For pollutants which have hourly observations (Ozone), Quadratic Rollback loops through each metric value and calculates the twenty four corresponding anthropogenic observations and non-anthropogenic observations as follows:

IF the metric value is at or below the Interday Background Level,

For each observation,

non-anthropogenic observation = observation

anthropogenic observation = 0

ELSE

For each observation,

IF the observation is at or below the Intraday Background Level

non-anthropogenic observation = observation

anthropogenic observation = 0

ELSE

non-anthropogenic observation = Intraday Background Level

anthropogenic observation = observation - Intraday Background Level

A new set of anthropogenic metric values is then calculated by generating the Attainment Test metric from the anthropogenic observations. The Quadratic Rollback algorithm is then called, passing in the anthropogenic metric values as Metrics, anthropogenic observations as Observations, anthropogenic standard as Standard, and anthropogenic out of attainment value as Out of Attainment Value. The result is a set of reduced anthropogenic observations. These are then added together with the non-anthropogenic observations to give a final set of reduced observations.

Then, if Quadratic Rollback was also selected as the Intraday Rollback method, these observations are used as the final reduced observations for the monitor. Otherwise, metric targets are generated from these hourly observations, and the observations themselves are discarded.

A.3.4.2 Quadratic Rollback - Intraday

Quadratic Rollback can also be used to adjust hourly observations to meet metric targets generated via a different rollback method. In this case, the algorithm is used to adjust each set of twenty four hourly observations to meet the corresponding metric target. Intraday Quadratic Rollback uses the normal set of anthropogenic observations as Observations, a single normal anthropogenic metric value as Metrics, and the normal anthropogenic metric target as Standard. Intraday Quadratic Rollback tends to always slightly miss its metric target, so it is not run in an iterative fashion as the other Intraday Rollback Methods are (doing so would sometimes result in an infinite loop).

Appendix B: Air Pollution Exposure Estimation Algorithms

BenMAP has grouped individuals into what we refer to as "population grid-cells," where the grid-cells typically conform to some type of grid used in an air quality model, such as the REMSAD air quality model, or just the counties of the United States. For each type of grid, the population is built in each grid-cell by aggregating census block data. In the next step, BenMAP estimates the air pollution exposure for each grid-cell, with the assumption 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. In a third general approach, you may combine both modeling and monitoring data to estimate exposure.

When combining modeling and monitoring data, BenMAP scales or adjusts the monitoring data with modeling data. The advantage of modeling data is that they can provide predictions for years in which monitoring data are not available, as well as to provide predictions in areas of the country for which monitoring data are not available. And the advantage of monitor data is that they are based on actual observations. Combining both sources of information, allows BenMAP to make more informed predictions.

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. To account for missing days, BenMAP represents the distribution of daily metrics with a certain number points or "bins," where each bin represents a certain range of the distribution, with the underlying assumption that missing days have the same distribution as the available data. For example, for analyses of the United States the Environmental Protection Agency has typically used 153 bins to represent the ozone season from May through September, and for particulate matter they have used 365 bins to represent the year. In addition to being able to account for incomplete or missing data, and using bins to represent the distribution provides a uniform approach that allows for easy comparison of different monitors.

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

Measurement Frequency	Metric Name	Metric Description
Daily (e.g., $PM_{2.5}$, PM_{10})	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 then represents the distribution of daily metrics with the number of days specified for each pollutant. By calculating bins with the available days, BenMAP assumes that the distribution of missing days is similar to the distribution of available data.

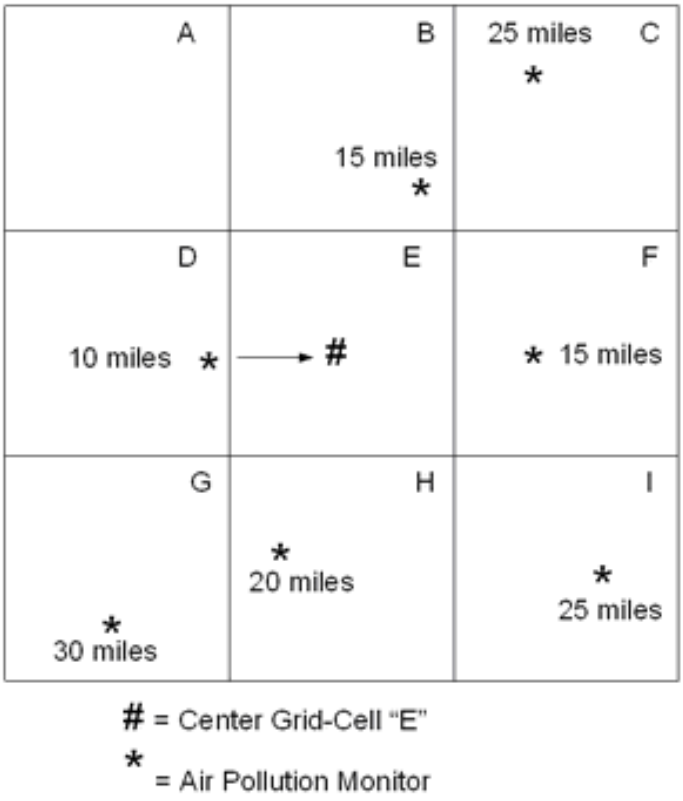
B.2 Closest Monitor

When using the closet 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, and then calculates the bins that represent the distribution of the daily metrics. The annual metrics and the binned daily metrics are then used in the calculation of 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).

To capture some of the information generated by air pollution models, BenMAP can also scale the data from the closest monitor with air pollution modeling data. BenMAP includes two types of scaling - "temporal" and "spatial" scaling. We discuss each below.



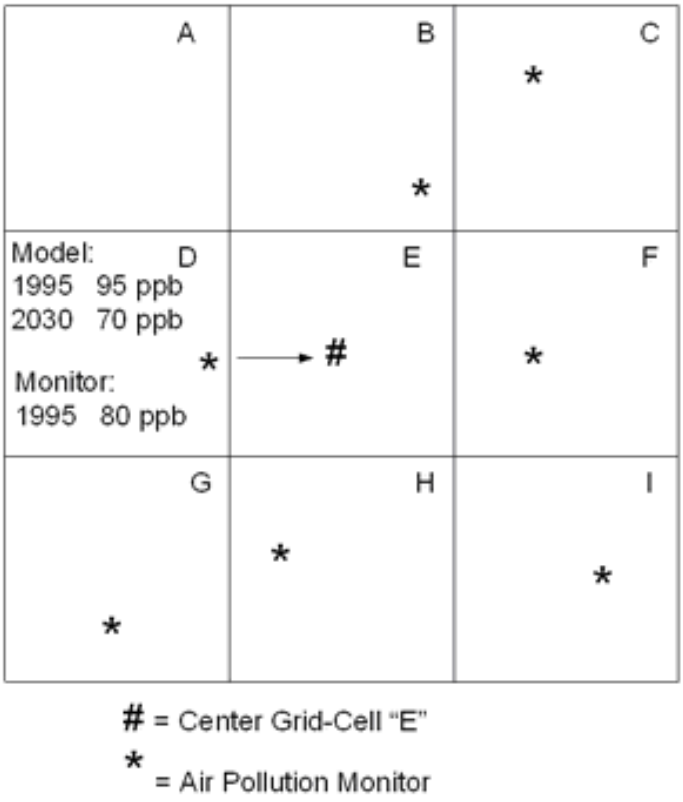
B.2.1 Closest Monitor - Temporal Scaling

With temporal scaling, BenMAP scales monitoring data with the ratio of the future-year to base-year modeling data, where the modeling data is from the modeling grid-cell containing the monitor. In the case of pollutants typically measured hourly, such as ozone, BenMAP scales the hourly monitor values, calculates the annual and daily metrics of interest, and then bins the daily metrics. In the case of pollutants typically measured daily, BenMAP scales the daily values, calculates the annual metrics of interest, and then bins the daily metric.

Consider the case in the figure below. To forecast air pollution levels for 2030, BenMAP would multiply the 1995 monitor value (80 ppb) by the ratio of the 2030 model value (70 ppb) to the 1995 model value (95 ppb):

Forecast2030 = Monitor Value 1995 × (Model Value D, 2030 / Model Value D, 1995)

Forecast2030 = 80 ppb × (70 ppb / 95 ppb) = 58.9 ppb.



In this example, we have examined the adjustment of a single monitor value with the ratio of single model values. The approach is essentially the same when there are multiple monitor values and multiple model values.

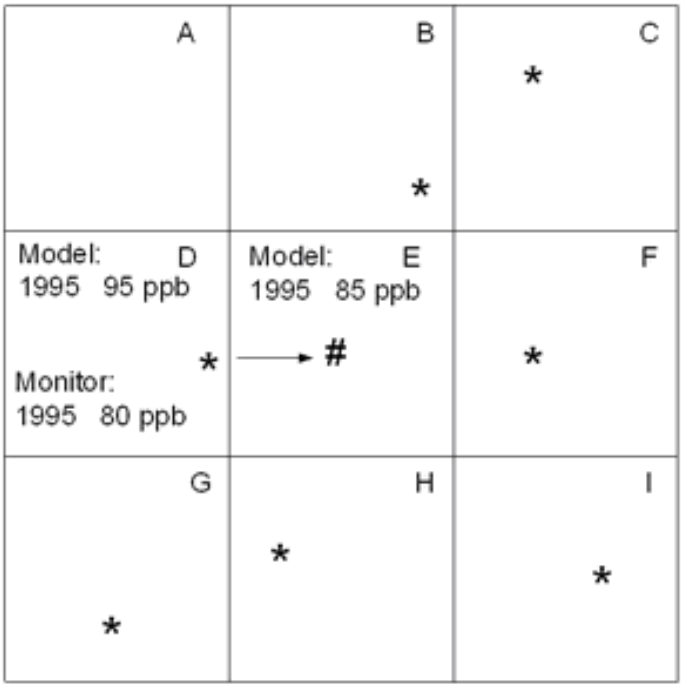
B.2.2 Closest Monitor - Spatial Scaling

With spatial scaling, we are estimating a monitor value for the center of each population grid-cell. We start by choosing the closest monitor to the center of each population grid-cell, and then we scale this closest monitor with modeling data. In particular, BenMAP multiplies the monitoring data with the ratio of the base-year modeling data for the destination grid-cell to the base-year modeling data for grid-cell containing the monitor. The spatial scaling occurs in the same fashion as with temporal scaling. In the case of pollutants typically measured hourly, such as ozone, BenMAP scales the hourly monitor values, calculates the annual and daily metrics of interest, and then bins the daily metrics. In the case of pollutants typically measured daily, BenMAP scales the daily values, calculates the annual metrics of interest, and then bins the daily metric.

To estimate air pollution levels for 1995 in grid-cell "E" below, BenMAP would multiply the 1995 closest monitor value (80 ppb) by the ratio of the 1995 model value for grid-cell "E" (70 ppb) to the 1995 model value for grid-cell "D" (95 ppb):

Forecast1995 = Monitor Value1995 × (Model Value E, 1995 / Model Value D, 1995)

Forecast1995 = 80 ppb × (85 ppb / 95 ppb) = 71.6 ppb.



B.2.3 Closest Monitor - Temporal and Spatial Scaling

Combining both temporal and spatial scaling, BenMAP first multiplies monitoring data with both the ratio of the future-year to base-year modeling data, where the modeling data is from the modeling grid-cell containing the monitor. This gives a temporary forecast for 2030. BenMAP then multiplies this temporary forecast with the ratio of the future-year modeling data for the destination grid-cell to the future-year modeling data for grid-cell containing the monitor. As seen below, this simplifies to multiplying monitoring data with both the ratio of future-year modeling data from the destination grid-cell to the base-year modeling data from the grid-cell containing the monitor. Again, as described for temporal and spatial scaling, BenMAP first scales the hourly and daily values, generates the metrics of interest and then bins the daily metrics.

To forecast air pollution levels for 2030 in the figure below, BenMAP would multiply the 1995 monitor value (80 ppb) by the ratio of the 2030 model value (70 ppb) to the 1995 model value (95 ppb):

Temporary Forecast 2030 = Monitor Value 1995 × (Model Value D, 2030 / Model Value D, 1995)

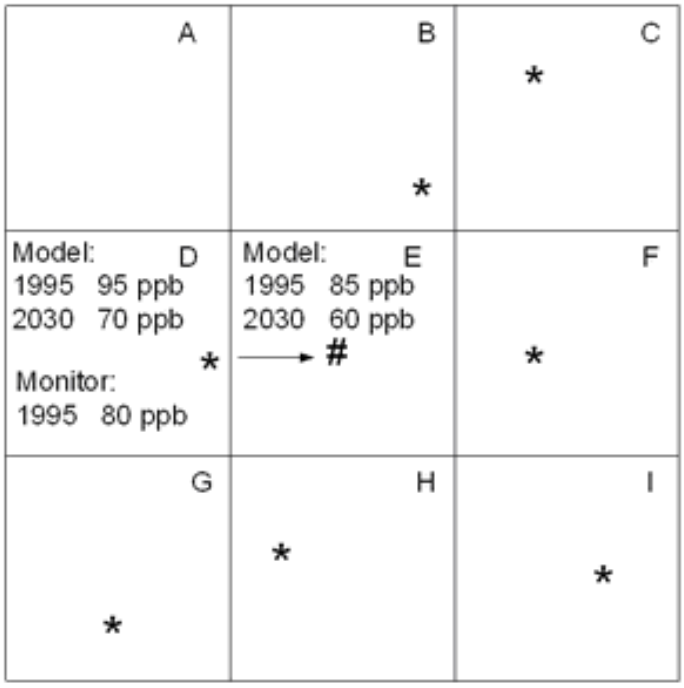
Temporary Forecast 2030 = 80 ppb × (70 ppb / 95 ppb) = 58.9 ppb.

Forecast 2030 = Temporary Forecast 2030 × (Model Value E, 2030 / Model Value D, 2030)

Forecast 2030 = 58.9 ppb × (60 ppb / 70 ppb) = 50.5 ppb.

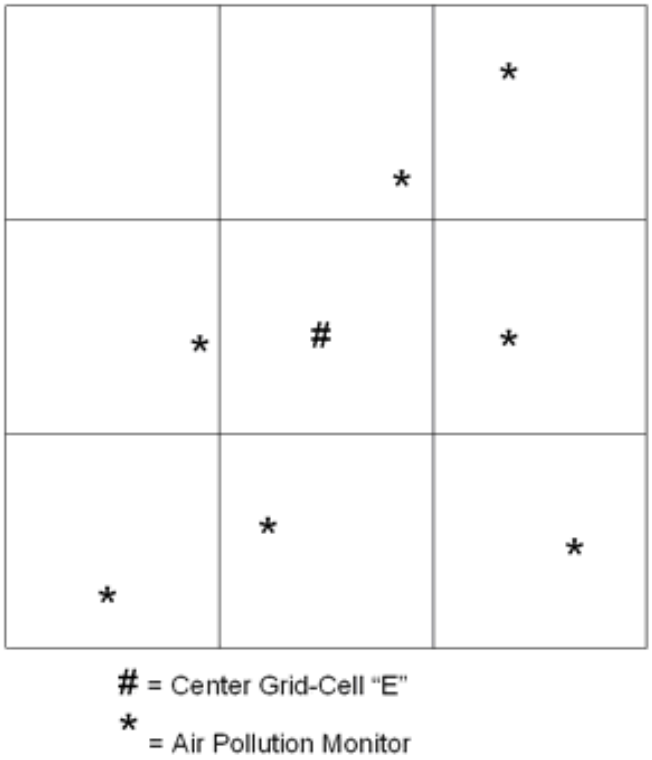
Note that through cancellation, this equation simplifies to:

Forecast 2030 = Monitor Value 1995 × (Model Value E, 2030 / Model Value D, 1995)

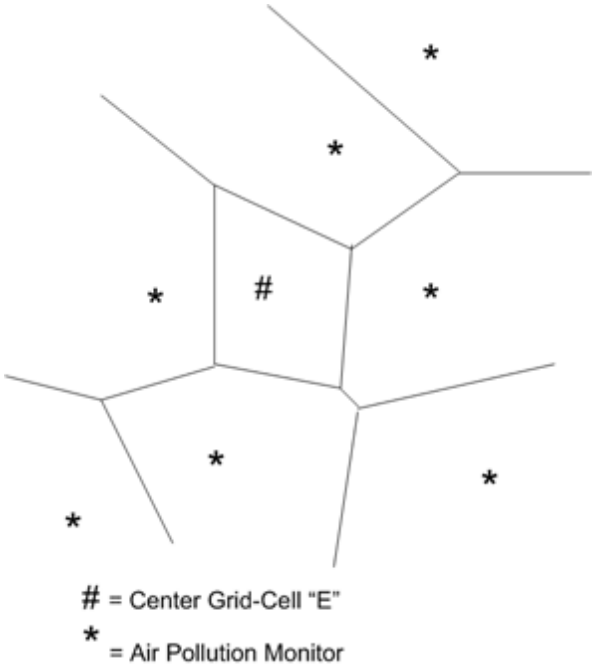


B.3 Voronoi Neighbor Averaging (VNA)

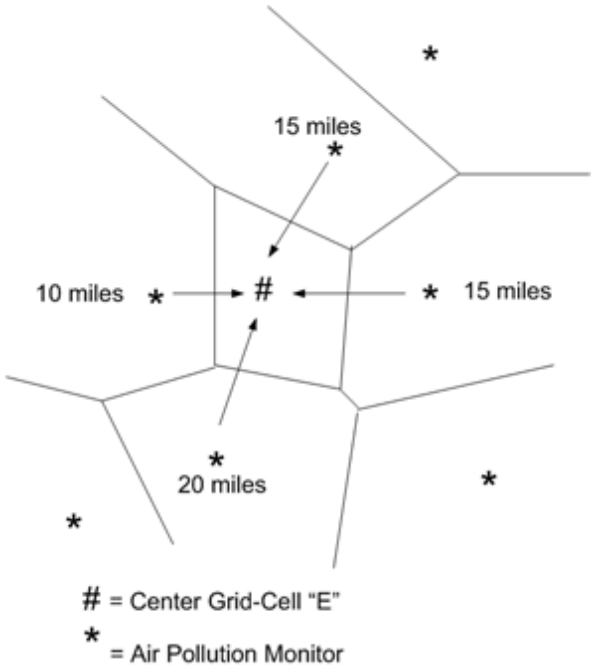
Like the closest monitor approach, the Voronoi Neighbor Averaging (VNA) algorithm uses monitor data directly or in combination with modeling data. However, instead of using the single closest monitor to estimate exposure at a population grid-cell, the VNA algorithm interpolates air quality at every population 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.



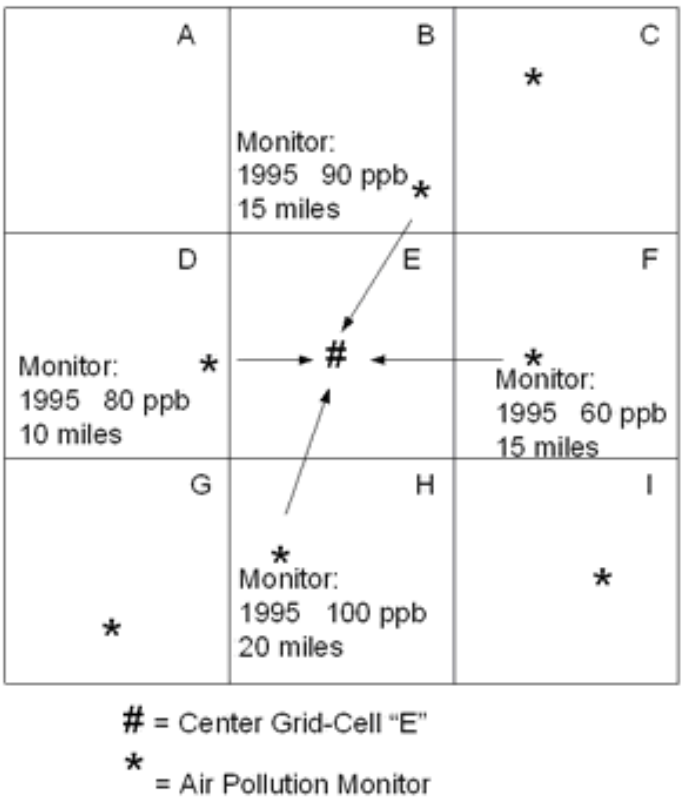
To estimate the air pollution level in each grid-cell, BenMAP calculates the metrics for each of the neighboring monitors, and then calculates an inverse-distance weighted average of the metrics. The further the monitor is from the BenMAP grid-cell, the smaller the weight.

In the figure below, the weight for the monitor 10 miles from the center of grid-cell E is calculated as follows:

$$weight_{10miles} = \frac{\frac{1}{10}}{\left(\frac{1}{10}+\frac{1}{15}+\frac{1}{15}+\frac{1}{20}\right)} = 0.35$$

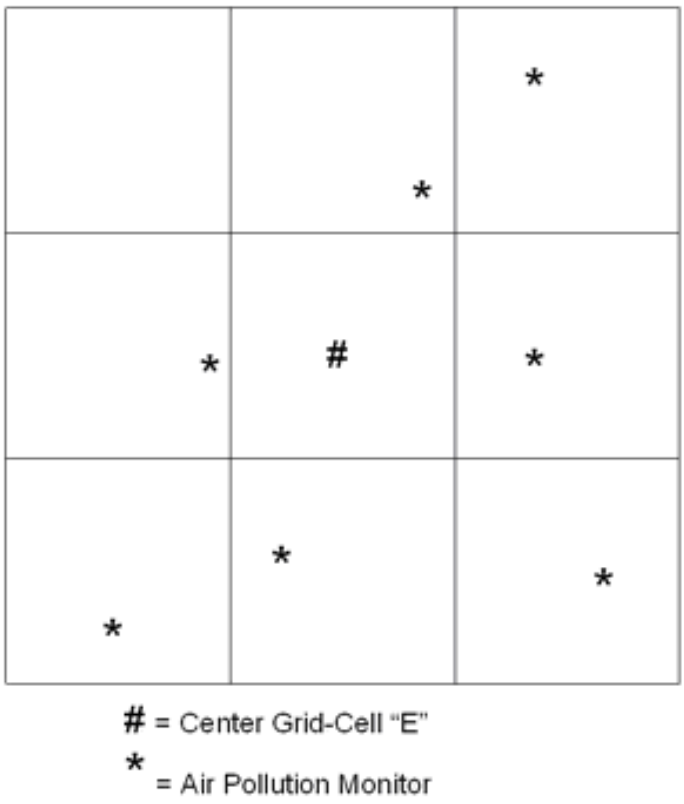
The weights for the other monitors would be calculated in a similar fashion. BenMAP would then calculate an inverse-distance weighted average for 1995 air pollution levels in grid-cell E as follows:

Forecast 1995 = 0.35×80 ppb + 0.24×90 ppb+ 0.24×60 ppb + 0.18×100 ppb = 81.2 ppb.

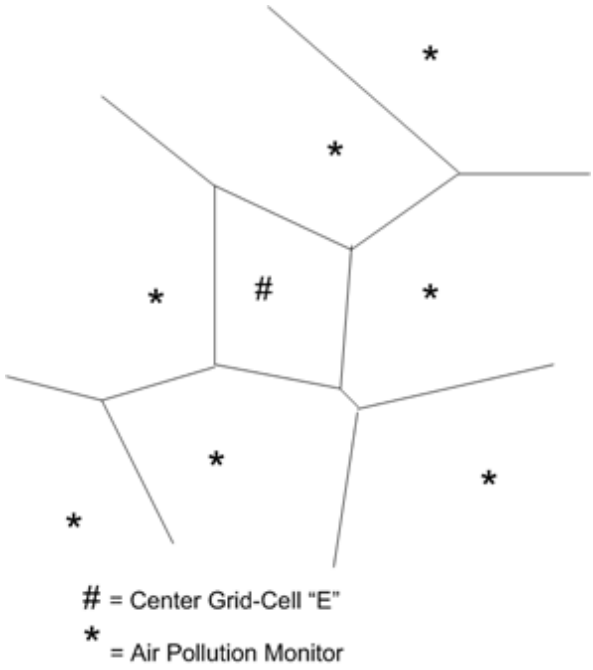


B.3.1 VNA / Temporal Scaling

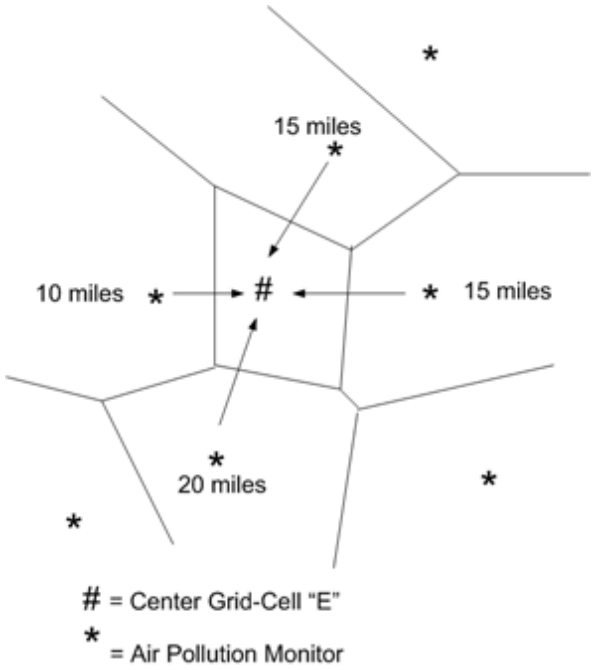
Like the closest monitor approach, the Voronoi Neighbor Averaging (VNA) algorithm uses monitor data directly or in combination with modeling data. However, instead of using the single closest monitor to estimate exposure at a population grid-cell, the VNA algorithm interpolates air quality at every population 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.



We then choose those monitors that share a boundary with the center of grid-cell "E." These are the nearest neighbors, we use 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 annual and the binned daily metrics for each of the neighboring monitors, and then calculates an

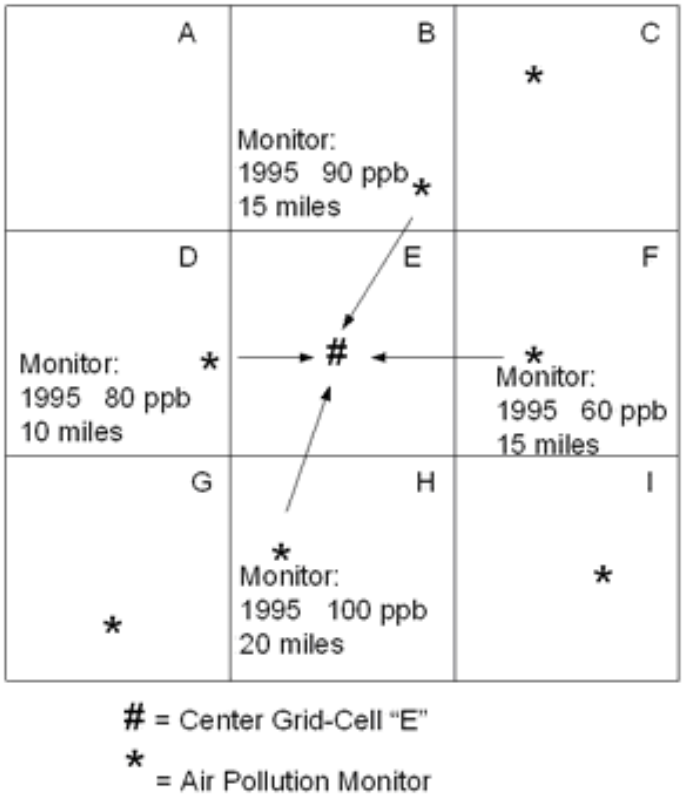
inverse-distance weighted average of the metrics. The further the monitor is from the BenMAP grid-cell, the smaller the weight.

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 = 0.35×80 ppb + 0.24×90 ppb+ 0.24×60 ppb + 0.18×100 ppb = 81.2 ppb .



Note that BenMAP is calculating an inverse-distance weighted average of the annual metrics and the binned daily metrics. Alternatively, BenMAP could calculate an inverse-distance weighted average of the hourly and daily observations, calculated the annual and daily metrics, and then binned the daily metrics.

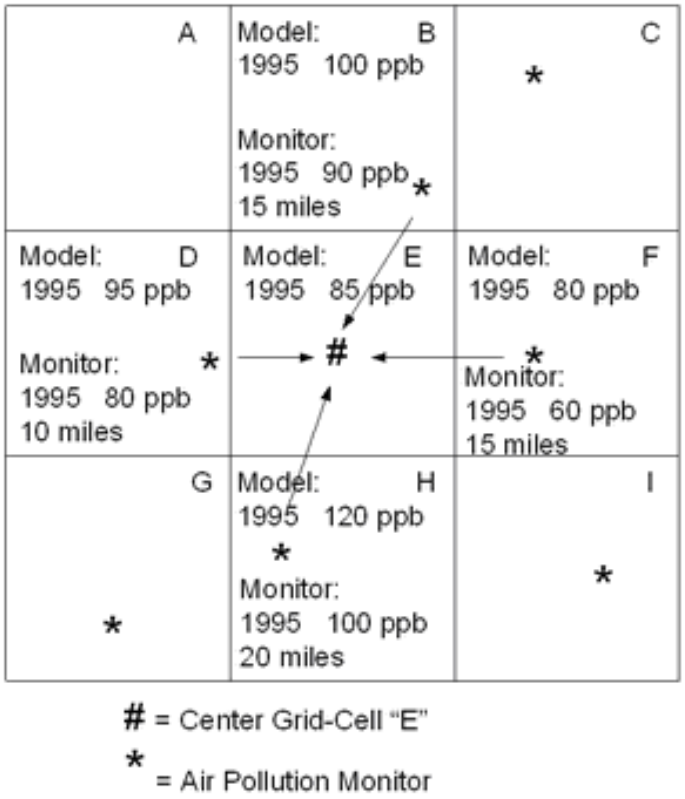
B.3.2 Voronoi Neighbor Averaging (VNA) - Spatial Scaling

BenMAP can also combine VNA with spatial scaling. For each of the neighbor monitors, BenMAP multiplies the monitoring data with the ratio of the base-year modeling data for the destination grid-cell to the base-year modeling data for grid-cell containing the monitor. The spatial scaling occurs in the same fashion as with temporal scaling. In the case of pollutants typically measured hourly, such as ozone, BenMAP scales the hourly monitor values, calculates the annual and daily metrics of interest, and then bins the daily metrics. In the case of pollutants typically measured daily, BenMAP scales the daily values, calculates the annual metrics of interest, and then bins the daily metric.

Consider the example in the figure below. To forecast air pollution levels for 1995, BenMAP would multiply the 1995 monitor value by the ratio of the 1995 model value (of the grid cell destination) to the 1995 model value (of the neighbor grid cell containing the monitor):

$$Forecast_{1995} = \sum_{i=1}^{4} Weight_i \times Monitor_i \times \frac{Model_{E,1995}}{Model_{i,1995}}$$

$$Forecast_{1995} = \left(0.35 \times 80 \times \frac{85}{95}\right) + \left(0.24 \times 90 \times \frac{85}{100}\right) + \left(0.24 \times 60 \times \frac{85}{80}\right) + \left(0.18 \times 100 \times \frac{85}{120}\right) = 70.8\,ppb$$



B.3.3 Voronoi Neighbor Averaging (VNA) - Temporal & Spatial Scaling

Combining both temporal and spatial scaling, BenMAP multiplies monitoring data with the ratio of the future-year to base-year modeling data, where the future-year modeling data are from the destination grid-cell and the base-year modeling data are from the grid-cell containing the monitor. One the hourly and daily monitoring data are scaled, BenMAP generates the metrics of interest, bins the daily metrics, and then uses the metrics to estimate adverse health effects in the population grid-cell.

The figure below gives an example of combining temporal and spatial scaling.

$$Forecast_{2030} = \sum_{i=1}^{4} Weight_i \times Monitor_i \times \frac{Model_{E,2030}}{Model_{i,1995}}$$

$$Forecast_{2030} = \left(0.35 \times 80 \times \frac{60}{95}\right) + \left(0.24 \times 90 \times \frac{60}{100}\right) + \left(0.24 \times 60 \times \frac{60}{80}\right) + \left(0.18 \times 100 \times \frac{60}{120}\right) = 50.0\,ppb$$

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 Temporal and Spatial Scaling Adjustment Factors

As presented in the preceding examples of temporal and spatial scaling, the closest monitor, VNA, and fixed radius approaches can use model data to scale monitor observations. In the examples above, we scaled single monitor values with the ratio of single model values. In fact, however, the scaling involves multiple monitor values and multiple model values.

To proceed with the scaling, BenMAP takes the modeling values and splits them into groups, depending on how the pollutant is defined. (See the section on defining pollutants in the Loading Data chapter.) The United States setup has defined ozone to have a default of 10 adjustment factors for the ozone season, where the first group represents the first 10 percent of the model observations; the second group represents the observations between the 10th and 20th percentile; and so on through the tenth group, which represents the observations between the 90th and 100th percentiles. BenMAP then averages the values in each group. The United States setup has defined particulate matter to have five adjustment factors for each of the four seasons in the year, where the first group in each season represents the first 20 percent of the model

observations; the second group represents the observations between the 20th and 40th percentiles; and so on. Then, as for ozone model values, BenMAP averages the particulate matter model values in each group.

BenMAP treats the monitor values in a similar way. It sorts the monitor values from low to high, and divides them into the same number groups as there are scaling factors.

B.5.1 Calculation of Scaling Factors

In developing scaling factors for the standard United States setup, BenMAP sorts the modeling data into either 10 groups or 20 groups, depending on the pollutant (e.g., 10 for ozone and 20 for particulate matter, 5 for each of the 4 seasons). Given the number of groups, BenMAP then determines how to assign the model values. In determining to which group a value belongs, BenMAP assigns a two-digit "percentile" to each value. With values in a given grid-cell sorted from low to high, the percentile for each value will equal: (the observation rank number minus 0.5) divided by (the total number of values) multiplied by (100). If there are 250 hourly values, the first hourly value will have a percentile = (1-0.5)/(250) × (100) = 0.20%; the 27th value will have a percentile = (27-0.5)/(250) × (100) = 10.60%; and so on.

Each data group is represented by "group-lo" and "group-hi" values. These are the minimum and the maximum percentiles in each group, where group-lo equals: (group rank minus 1) multiplied by (100) divided by (the number of groups); and group-hi equals: (group rank) multiplied by (100) divided by (the number of groups) minus 0.001. If there are ten groups: the first group will have: group-lo = (1-1)/100×10 = 0.000%, and group-hi = (1/100×10)-0.001 = 9.999% ; the second group will have: group-lo = (2-1)/100×10 = 10.000%, and group-hi = (2/100×10)-0.001 = 19.999% ; and so on to the tenth group, which will have: group-lo = (10-1)/100×10 = 90.000%, and group-hi = (10/100×10)-0.001 = 99.999%. BenMAP assigns each observation to a particular group with the following algorithm: if "group-lo" <"percentile" < "group-hi", then assign the observation to that data group.

Below we give some examples of the calculations that BenMAP performs when scaling.

B.5.1.1 Example: $PM_{2.5}$ Scaling Factors in U.S. Setup

After preparing the $PM_{2.5}$ model and monitor data, BenMAP calculates the following:

$$adjusted\ monitor_{i,j,future} = monitor_{i,j,base} \times \frac{REMSAD_{j,k,future}}{REMSAD_{j,l,base}}$$

Where:

adjusted monitor = predicted daily $PM_{2.5}$ level, after adjustment by model data ($\mu g/m^3$)

monitor = observed daily $PM_{2.5}$ monitor level ($\mu g/m^3$)

i = day identifier

j = model season/quintile group (1 to 20)

k = grid cell identifier for population grid cell

l = grid cell identifier for grid cell containing monitor

base = base-year (e.g., 2000)

future = future-year (e.g., 2020)

REMSAD = representative model season/quintile value ($\mu g/m^3$)

After adjusting the monitor values to reflect air quality modeling, BenMAP calculates for each monitor the $PM_{2.5}$ metrics needed to estimate adverse health effects. In the case of VNA, BenMAP then calculates a weighted average (e.g., inverse-distance weighted average) of the neighbors identified for each population grid cell:

$$population\ grid\ cell_{future} = \sum_{m=1}^{n} adjusted\ monitor_{m,\,future} \times weight_{m}$$

Where:

population grid cell = inverse distance-weighted $PM_{2.5}$ metric at population grid cell ($\mu g/m^3$)

adjusted monitor = predicted $PM_{2.5}$ metric, after adjustment by model data ($\mu g/m^3$)

m = monitor identifier

base = base-year (e.g., 2000)

future = future-year (e.g., 2020)

weight = inverse-distance weight for monitor

After generating the bins for both the baseline and control scenarios, BenMAP uses these to calculate the change in air quality needed in most health impact functions to calculate the change in adverse health effects. To calculate the change in air quality, BenMAP subtracts the baseline value in the first bin from the control value in the first bin, and so on for each of the bins created for the daily $PM_{2.5}$ average.

B.5.1.2 Example: Ozone Scaling in U.S. Setup

After preparing the ozone model and monitor data, BenMAP calculates the following:

$$adjusted\ monitor_{i,j,future} = monitor_{i,j,base} \times \frac{CAMX_{j,k,future}}{CAMX_{j,l,base}}$$

Where:

Adjusted monitor = predicted hourly ozone level, after adjustment by model data (ppb)

Monitor = observed hourly ozone monitor level (ppb)

i = hour identifier

j = model decile group (1 to 10)

l = grid cell identifier for grid cell containing monitor

base = base-year (e.g., 1996)

future = future-year (e.g., 2030)

CAMX = representative model decile value (ppb)

After adjusting the monitor values to reflect air quality modeling, BenMAP calculates for each monitor the ozone metrics needed to estimate adverse health effects. In the case of VNA, BenMAP then calculates a weighted average (e.g., inverse-distance weighted average) of the neighbors identified for each population grid cell:

$$population\ grid\ cell_{future} = \sum_{m=1}^{n} adjusted\ monitor_{m,future} \times weight_{m}$$

Where:

population grid cell = inverse distance-weighted ozone metric at population grid cell (ppb)

adjusted monitor = predicted ozone metric, after adjustment by model data (ppb)

m = monitor identifier

future = future-year (2020, 2030)

weight = inverse-distance weight for monitor

After generating the bins for both the baseline and control scenarios, BenMAP can use these to calculate the change in air quality needed in most C-R functions to calculate the change in adverse health effects. To calculate the change in air quality, BenMAP subtracts the baseline value in the first bin from the control value in the first bin, and so on for each of the bins created for the daily ozone metrics.

B.6 Binned Metrics

When estimating air pollution exposure, it will often happen that metrics are often not available for each day in the year. To remedy this, BenMAP calculates representative values or bins with the available daily metrics, under the assumption that the missing days have a similar distribution. Each bin represents a day. In the case where there are 365 bins, the set of bins represents the entire year.

When combining air pollution metrics from multiple monitors, BenMAP first calculates the bins for the daily metrics, and then combines the bins, such as with some form of VNA. Once BenMAP has calculated binned exposure measures for both a baseline and a control scenario, BenMAP then takes the difference between the two scenarios for each bin - taking the difference between the baseline value in the first bin and the control value in the first bin, and so on for each of the bins.

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, Δx = ΔPM - 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 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 may be estimated by regression techniques but is 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 the pollutant may be fine particulate matter ($PM_{2.5}$) in $\mu g/m^3$ per day, and the population response may be the number of premature deaths per 100,000 population per day. C-R functions are estimated in epidemiological studies. A functional form is chosen by the researcher, and the parameters of the function are estimated

using data on the pollutant (e.g., daily levels of $PM_{2.5}$) and the health response (e.g., daily mortality counts). There are several different functional forms, discussed below, that have been used for 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 μg/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	$y_0/(1-y_c)$
Control Pollutant Exposure	y_c	$1-y_c$		$y_c/(1-y_c)$

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

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

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:

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

This can be rearranged as follows:

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

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:

$$\Delta y = y0 - yc = \beta \times \left(PM\,0 - PMc\right) = \beta \times \Delta PM$$

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:

$$\beta_{lower\ bound} = \beta - \left(1.96 \times \sigma_\beta\right) = 0.0006 - \left(1.96 \times 0.0003\right) = 1.2 \times 10^{-5}$$
$$\beta_{lower\ bound} = \beta + \left(1.96 \times \sigma_\beta\right) = 0.0006 + \left(1.96 \times 0.0003\right) = 0.00119$$

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:

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

Or, equivalently,

$$In(y) = \alpha + \beta \times PM,$$

where the parameter B is the incidence rate of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and α = ln(B). Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is,

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

where Bo is the incidence of y when all covariates in the model are zero, and x1, ... , xn are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in y are calculated, and is therefore not important.

The relationship between ΔPM and Δy is:

$$\Delta y = y_0 - y_c = B\left(e^{\beta PM_0} - e^{\beta PM_c}\right)^1$$

This may be rewritten as:

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

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

The change in the incidence of adverse health effects can then be calculated by multiplying the change in the incidence rate, 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 (σβ) 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:

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

$$\beta_{upper\ bound} = \beta + \left(1.96 \times \sigma_\beta\right)$$

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:

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

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

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

The coefficients associated with the lower and upper bounds (e.g., the 2.5 and 97.5 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:

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

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

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

C.5 Logistical 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:

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

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

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

The odds of an occurrence is:

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

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

$$\Rightarrow \quad In(odds) = X \times \beta$$

The odds ratio for the control scenario ($odds_c$) versus the baseline ($odds_0$) is then:

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

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

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

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

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

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

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

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

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

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

When the coefficient (β) and its standard error (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:

$$\beta_{lower\ bound}=\beta-(1.96\times\sigma\beta)$$

$$\beta_{upper\ bound}=\beta-(1.96\times\sigma\beta)$$

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:

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

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

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:

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

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

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

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

The relative risk (RR) is simply:

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

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:

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

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

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

The odds ratio and the coefficient calculated from it are dependent on the baseline and control incidence rates. Unfortunately, it is not always clear what the baseline and control incidence rates should be. Abbey et al. (1995b, Table 2) reported that there are 117 new cases of chronic bronchitis out of a sample of 1,631, or a 7.17 percent rate. In addition, they reported the relative risk (RR = 1.81) for a new case of chronic bronchitis associated with an annual mean concentration "increment" of 45 μg/m^3 of $PM_{2.5}$ exposure.

Assuming that the baseline rate for chronic bronchitis (y0) should be 7.17 percent, the question becomes whether the "increment" of 45 μg/m^3 should be added to or subtracted from the existing $PM_{2.5}$ concentration. If added the control incidence rate (y_c) would be greater than the baseline rate (y_0), while subtraction would give a control rate less than the incidence rate. In effect, one might reasonably derive two estimates of the odds ratio:

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

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

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

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

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

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

Assuming that:

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

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

It is then possible to calculate the underlying coefficient:

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

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

Since this coefficient estimate is based on the assumption that

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

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

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

Using the formula for the change in the incidence rate and assuming a 10 μg/m^3 decline in $PM_{2.5}$, it is shown that this results in changes within the bounds suggested by the two estimates based on using the estimated odds ratios:

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

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

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

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 seems like 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:

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

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

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

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

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

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

The relative risk is often presented rather than the coefficient β, so it is necessary to estimate β in order to develop 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 the development of county mortality rates for years 2010 through 2050 for use in BenMAP. First, we describe the source of 2004-2006 individual-level mortality data and the calculation of county-level mortality rates. Then we describe how we use national-level Census mortality rate projections to develop county-level mortality rate projections for years 2010-2050.

D.1.1 Mortality Data for 2004-2006

We obtained individual-level mortality data from 2004-2006 for the whole United States from the Centers for Disease Control (CDC), National Center for Health Statistics (NCHS). The data were compressed into a CD-ROM, which contains death information for each decedent, including residence county FIPS, age at death, month of death, and underlying causes (ICD-10 codes).

Since the detailed mortality data obtained from CDC do not include population, we combined them with U.S. Census Bureau postcensal population estimates exported from BenMAP. We then generated age-, cause-, and county-specific mortality rates using the following formula:

$$R_{i,j,k} = \frac{D_{i,j,k}(2004) + D_{i,j,k}(2005) + D_{i,j,k}(2006)}{P_{i,k}(2004) + P_{i,k}(2005) + P_{i,k}(2006)}$$

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.

Following CDC Wonder (http://wonder.cdc.gov), we treated mortality rates as "unreliable" when the death count is less than 20. Among all the calculated age-, cause-, and county-specific mortality rates, there were about 67% "unreliable" rates. For each combination of age group and mortality cause, we used the following procedure to deal with the problem of "unreliable" rates:

- For a given state, we grouped the counties where the death count (i.e., the numerator on the right-hand side of the above equation) was less than 20 and summed those death counts across those counties. If the sum of deaths was greater than or equal to 20, we then summed the populations in those counties, and calculated a single rate for the "state collection of counties" by dividing the sum of deaths by the sum of populations in those counties. After this adjustment, there were 17% unreliable rates left. This rate was then applied to each of those 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.SS. 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." Even after this national adjustment, there were about 1% unreliable rates left. In these cases, we simply calculated a single rate for the "national collection of counties, even though it was "unreliable," and assigned it to those counties in the "national collection of counties."

Table D-2. National Mortality Rates (per 100 people per year) by Health Endpoint and Age Group

Mortaity 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	0.22981	0.02813	0.08869	0.10429	0.19248	0.43195	0.90832	2.12421	5.26957	13.97875
Mortality, Non-Accidental	A00-R99	0.19705	0.01138	0.02118	0.04298	0.12831	0.36278	0.85309	2.06079	5.13808	13.66477
Mortality, Respiratory	J00-J98	0.01570	0.00100	0.00126	0.00211	0.00560	0.01817	0.06366	0.22866	0.63404	1.51364
Mortality, Chronic Lung	J40-J47, J67	0.00076	0.00029	0.00040	0.00064	0.00200	0.00906	0.04086	0.15507	0.37626	0.62631
Mortality, Lung Cancer	C34	0.00001	0.00001	0.00006	0.00036	0.00516	0.02980	0.10372	0.25966	0.37795	0.30788
Mortality, Ischemic Heart Disease	I20-I25	0.00019	0.00006	0.00041	0.00262	0.01574	0.05952	0.15438	0.37559	1.03204	3.19986
Mortality, Cardio-Pulmonary	I00-I78, J10-J18, J40-J47, J67	0.01685	0.00193	0.00468	0.01171	0.04044	0.12568	0.31814	0.86408	2.52456	7.83928

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

D.1.2 Mortality Rate Projections 2010-2050

To estimate age- and county-specific mortality rates in years 2010 through 2050, we calculated 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 2004- 2006 data as described above. We used the following procedure:

- For each age group, we obtained the series of projected national mortality rates from 2005 to 2050 (see the 2005 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 = 2010, 2011, ..., 2050) to the 2005 rate. These ratios are shown for selected years in Table D-4.

- Finally, to estimate mortality rates in year Y (Y = 2010, 2010, ..., 2050) that are both age group-specific and county-specific, we multiplied the county- and age-group-specific mortality rates for 2004-2006 by the appropriate ratio calculated in the previous step. For example, to estimate the projected mortality rate in 2010 among ages 18-24 in Wayne County, MI, we multiplied the mortality rate for ages 18-24 in Wayne County in 2004-2006 by the ratio of Census Bureau

projected national mortality rate in 2010 for ages 18-24 to Census Bureau national mortality rate in 2005 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 2004-2006	0.230*	0.028	0.089	0.104	0.192	0.432	0.908	2.124	5.270	13.979
Census Bureau 2005**	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 Population Projections of the United States: 1999 to 2100, Working Paper #38," which is available on http://www.census.gov/population/www/documentation/twps0038/twps0038.html

Table D-4. Ratio of Future Year All-Cause Mortality Rate to 2005 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+
2010	0.95	0.93	0.95	0.96	0.94	0.95	0.96	0.93	0.95	0.96
2015	0.90	0.86	0.90	0.93	0.89	0.90	0.91	0.88	0.89	0.94
2020	0.85	0.81	0.86	0.90	0.83	0.85	0.87	0.85	0.83	0.91
2025	0.78	0.76	0.81	0.85	0.79	0.79	0.82	0.81	0.78	0.87
2030	0.71	0.70	0.76	0.79	0.74	0.74	0.77	0.78	0.76	0.82
2035	0.66	0.65	0.71	0.74	0.70	0.70	0.72	0.74	0.72	0.77
2040	0.60	0.60	0.67	0.70	0.65	0.66	0.67	0.70	0.70	0.74
2045	0.55	0.56	0.63	0.66	0.61	0.62	0.64	0.66	0.67	0.73
2050	0.51	0.52	0.59	0.62	0.57	0.57	0.60	0.63	0.63	0.71

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:

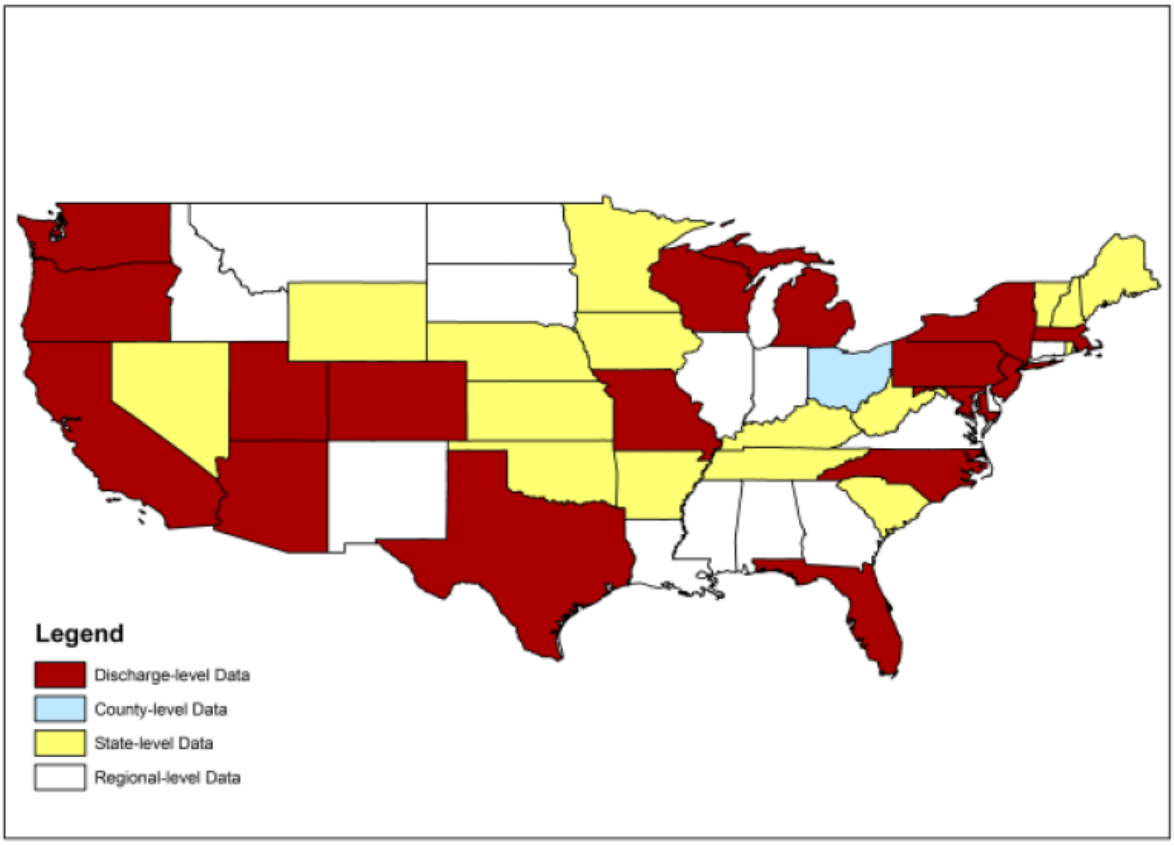
- The HCUP Central Distributor: Many of the HCUP databases are available for purchase through the HCUP Central Distributor. The databases include detailed information for individual discharges, such as primary diagnosis (in ICD-9 codes), patient's age and residence county.

- HCUP State Partners: Some HCUP participating states do not release their data to the Central Distributor; however, the data may be obtained through contacting the State Partners. Some State Partners (e.g., CA, TX, PA, MO, and NY) provided discharge-level data; others (e.g., OH) provided summarized data.

- HCUPnet: This is a free, on-line query system based on data from HCUP. It provides access to summary statistics at the state, regional and national levels.

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

Figure D-1. Hospitalization Data from HCUP



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

The procedures for calculating hospitalization rates are summarized as follows:

- For states with discharge-level data:
  - We calculated age-, health endpoint-, and county-specific hospitalization counts. Ohio was the only state that, while not providing discharge-level data, did provide county-level data for each age group-endpoint combination.
  - The above calculation excluded hospitalizations with missing patient age or county FIPS, which may lead to underestimation of rates. Therefore we scaled up the previously calculated age-, endpoint-, and county-specific counts using an adjustment factor obtained as follows:
    - We first counted the number of discharges for a specific endpoint in the state including those discharges with missing age or county FIPS.
    - We then counted the number of discharges for the endpoint in the state excluding those records with missing age or county FIPS.
    - The adjustment factor is the ratio of the two counts.
  - 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 (2006 or 2007). 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 ith endpoint in the jth region as "TOTALij")
  - For those states in the jth region that do have discharge-level or state-level data, we summed the hospital admissions by endpoint (we refer to this count for the ith endpoint in the jth region as "SUB ij").
  - We then estimated the hospitalization count for states without discharge or state data for the ith endpoint in the jth 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 National Hospital Discharge Survey (NHDS) and assumed the same distribution for the HCUP hospitalizations. We then applied this distribution to the estimated hospital counts (i.e., TOTALij - SUB ij) to obtain endpoint-, region-, and age-specific counts.

- Using the corresponding age- and region-specific populations, we calculated age-specific hospitalization rates for the ith endpoint in the jth 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-9410-414, 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)

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-5 presents a summary of the national hospitalization rates for 2007 from HCUP.

Table D-5. 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	2.872	0.426	0.205	0.231	0.408	0.791	1.313	2.972	4.970	8.045
Pneumonia	480-486	0.695	0.144	0.057	0.078	0.132	0.250	0.412	1.046	1.972	3.822
Chronic Lung Disease	490-496	0.350	0.158	0.057	0.064	0.135	0.299	0.503	1.028	1.502	1.506
Asthma	493	0.330	0.155	0.051	0.055	0.094	0.136	0.160	0.198	0.240	0.302
Cardiovascular											
All Cardiovascular	390-429	0.056	0.017	0.107	0.164	0.481	1.221	2.272	4.681	7.749	11.583
Acute Myocardial Infarction, Nonfatal	410	0.000	0.000	0.010	0.017	0.071	0.215	0.379	0.673	1.096	1.858
Ischemic Heart Disease	410-414	0.001	0.000	0.019	0.032	0.177	0.597	1.144	2.001	2.754	3.078
Dysrhythmia	427	0.017	0.006	0.022	0.031	0.071	0.166	0.345	0.870	1.608	2.216
Congestive Heart Failure	428	0.015	0.001	0.014	0.025	0.075	0.198	0.399	1.080	2.183	4.512
Stroke	431-437	0.007	0.003	0.014	0.020	0.071	0.204	0.415	1.043	1.860	2.953

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.

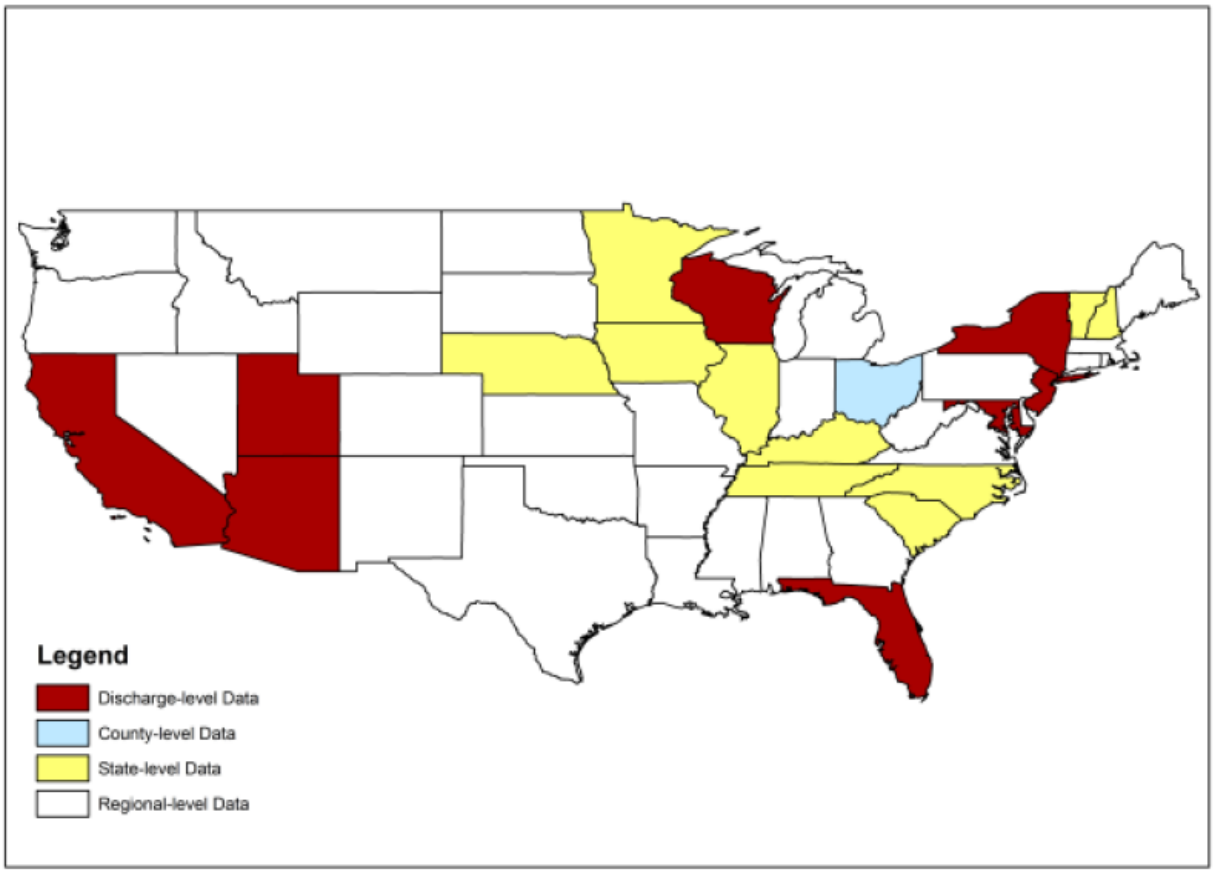
An alternative approach to the estimation of heart attack rates is to use data from the Healthcare Cost and Utilization Project (HCUP), assuming that all heart attacks that are not instantly fatal will result in a hospitalization. Details about HCUP data are described in Section D.2. According to the 2007 HCUP data there were approximately 624,936 hospitalizations due to heart attacks (acute myocardial infarction: ICD-9 410, primary diagnosis). We used rates based on HCUP data over estimates extrapolated from cohort studies because the former is a national database with a larger sample size, which is intended to provide reliable national estimates. The incidence rate calculation is also described in Section D.2 and the incidence rates for AMI hospitalization are presented in Table D-5.

Rosamond et al. (1999) reported that approximately six percent of male and eight percent of female hospitalized heart attack patients die within 28 days (either in or outside of the hospital). We, therefore, applied a factor of 0.93 to the estimated number of PM-related acute myocardial infarctions to exclude the number of cases that result in death within the first month. Note that we did not adjust for fatal AMIs in the incidence rate estimation, due to the way that the epidemiological studies are designed. Those studies consider total admissions for AMIs, which includes individuals living at the time the studies were conducted. Therefore, we use the definition of AMI that matches the definition in the epidemiological studies.

D.4 Emergency Department Visits

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

Figure D-2. Emergency Department Data from HCUP



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

- The SEDD databases include only those ER visits that ended with discharge. To identify the ER visits that ended in hospitalization, we used a variable called "admission source" in the SID databases. Admission source identified as "emergency room" indicates that the hospital admission came from the ER - i.e., the ER visit ended in hospitalization. For each combination of age group, 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 2005 to 2007; we assumed that ER visit rates are reasonably constant across these three years and consider them as 2007 rates.

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

Table D-6. Emergency Department Visit Rates (per 100 people per year) by Health Endpoint and Age Group

Emergency Department Category	ICD-9 Codes	Age 0-17	18-44	45-64	65-84	85+
Asthma	493	0.865	0.557	0.441	0.381	0.368
Respiratory	491-493, 460-466, 477.0-477.9, 480-486, 496, 786.07, 786.09	5.772	2.889	2.273	4.275	6.817
Cardiovascular	410-414, 427-428, 433-437, 440.0-440.9, 443-445, 451-453	0.029	0.302	1.695	5.950	12.512

D.5 School Loss Days

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

Table D-7 presents the estimated school loss day rates. Further detail is provided below on these rates. Table D-7. 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-8 presents a summary of these baseline rates.

Table D-8. 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. Bureau of the Census (1997, No.22)
	18-24		1.971	
	25-44		2.475	
	45-64		1.796	

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

D.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 bronchitis 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 = 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-9. The prevalence of asthma is summarized in Table D-10.

Table D-9. Asthma-Related Health Effects Incidence Rates

Endpoint	Age	Parameter	Rate	Source
Asthma Exacerbation, Shortness of Breath, African American	8-13	Incidence	13.51	Ostro et al. (2001, p. 202)
	8-13	Prevalence	7.40%	
Asthma Exacerbation, Wheeze, African American	8-13	Incidence	27.74	Ostro et al. (2001, p. 202)
	8-13	Prevalence	17.30%	
Asthma Exacerbation, Cough, African American	8-13	Incidence	24.46	Ostro et al. (2001, p. 202)
	8-13	Prevalence	14.50%	
Upper Respiratory Symptoms (URS)	9-11	Incidence	124.79	Pope et al. (1991, Table 2)

NOTE: The incidence rate is the number of asthma attacks per person per year. Prevalence refers to the fraction of people that have a particular illness during a particular time period.

D.7.1 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).

D.7.2 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.

D.7.3 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.

D.7.4 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.7.5 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-10 presents asthma prevalence rates used to define asthmatic populations in the health impact functions. Table D-10. Asthma Prevalence Rates Used to Estimate Asthmatic Populations

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

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

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

In this Appendix, we present the PM-related health impact functions in BenMAP. 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. And Appendix D presents a description of the sources for the incidence and prevalence data used in the health impact functions.

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 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. 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 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
Mortality, All Cause	Laden et al.	2006	6 cities	25-99	Annual	0.014842	0.004170	Log-linear	
Mortality, All Cause	Pope et al.	2002	51 cities	30-99	Annual	0.005827	0.002157	Log-linear	
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	
Mortality, All Cause	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.005827	0.000963	Log-linear	
Mortality, Lung Cancer	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.013103	0.003795	Log-linear	
Mortality, IHD	Krewski et al.	2009	116 U.S. cities	30-99	Annual	0.021511	0.002058	Log-linear	

E.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.

E.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;å θ**,min,max**) be a truncated continuous parametric (cumulative) distribution function with (vector of) parameters θ and truncation points **min** and **max**. The nth percentile point is defined as the value xn such that F(xn; θ**,min,max**)=n/100. Thus, if we know that the expert distribution's nth percentile point is xn and mth percentile point is xm then the following has to hold:

$$F(xn;\theta,\min,\max) = n/100$$
$$F(xm;\theta,\min,\max) = m/100$$

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 E-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 E-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		
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/m³	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/m³	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:

$$N(0.29; mean = ?, sd = ?, min = 0, max = 4) = 0.05$$
$$N(2.90; mean = ?, sd = ?, min = 0, max = 4) = 0.95$$

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/m^3 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/m^3 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 E-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 E-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

Expert	Mean	Standard Deviation	Min	P25	P50	P75	Max
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

E.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 μg/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 E-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 E-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	input	0.290	0.770		0.960	1.100	1.400	1.600	1.800

Expert	Information	Min	P5	P10	P25	P50	P75	P95	Max
µg/m^3	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 E-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 E-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
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

E.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/m^3 to 16 µg/m^3 and another log-linear function if the baseline value

falls within the range from >16 μg/m^3 to 30 μg/m^3. 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/m^3 to 10 μg/m^3, and a 14% probability that there is a causal relationship with a threshold somewhere between 0 μg/m^3 to 5 μg/m^3. Thus, the set of log-linear functions in BenMAP for expert K on the range from 4 μg/m^3 to 16 μg/m^3 contains:

- a function with $PM_{2.5}$ coefficient = 0 (no causality), which BenMAP selects with 65% probability;
- a function with the $PM_{2.5}$ coefficient expert K specified for the log-linear function on that range and no threshold, which BenMAP selects with 18% probability;
- a function with the $PM_{2.5}$ coefficient expert K specified for the log-linear function on that range and a threshold (with uniform probability) between 0 μg/m^3 to 5 μg/m^3, which BenMAP selects with 14% probability; and
- a function with the $PM_{2.5}$ coefficient expert K specified for the log-linear function on that range and a threshold (with uniform probability) between 5 μg/m^3 to 10 μg/m^3, which BenMAP selects with 4% probability.

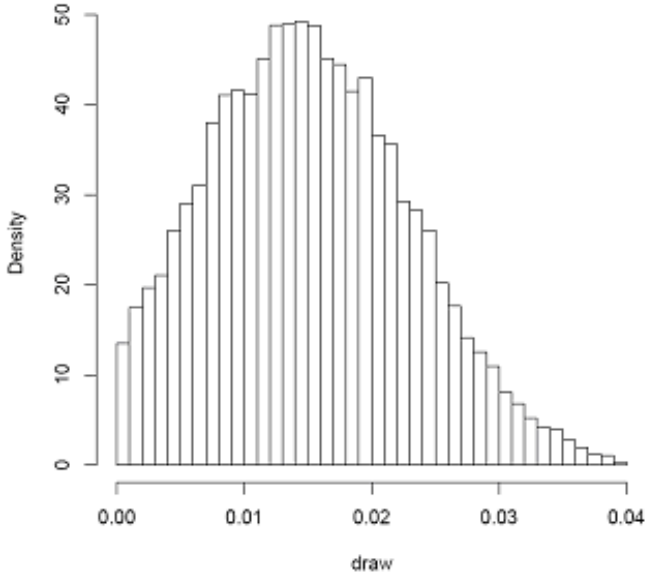
If the $PM_{2.5}$ baseline value is greater than 16 μg/m^3, BenMAP goes through an analogous procedure to select a function from among the two functions in that set.

E.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).

E.1.1.4.1 Expert A

Figure E-1. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert A

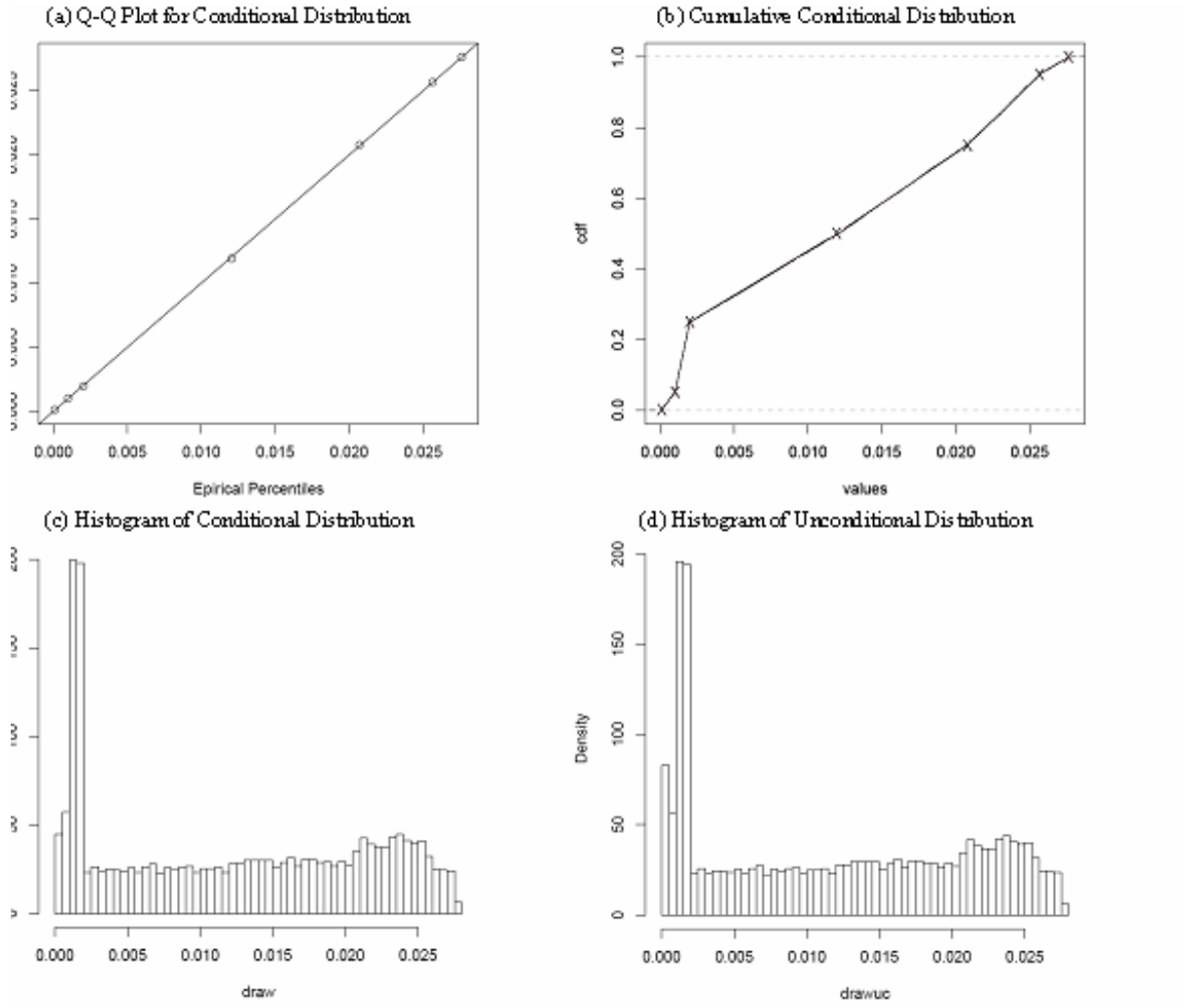


Notes:

- 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)).

E.1.1.4.2 Expert B

Figure E-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 µg/m³

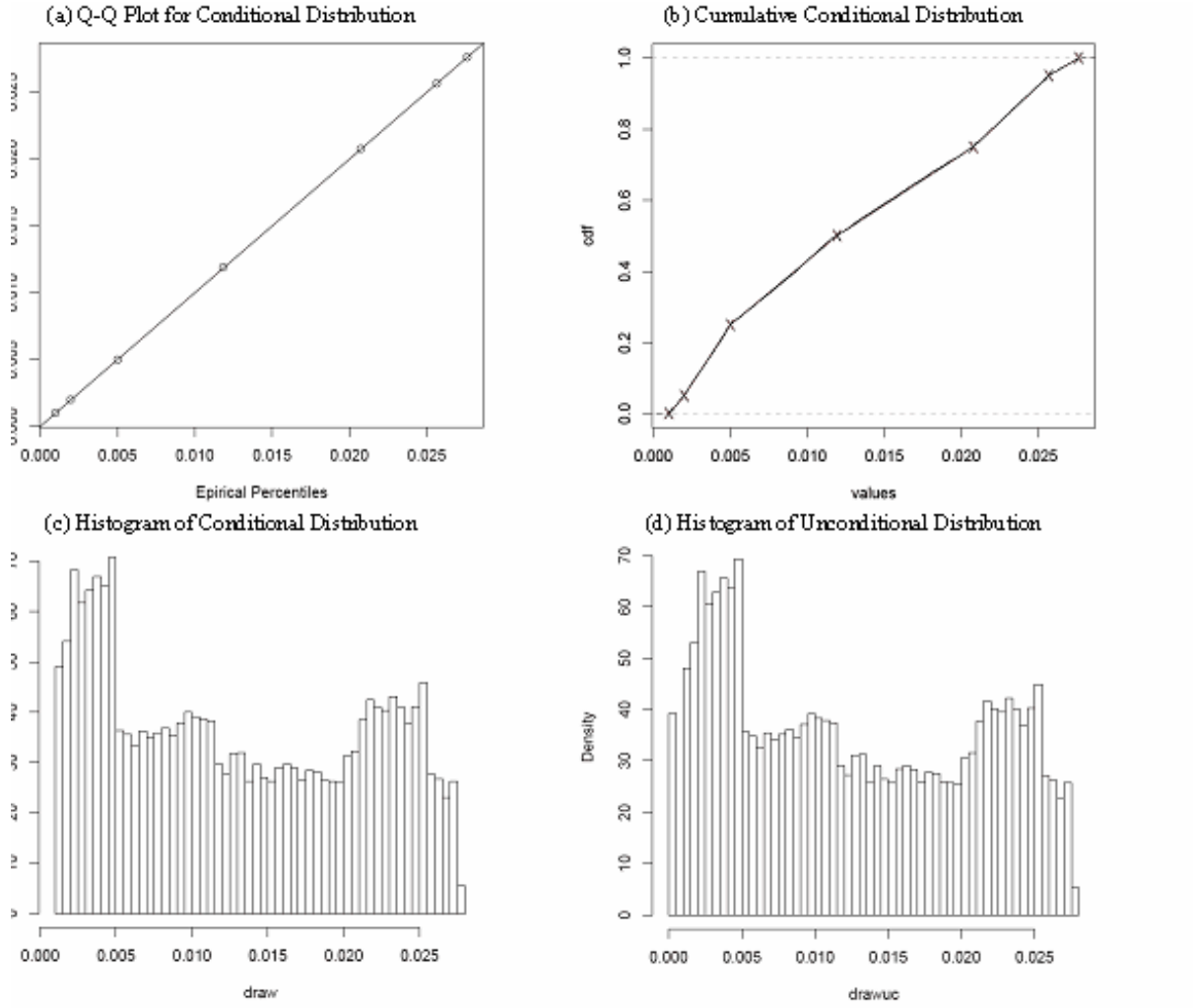


Notes:

- 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 E-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 µg/m³



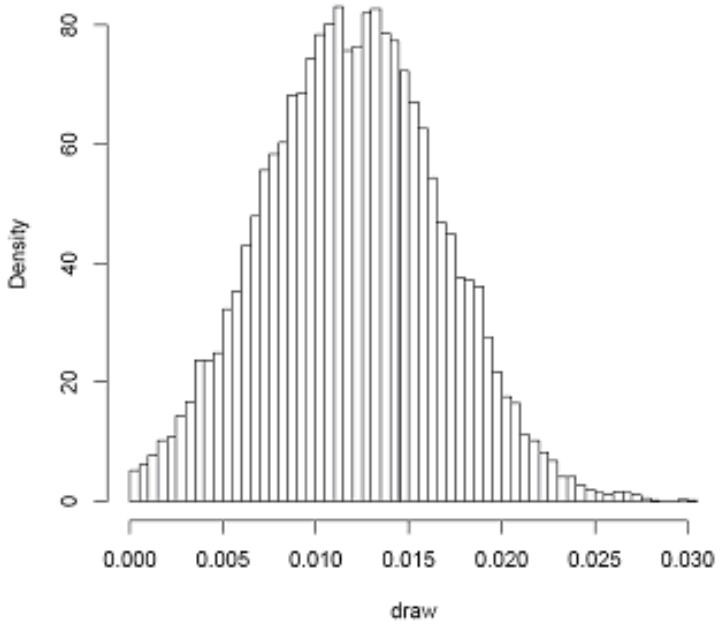
Notes:

- 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)).

E.1.1.4.3 Expert C

Figure E-3. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert C



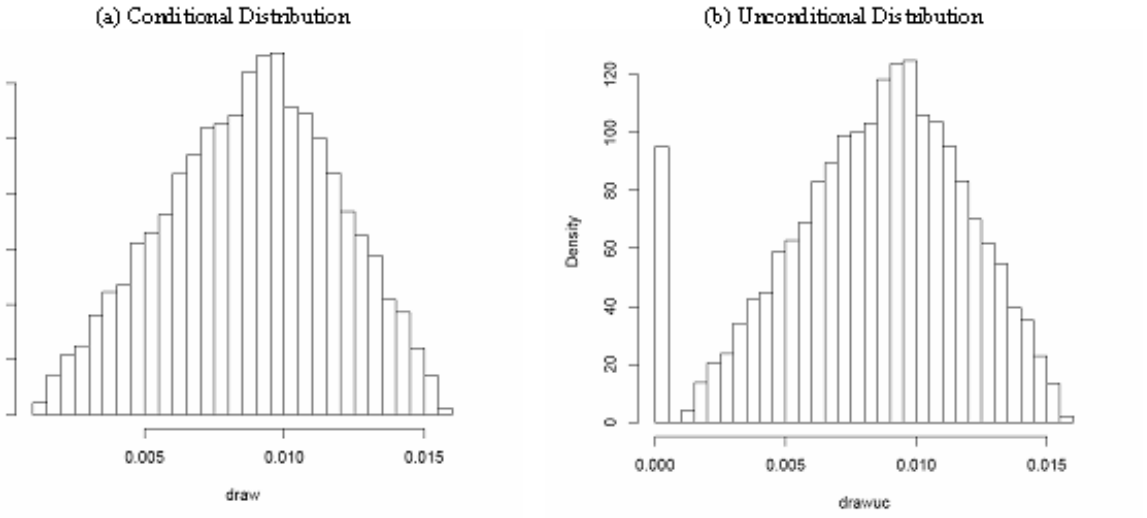
Notes:

- 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)).

E.1.1.4.4 Expert D

Figure E-4. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert D



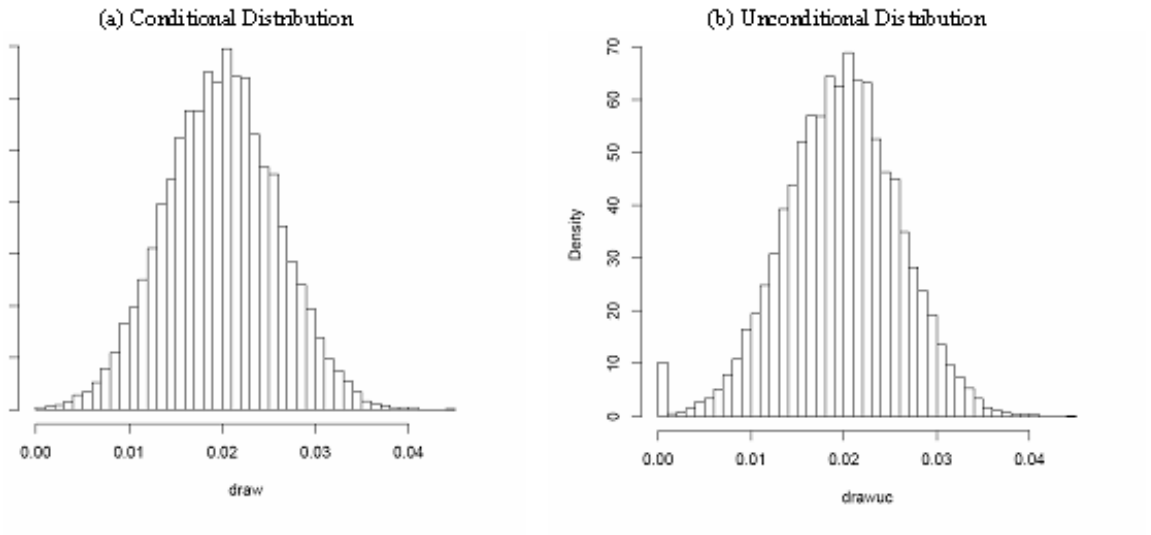
Notes:

- 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)).

E.1.1.4.5 Expert E

Figure E-5. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert E



Notes:

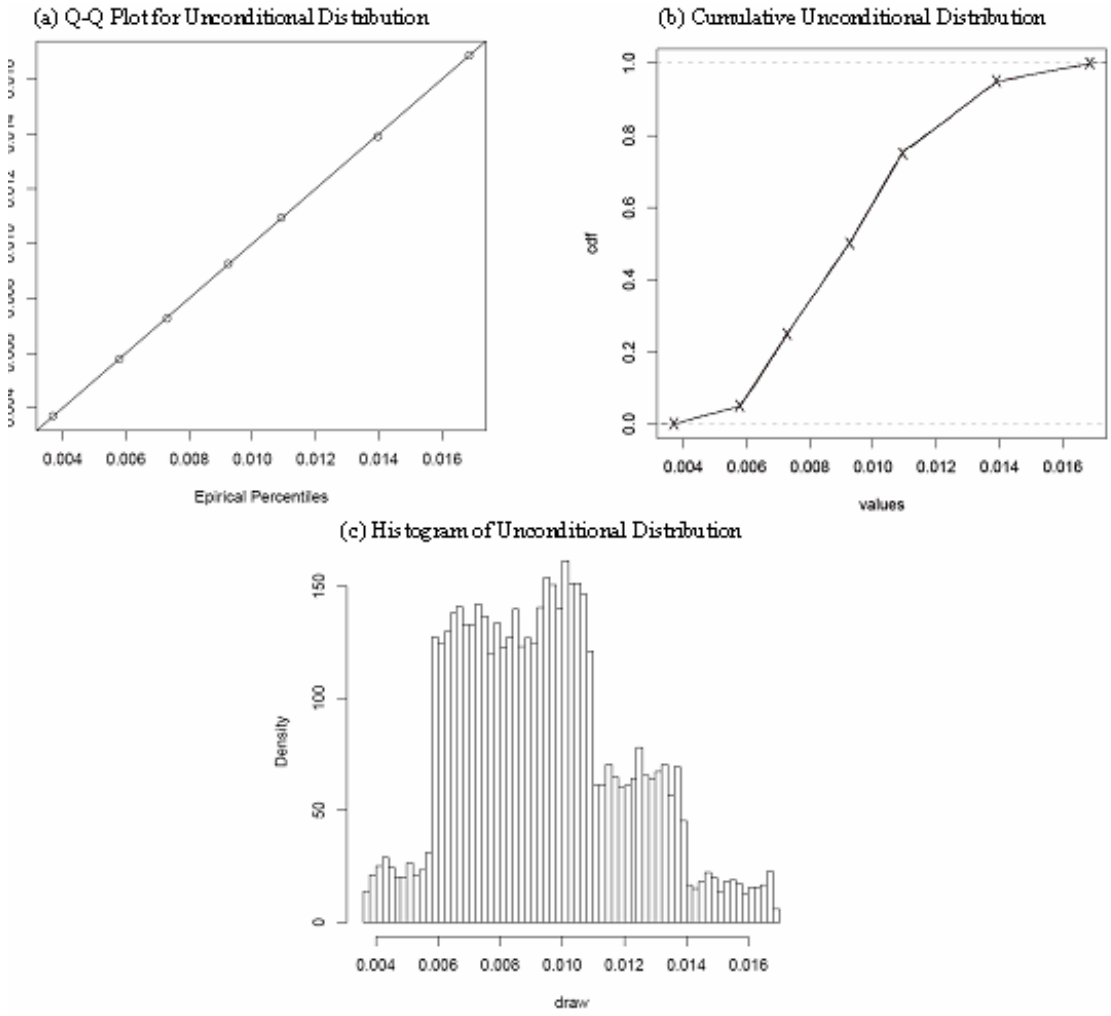
- Expert E specified a truncated Normal Distribution. We inferred the following parameters for this distribution: mean=2.00 and standard deviation=0.61. The distribution was conditional on causality. We created a corresponding unconditional distribution by adding extra 1 percent zeros to the draw.

- 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)).

E.1.1.4.6 Expert F

Figure E-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

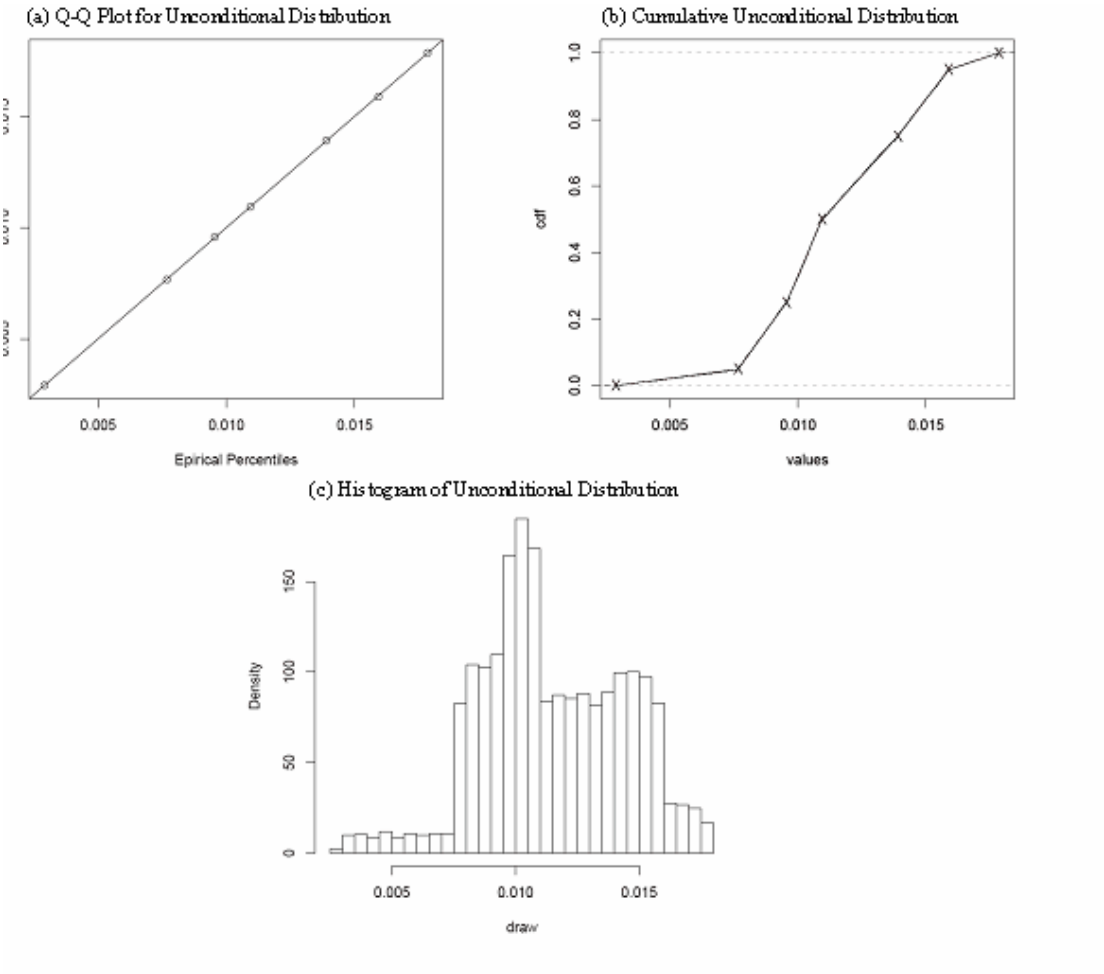


Notes:

- 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 E-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



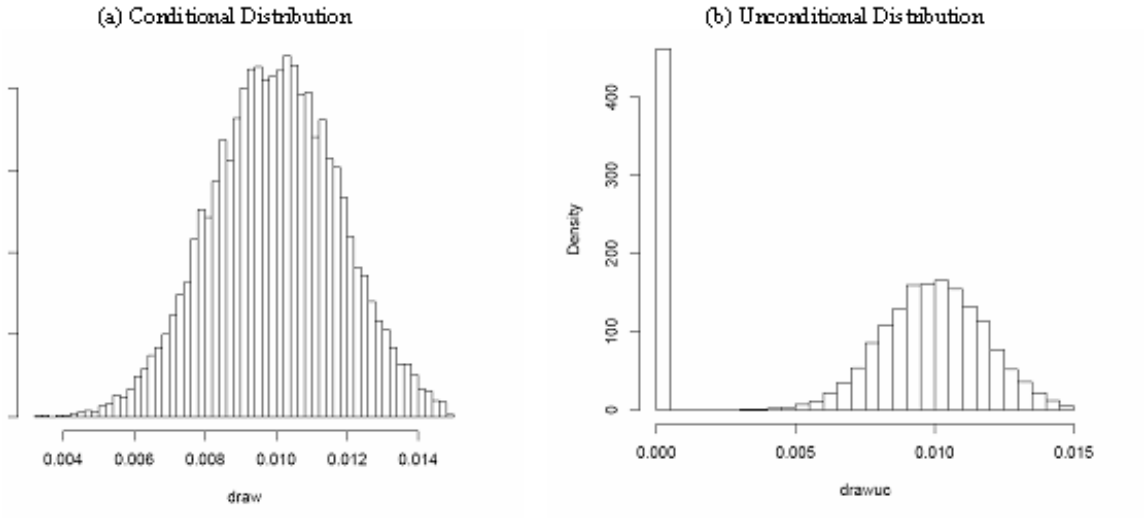
Notes:

- 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)).

E.1.1.4.7 Expert G

Figure E-7. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert G



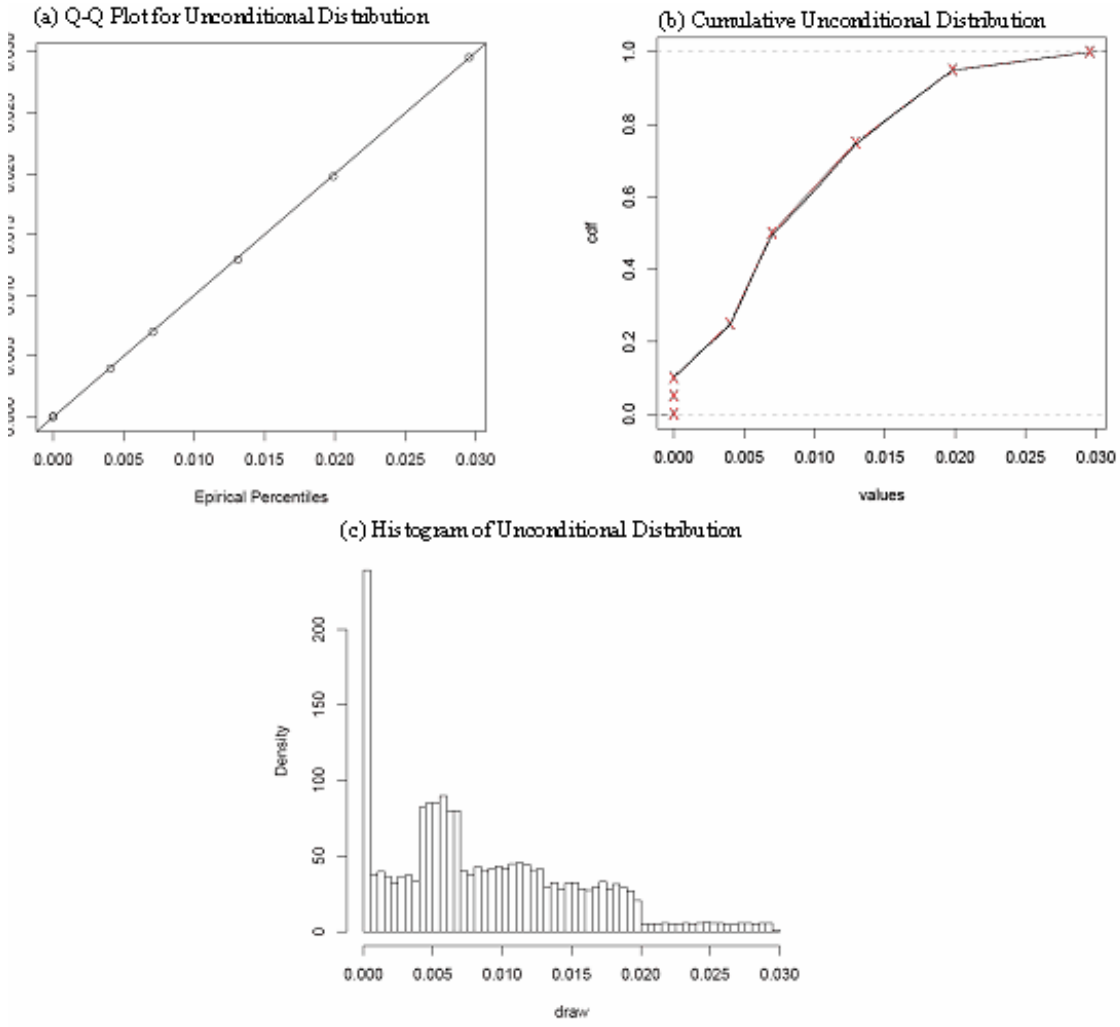
Notes:

- 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)).

E.1.1.4.8 Expert H

Figure E-8. Characteristics of the Random Draw from the Approximated Distribution of the $PM_{2.5}$ Effect Specified by Expert H



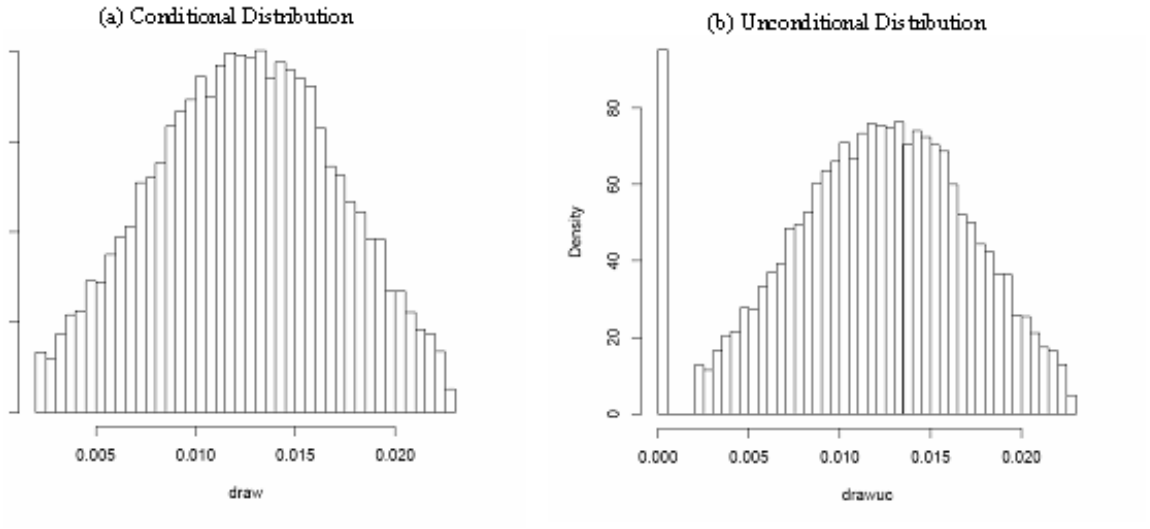
Notes:

- 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)).

E.1.1.4.9 Expert I

Figure E-9. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert I



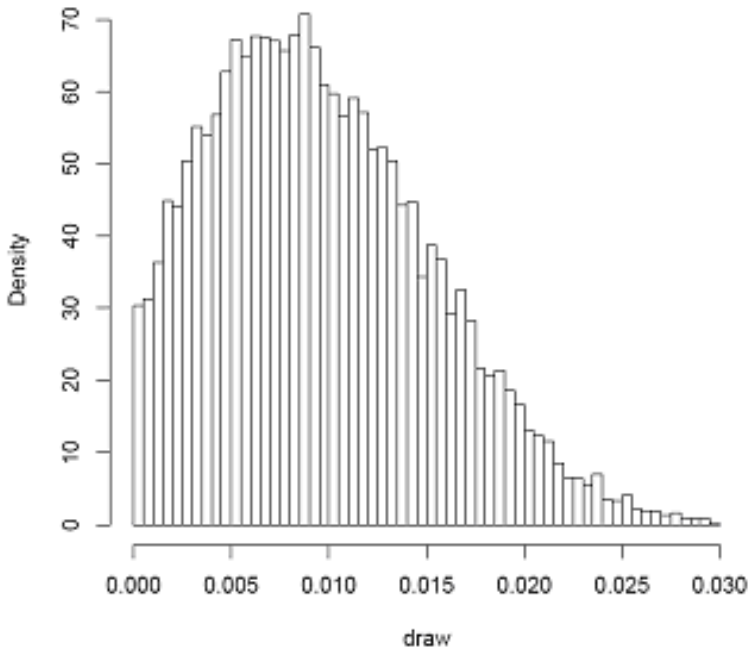
Notes:

- 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)).

E.1.1.4.10 Expert J

Figure E-10. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert J

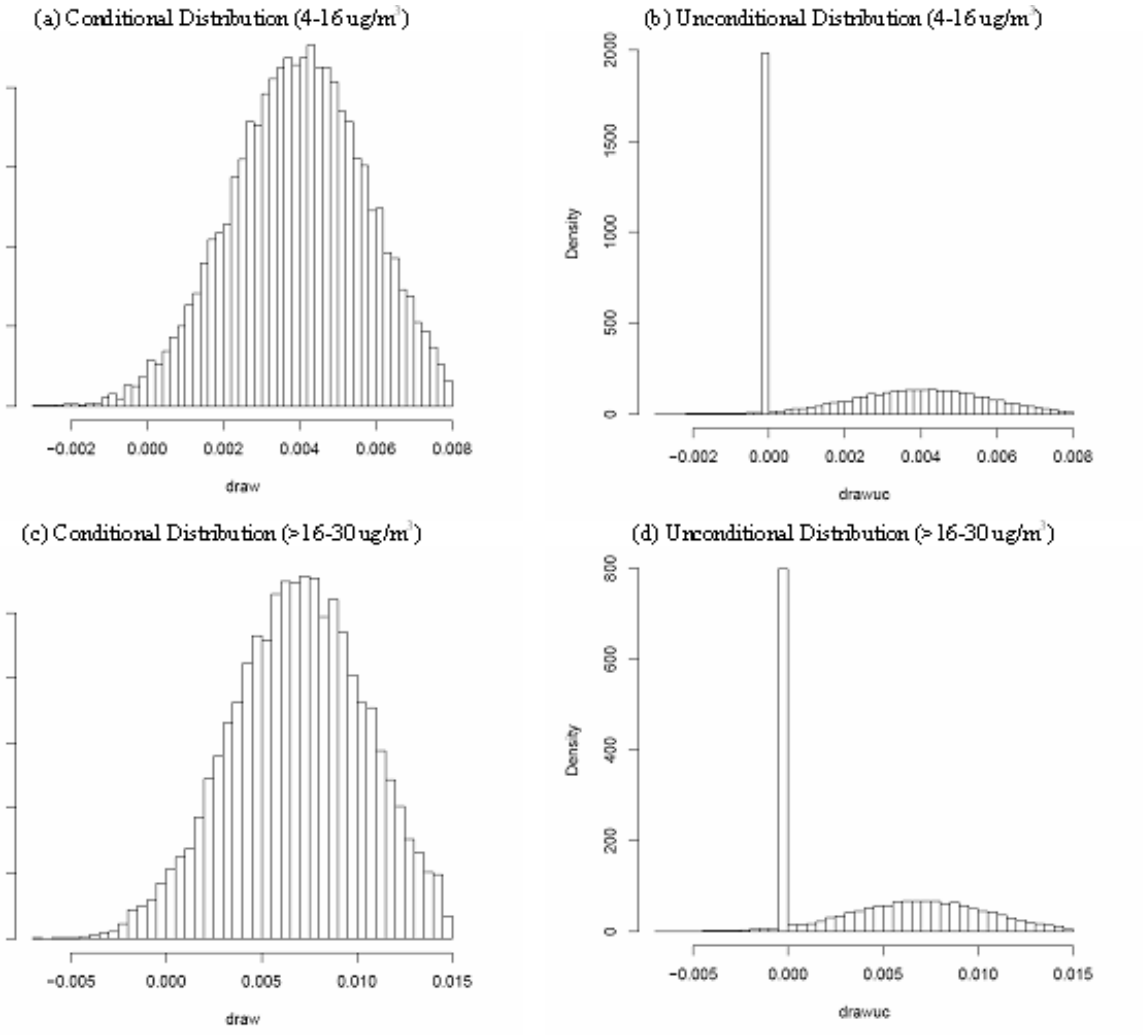


Notes:

- 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)).

E.1.1.4.11 Expert K

Figure E-11. Histogram of the Random Draw from the Distribution of the $PM_{2.5}$ Effect Specified by Expert K



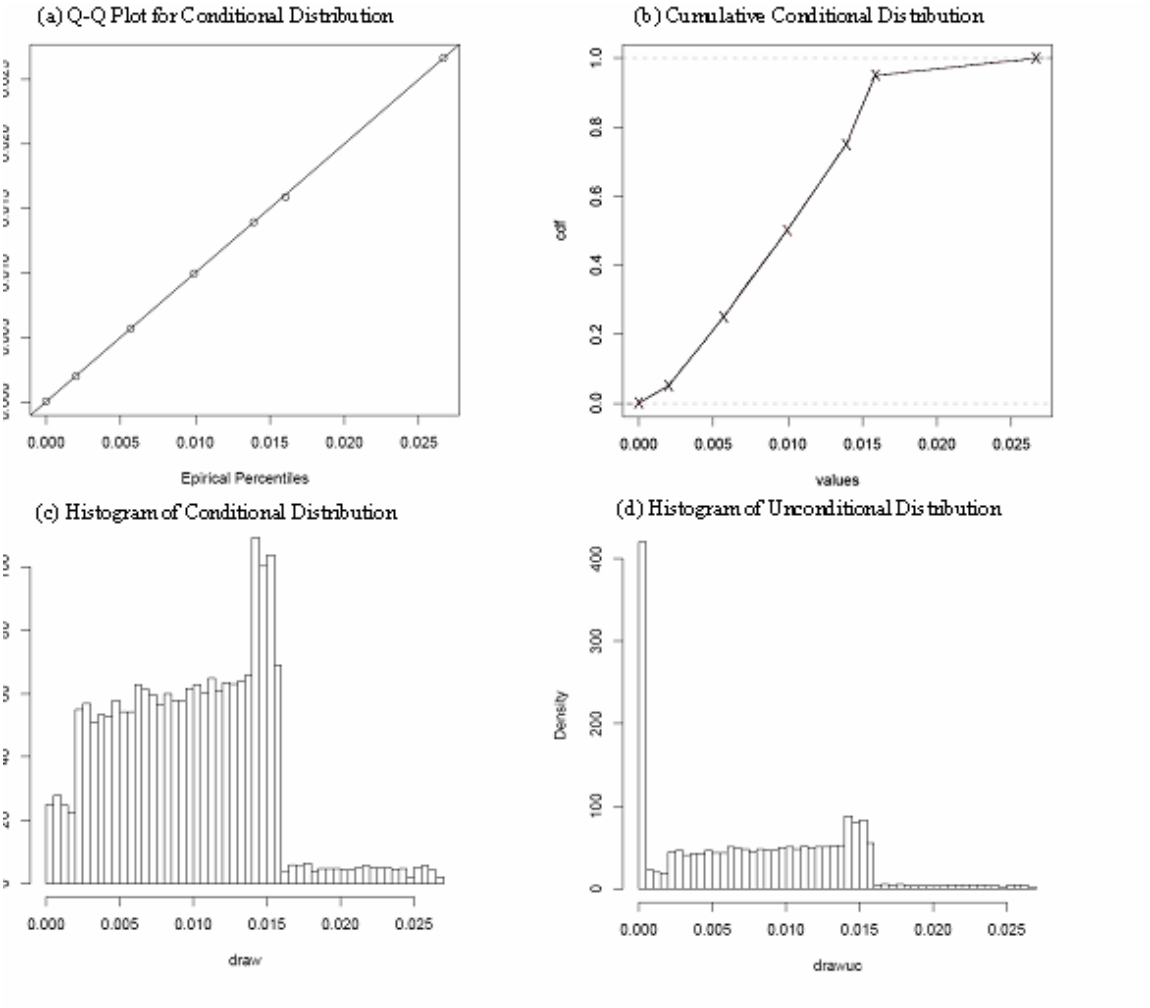
Notes:

- Expert K specified a truncated Normal Distribution for two ranges separately (4-16 µg/m^3 and >16-30 µg/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)).

E.1.1.4.12 Expert L

Figure E-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

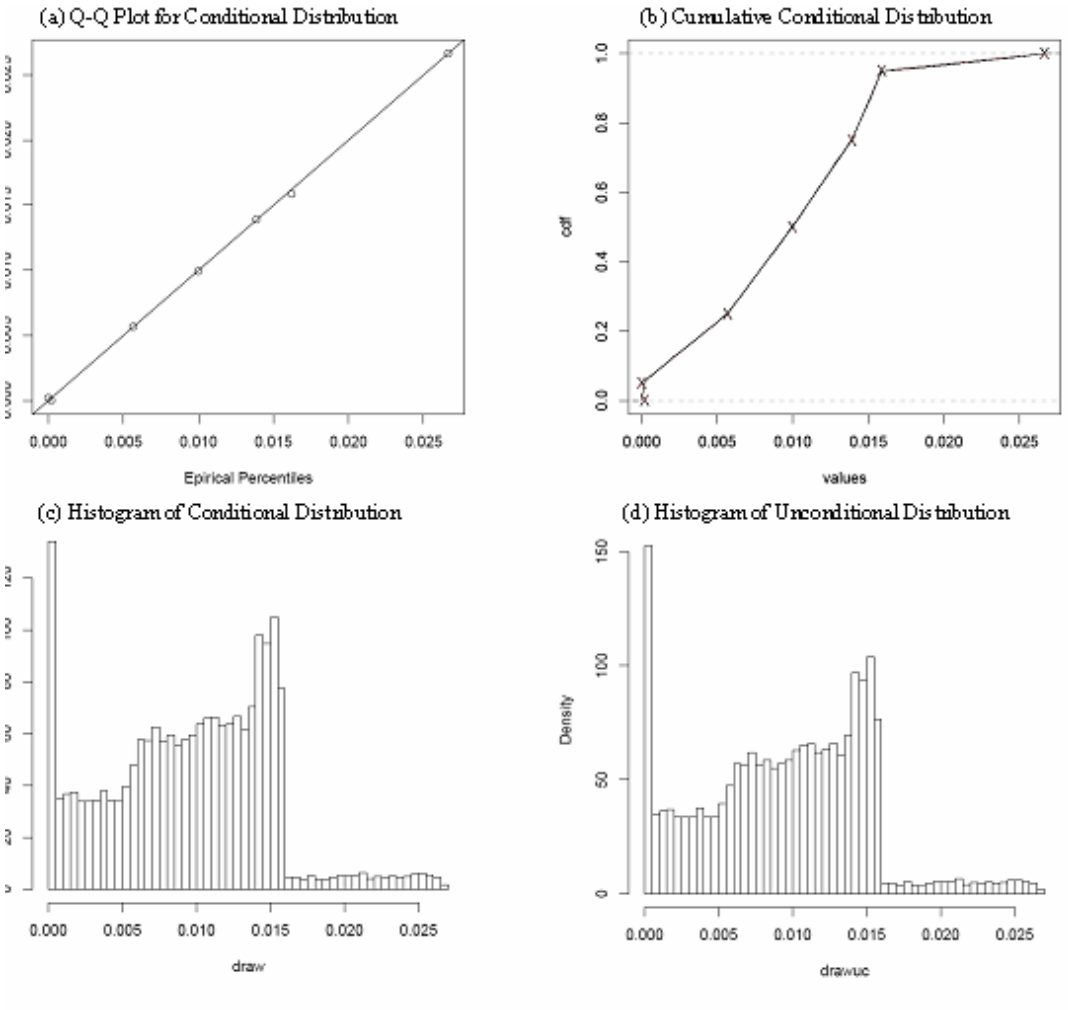


Notes:

- 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 E-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)).

E.1.2 Laden et al. (2006)

A large body of epidemiologic literature has found an association of increased fine particulate air pollution ($PM_{2.5}$) 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations were measured between 1979-1988, and estimated for later years from publicly available data. E xposure was defined as (1) city-specific mean $PM_{2.5}$ during the two follow-up periods, (2) mean $PM_{2.5}$ in the first period and change between these periods, (3) overall mean $PM_{2.5}$ across the entire follow-up, and (4) year-specific mean $PM_{2.5}$. 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 $\mu g/m^3$ increase in $PM_{2.5}$ 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). $PM_{2.5}$ 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 $PM_{2.5}$ (10 microg/m(3)) between periods (RR=0.73, 95% CI=0.57-0.95). Total, cardiovascular, and lung cancer mortality were each positively associated with ambient $PM_{2.5}$ concentrations. Reduced $PM_{2.5}$ concentrations were associated with reduced mortality risk.

All-Cause Mortality

The coefficient and standard error for $PM_{2.5}$ 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 $\mu g/m^3$ (Laden et al., 2006, p. 667).

E.1.3 Pope et al. (2002)

The Pope et al. (2002) analysis is a longitudinal cohort tracking study that uses the same American Cancer Society (ACS) 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 SO_2, NO_2, CO, and ozone. They did not find significant relationships between NO_2, CO, and ozone and premature mortality, but there were significant relationships between SO_4 (as well as SO_2), 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 $\mu g/m^3$ (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 $\mu g/m^3$ (Pope et al., 2002, Table 2).

E.1.4 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 $\mu g/m^3$ change in PM_{10} (Woodruff et al., 1997, Table 3).

E.1.5 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 $PM_{2.5}$ to infants born in California in 1999 and 2000 using maternal addresses for mothers who lived within 5 miles of a $PM_{2.5}$ 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 $PM_{2.5}$ 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/m^3 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/m^3 (Woodruff et al., 2006, p. 786).

E.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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 are estimated from the relative risks (1.06) and 95% confidence intervals (95% CI: 1.04-1.08) for a 10 $\mu g/m^3$ increase in the average of $PM_{2.5}$ 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 $\mu g/m^3$ increase in the average of $PM_{2.5}$ 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 $\mu g/m^3$ increase in the average of $PM_{2.5}$ 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.

E.2 Chronic/Severe Illness

Table E-6 below summarizes the 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 E-6. 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 Myocardinal Infractin, Nonfatal	Peters et al.	2001	Boston, MA	18-99		D24HourMean	0.024121	0.009285	Logistic	
Acute Myocardial	Pope et al.	2006	Greater Salt Lake City, UT	0-99		D24HourMean	0.0048	0.0019	Logistic	Index MI and

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Infraction, Nonfatal										unstable angina
Acute Myocardial Infraction, Nonfatal	Sullivan et al.	2005	King County, WA	0-99		D24HourMean	0.0019	0.0022	Logistic	
Acute Myocardial Infraction, 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 Infraction, 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 Abbey et al. (1995b)

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

Chronic Bronchitis

The estimated coefficient (0.0137) is presented for a one $\mu g/m^3$ 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 $\mu g/m^3$ 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%.

E.2.2 Peters et al. (2001)

Peters et al. (2001) studied the relationship between increased particulate air pollution and onset of heart attacks in the Boston area from 1995 to 1996. The authors used air quality data for PM_{10}, PM_{10}-2.5, $PM_{2.5}$,"black carbon", O_3, CO, NO_2, and SO_2 in a case-

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

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

Acute Myocardial Infarction, Nonfatal

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

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

Population: Population of ages 18 and older

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

E.2.3 Pope et al. (2006)

Pope et al. (2006) evaluated the association between short-term exposure to $PM_{2.5}$ and acute ischemic heart disease events, including acute nonfatal myocardial infarction, all acute coronary events, and subsequent myocardial infarctions in individuals living in

greater Salt Lake City, Utah. In a case-crossover study, these ischemic events were assessed in relation to a 10 μg/m^3 increase in $PM_{2.5}$. The researchers determined that a 10 μg/m^3 increase in $PM_{2.5}$ resulted in a 4.5% increase (95% CI: 1.1-8.0) in unstable angina and myocardial infarction.

Acute Myocardial Infarction, Nonfatal

Index MI and Unstable Angina

In a single-pollutant model the coefficient and standard error were estimated from the percent increase (4.81%) and 95% confidence interval (95% CI: 0.98-8.79) for a 10 μg/m^3 increase in daily 24-hour mean $PM_{2.5}$ (Pope et al., 2006, Table 3).

Incidence Rate: AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.2.

Population: All ages

Adjustment: See the adjustment description in Section E.2.2.

E.2.4 Sullivan et al. (2005)

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

Acute Myocardial Infarction, Nonfatal

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

Incidence Rate: AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.2.

Population: All ages

Adjustment: See the adjustment description in Section E.2.2.

E.2.5 Zanobetti and Schwartz (2006)

Zanobetti and Schwartz (2006) analyzed hospital admissions through emergency department for myocardial infarction (ICD-9 code 410) and pneumonia (ICD-9 codes 480-487) for associations with fine particulate air pollution, ozone, black carbon, nitrogen dioxide, PM not from traffic, and CO in the greater Boston area from 1995-1999. The authors used a case- crossover analysis with control days matched on temperature. Significant associations were detected for NO_2 with a 12.7% increase 95% CI: 5.8-18.0), $PM_{2.5}$ with an 8.6% increase (95% CI: 1.2-15.4), and black carbon with an 8.3% increase (95% CI: 0.2-15.8) in emergency myocardial infarction hospitalizations. Similarly, significant associations were identified for $PM_{2.5}$ with a 6.5% increase (95% CI: 1.1-11.4) and CO with a 5.5% increase (95% CI: 1.1-9.5) in pneumonia hospitalizations.

Acute Myocardial Infarction, Nonfatal

The study looked at hospital admissions of AMI through 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 $\mu g/m^3$ increase in daily 24-hour mean $PM_{2.5}$ for an average of the 0- and 1-day lag (Zanobetti A. and Schwartz, 2006, Table 4).

Incidence Rate: AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.2.

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.2.2.

E.2.6 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 $\mu g/m^3$ increase in 2-day averaged $PM_{2.5}$, a 1.89% (95% CI: 1.34-2.45) increase in cardiovascular disease admissions, a 2.25% (95% CI: 1.10-3.42) increase in myocardial infarction admissions, a 1.85% (95% CI: 1.19-2.51) increase in congestive heart failure admissions, a 2.74% (95% CI: 1.30-4.20) increase in diabetes admissions, and a 2.07% (95% CI: 1.20-2.95) increase in

respiratory admissions were observed. The relationship between $PM_{2.5}$ and cardiovascular admissions was significantly modified when the mass of $PM_{2.5}$ was high in Br, Cr, Ni, and sodium ions, while mass high in As, Cr, Mn, organic carbon, Ni and sodium ions modified the myocardial infarction relationship and mass high in As, orgarnic 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 $\mu g/m^3$ increase in 2-day averaged $PM_{2.5}$ (Zanobetti et al., 2009, Table 3).

Incidence Rate: AMI hospital admission rate for all ages. See the incidence rate discussion under Peters et al. (2001) in Section E.2.2.

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 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.2.2.

E.3 Hospitalizations

Table E-7 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-7. 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
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	
Chronic Lung	Ito	2003	Detroit, MI	65-99		D24HourMean	0.001169	0.002064	Log-linear	
Pneumonia	Ito	2003	Detroit, MI	65-99		D24HourMean	0.003979	0.001659	Log-linear	
All Cardiovascular (less Myocardial Infarctions)	Moolgavkar	2000	Los Angeles, CA	18-64		D24HourMean	0.001400	0.000341	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	2003	Los Angeles, CA	65-99		D24HourMean	0.001580	0.000344	Log-linear	
Chronic lung	Moolgavkar	2003	Los Angeles, CA	65-99		D24HourMean	0.001850	0.000524	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

E.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 $\mu g/m^3$ 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.

E.3.2 Bell et al. (2008)

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 $\mu g/m^3$ 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 $\mu g/m^3$ 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.

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

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/m^3 increase in $PM_{2.5}$ in the 3-day lag GAM stringent model (Ito, 2003, Table 8).

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/m^3 in the 1-day lag GAM stringent model (Ito, 2003, Table 7).

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/m^3 increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Ito, 2003, Table 10).

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/m^3 increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Ito, 2003, Table 11).

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/m^3 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.

E.3.4 Moolgavkar (2000a), Chronic Lung

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, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group in Chicago and Phoenix, weak associations were observed between the gaseous pollutants and admissions. No consistent associations were observed for PM_{10}. In Los Angeles, marginally significant associations were observed for $PM_{2.5}$, which were generally lower than for the gases. In co-pollutant models with CO, the $PM_{2.5}$ effect was reduced. Similar results were observed in the 0-19 and 20-64 year old age groups.

The $PM_{2.5}$ C-R functions are based on the 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 μg/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 (RR - 1) * 100.

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

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.

E.3.5 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, SO_2, NO_2, CO, and PM_{10} in all three areas. $PM_{2.5}$ data was available only in Los Angeles. The data were analyzed using a Poisson regression model with generalized additive models to adjust for temporal trends. Separate models were run for 0 to 5 day lags in each location. Among the 65+ age group, the gaseous pollutants generally exhibited stronger effects than PM_{10} or $PM_{2.5}$. The strongest overall effects were observed for SO_2 and CO. In a single pollutant model, $PM_{2.5}$ was statistically significant for lag 0 and lag 1. In co-pollutant models with CO, the $PM_{2.5}$ effect dropped out and CO remained significant. For ages 20-64, SO_2 and CO exhibited the strongest effect and any $PM_{2.5}$ effect dropped out in co-pollutant models with CO.

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 $\mu g/m^3$ 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.

E.3.6 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 μg/m^3 increase in $PM_{2.5}$ 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 (RR - 1) * 100.

The $PM_{2.5}$ C-R functions for the 65+ age group are based on the reanalysis in Moolgavkar (2003) of the single and co-pollutant models ($PM_{2.5}$ 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 μg/m^3 increase in $PM_{2.5}$ 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 (RR - 1) * 100.

E.3.7 Peng et al. (2008)

Peng et al. (2008) examined the risk of hospital admissions for cardiovascular and respiratory diseases in relation to particulate matter (PM_{10}-2.5 and $PM_{2.5}$). 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. PM_{10}-2.5 and $PM_{2.5}$ 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 μg/m^3 increase in 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 $PM_{2.5}$. For respiratory disease admissions, a 10 μg/m^3 increase in 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 $\mu g/m^3$ 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.

E.3.8 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 $\mu g/m^3$ increase in daily 24-hour mean $PM_{2.5}$ 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.

E.3.9 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}, $PM_{2.5}$, coarse 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. $PM_{2.5}$ levels were estimated from light scattering data. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $\mu g/m^3$ increase in $PM_{2.5}$ in the 1-day lag GAM stringent model (Sheppard, 2003, pp. 228-229).

E.3.10 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 $\mu g/m^3$ increase in 2-day averaged $PM_{2.5}$, 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 $PM_{2.5}$ 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.

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

E.4 Emergency Room Visits

Table E-8 summarizes the health impacts functions used to estimate the relationship between $PM_{2.5}$ and emergency room visits. Below, we present a brief summary of each of the studies and any items that are unique to the study.

Table E-8. Health Impact Functions for Particulate Matter and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form
Asthma	Mar et al.	2010	Greater Tacoma, WA	0-99		D24HourMean	0.0056	0.0021	Log-linear
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

E.4.1 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/m^3 increase in daily 24-hour mean $PM_{2.5}$ at lag day 2 (Mar et al., 2010, Table 4).

E.4.2 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 PM_{10}, light scattering (used to estimate fine PM), CO, SO_2, NO_2, and O_3 in a Poisson regression model with adjustments for day of the week, time trends, temperature, and dew point. They found significant associations between asthma ER visits and light scattering (converted to $PM_{2.5}$), PM_{10}, and CO. No association was found between O_3, NO_2, or SO_2 and asthma ER visits, although O_3 had a significant amount of missing data. In multipollutant models with either PM metric (light scattering or PM_{10}) and NO_2 and SO_2, the PM coefficients remained significant while the gaseous pollutants were not associated with increased asthma ER visits. The PM C-R functions are based on results of the single and multipollutant models reported.

Emergency Room Visits, Asthma

In a model with NO_2 and SO_2, the $PM_{2.5}$ coefficient and standard error are calculated from a relative risk of 1.17 (95% CI 1.08-1.26) for a 9.5 μg/m^3 increase in $PM_{2.5}$ (Norris et al., 1999, p. 491).

E.4.3 Slaughter et al. (2005)

Slaughter et al. (2005) examined the short-term association of particulate matter (PM1, $PM_{2.5}$, PM_{10}, 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/m^3 increase in daily 24-hour mean $PM_{2.5}$ at 1-day lag (Slaughter et al., 2005, Table 4).

E.5 Minor Effects

Table E-9 summarizes the health impacts functions used to estimate the relationship between $PM_{2.5}$ and minor effects. Below, we present a brief summary of each of the studies and any items that are unique to the study.

Table E-9. 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

E.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 PM2.1 and PM_{10} were marginally significantly related to bronchitis. The original study measured PM2.1, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of PM2.1 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 PM15. 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 (PM2.1 = 20.7 μg/m^3) versus the least polluted city (PM2.1 = 5.8 μg/m^3) (Dockery et al., 1996, Tables 1 and 4). The original study used PM2.1, however, we use the PM2.1 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.

E.5.2 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:

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

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

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

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.5.3 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.5.4 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 PM_{10}, 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/m^3 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

E.6 Asthma-Related Effects

Table E-10 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-10. 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 eta 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	

E.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/m^3 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/m^3 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).

E.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 PM_{10}, $PM_{2.5}$, NO_2, and O_3 in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. The authors note that there were 26 days in which $PM_{2.5}$ concentrations were reported higher than PM_{10} concentrations. The majority of results the authors reported were based on the full dataset. These results were used for the basis for the C-R functions. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom- free day.

The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among asthmatics. The authors found both the prevalence and incidence of wheeze associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics.

The 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/m^3 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 μg/m^3 increase in 12-hour average $PM_{2.5}$ 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 μg/m^3 increase in 12-hour average $PM_{2.5}$ 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.)

E.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 PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Upper Respiratory Symptoms

The coefficient and standard error for a one μg/m^3 change in PM_{10} 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.)

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

In this Appendix, we present the health impact functions used to estimate ozone-related adverse health effects. 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 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 F-1. Health Impact Functions for Ozone and Short-Term 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		D24HourMean	0.000390	0.000133	Log-linear	Warm season
Non-Accidental	Bell et al.	2004	95 US cities	0-99		D24HourMean	0.000520	0.000128	Log-linear	All year
Non-Accidental	Bell et al.	2004	95 US cities	0-99		D8HourMean	0.000261	0.000089	Log-linear	Warm season. 8-hour max from24-hour mean
All Cause	Bell et al.	2005	US & non-US	0-99		D24HourMean	0.001500	0.000401	Log-linear	Warm season
All Cause	Bell et al.	2005	US & non-US	0-99		D8HourMean	0.000795	0.000212	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
Cardio-pulmonary	Huang et al.	2005	19 US cities	0-99		D8HourMean	0.000813	0.000259	Log-linear	Warm season. 8-hour max from 24-hour mean.

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
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		D1HourMax	0.000400	0.000066	Log-linear	1-hour max
Non-Accidental	Ito et al.	2005		0-99		M24HourMean	0.001750	0.000357	Log-linear	Warm season. 24-hour mean
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		D8HourMax	0.000532	0.000088	Log-linear	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.
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.
Non-Accidental	Moolgavkar et al.	1995	Phila, PA	0-99		D24HourMean	0.001398	0.000266	Log-linear	Warm season.
Non-Accidental	Moolgavkar et al.	1995	Phila, PA	0-99	TSP, SO_2	D24HourMean	0.001389	0.000373	Log-linear	Warm season.
Non-Accidental	Moolgavkar et al.	1995	Phila, PA	18-99	TSP, SO_2	D24HourMean	0.000611	0.000216	Log-linear	
Non-Accidental	Samet et al.	1997	Phila, PA	18-99	CO, NO_2, SO_2, TSP	D24HourMean	0.000936	0.000312	Log-linear	
Non-Accidental	Schwartz	2005	14 US cities	0-99		D1HourMax	0.000370	0.000130	Logistic	Warm season
Non-Accidental	Schwartz	2005	14 US cities	0-99		D8HourMax	0.000426	0.000150	Logistic	Warm season. 8-hour max from 1-hour max.

F.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).

F.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).

F.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 PM_{10}, but are robust to: (i) the adjustment for long-term trends, other gaseous pollutants (NO_2, SO_2 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.

F.1.4 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 tjey 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 $\mu g/m^3$ change in ozone. We converted $\mu g/m^3$ to ppb with an assumed relationship of 1.96 $\mu g/m^3$ per 1.0 ppb.

F.1.5 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 -- PM_{10}, ozone, CO, SO_2, and visual range-derived extinction coefficient -- both PM_{10} 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 PM_{10} or ozone.

The authors reported a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. In single pollutant models the effects were slightly larger. The health impact function for ozone is based on results from the co-pollutant models.

Non-Accidental Mortality

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

F.1.6 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 both 1-hour daily maximum and 24-hour daily average metrics. We present both below.

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%.

F.1.7 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 SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant. In season-specific models, ozone was significantly associated with mortality only in the summer months.

Mortality, Non-Accidental

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.

F.1.8 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, SO_2, NO_2, and CO in a Poisson regression model. In single pollutant models, ozone, SO_2, TSP, and CO were significantly associated with mortality. In a five-pollutant model, they found a positive statistically significant relationship for each pollutant except NO_2.

Mortality, Non-Accidental

The health impact function for ozone is based on the five-pollutant model (ozone, CO, NO_2, SO_2 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.

F.1.9 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 03%, 0.35%). Control for suspended particulate matter with an aerodynamic diameter of 10 mum or less (PM(10)) 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%.

F.2 Long-Term Mortality

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

Table F-2. Health Impact Functions for Ozone and Long-Term Mortality

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Fun. Form
Mortality, Respiratory	Jerrett et al.	2009	86 urban areas	30-99	$PM_{2.5}$	Annual	0.003922	0.001325	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Northeast	30-99		Annual	-0.001005	0.003853	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Industrial Midwest	30-99		Annual	0.000000	0.004604	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Southeast	30-99		Annual	0.011333	0.003193	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Upper Midwest	30-99		Annual	0.013103	0.026212	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Northwest	30-99		Annual	0.005827	0.003118	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Southwest	30-99		Annual	0.019062	0.007583	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	Southern California	30-99		Annual	0.000995	0.002767	Log-linear
Mortality, Respiratory	Jerrett et al.	2009	86 urban areas	30-99	$PM_{2.5}$	Annual	0.004471	0.001510	Log-linear

F.2.1 Jerret 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 & Bell table 2).

F.3 Hospital Admissions

Table F-3 summarizes the health impacts 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-3. 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	$PM_{2.5}$	D1HourMax	0.007301	0.002122	Log-linear	Warm season
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.
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
Chronic Lung (less Asthma)	Schwartz	1994	Detroit, MI	65-99	PM_{10}	D24HourMean	0.005523	0.002085	Log-linear	All year

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}	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

F.3.1 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, NO_2, SO_2, and ozone. Daily measures of particulate matter were estimated for the May to August period of 1992-1994 using TSP, sulfates, and coefficient of haze data. The authors report a positive association between ozone in the May through August months and respiratory hospital admissions, for several single days after elevated ozone levels.

The strongest association was found using a five-day moving average of ozone. No association was found in the September through April months. In co-pollutant models with a particulate matter or another gaseous pollutant, the ozone effect was only slightly diminished. The effects for PM and gaseous pollutants were generally significant in single pollutant models but diminished in co-pollutant models with ozone,

with the exception of CO. The C-R 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).

F.3.2 Moolgavkar et al. (1997)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions (ICD codes 490-496) for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a Poisson regression, they found no significant effect for any of the pollutants (PM_{10}, ozone, or CO). The effect for ozone was marginally significant. The model with a 100 df smoother was reported to be optimal (p. 368). The health impact function for chronic lung disease is based on the results from a three-pollutant model (ozone, CO, PM_{10}) 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 PM_{10}, the estimated coefficient and standard error are based on the percent increase (4.2) and 95% confidence interval of the percent increase (-1.0-9.4) associated with a change in daily average ozone levels of 15 ppb (Moolgavkar et al., 1997, Table 4).

Hospital Admissions, Pneumonia (ICD-9 codes 480-487)

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

F.3.3 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 PM_{10} were significantly associated with pneumonia admissions. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. The results were not sensitive to the methods used to control for seasonal patterns and weather. The ozone C-R functions are based on the results of the single pollutant model and the two-pollutant model (PM_{10} and ozone) with spline smoothing for temporal patterns and weather.

Hospital Admissions, Pneumonia (ICD-9 codes 480-487)

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

F.3.4 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 PM_{10} and ozone significantly linked to pneumonia and COPD. The authors state that effect estimates were relatively unchanged compared to the unreported single pollutant models. No significant associations were found between either pollutant and asthma admissions. The C-R function for chronic lung disease incidence is based on the results of the "basic" co-pollutant model (ozone and PM_{10}) presented in Table 4 (p. 651). 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).

F.3.5 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 (SO_2) 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 SO_2, inhalable particles (PM_{10}), 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 PM_{10} associations were little changed by control for either ozone or SO_2. The ozone association was likewise independent of the other pollutants. The SO_2 association was substantially attenuated by control for ozone in both cities, and by control for PM_{10} 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/m^3 increase in PM_{10}, 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 PM_{10}, followed by ozone.

Hospital Admissions, All Respiratory (ICD-9 codes 460-519) -- Tacoma

In a model with PM_{10}, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.06-1.37) for a 50 µg/m^3 increase in average daily ozone levels (Schwartz, 1995, Table 6, p. 535).To calculate the coefficient, a conversion of 1.96 µg/m^3 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 PM_{10}, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.15) for a 50 µg/m^3 increase in average daily ozone levels (Schwartz, 1995, Table 3, p. 534).To calculate the coefficient, a conversion of 1.96 µg/m^3 per ppb was used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius).

F.4 Emergency Room Visits

Table F-4 summarizes the 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-4. Health Impact Functions for Ozone and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form
Asthma	Peel et al.	2005	Atlanta, GA	0-99		D8HourMax	0.000870	0.000529	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

F.4.1 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 PM_{10}, ozone, NO_2, CO, and SO_2 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, NO_2, CO, and PM_{10} 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 NO_2 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, NO_2, 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.

F.4.2 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 SO_2, 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). I n Portland, an IQR increase in SO_2 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 O_3 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 SO_2 and O_3 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).

F.5 Minor Effects

Table F-5 summarizes the 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-5. 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	D1HourMax	0.013247	0.004985	Linear	
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	Gilliland et al.	2001	Southern California	5-17		D8HourMax	0.007824	0.004445	Log-linear	All year, 8-hour max from 8-hour mean.
School Loss Days, All Cause	Gilliland et al.	2001	Southern California	5-17		D8HourMean	0.008150	0.004630	Log-linear	
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	0.002596	0.000776	Log-linear	8-hour max from 1-hour max.

F.5.1 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. PM_{10} was negatively associated with absence rate, after adjustment for ozone, CO, and meteorological and temporal variables. The C-R function for ozone is based on the results from a multiple linear regression model with CO, ozone, and PM_{10}.

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*5/7).)

F.5.2 Gilliland et al. (2001)

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

School Loss Days

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

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

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

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

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

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

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

Incidence Rate: daily school absence rate = 0.055 (U.S. Department of Education, 1996, Table 42-1)

Population: population of children ages 9-10 not absent from school on a given day = 94.5% of children ages 9-10 (The proportion of children not absent from school on a given day (5.5%) is based on 1996 data from the U.S. Department of Education (1996, Table 42-1).)

Scaling Factor: Proportion of days that are school days in the ozone season = 0.393.

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

F.5.3 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.6 Converting Functions to 8-Hour Daily Maximum Metric

A number of health impact functions were converted from 1-hour maximum, 24-hour average, and 8-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. We calculated ozone metric ratios for each quarter and year in the period 2000-2007. We calculated ratios by monitor, and by county, core business statistical area (CBSA), state, and nation.

For each monitor, a day was considered valid if it had at least 18 hourly values out of 24. A quarter was considered valid if it had at least 85 percent valid days. Ratios are calculated for the year, only if that year had four quarterly values. The CBSA codes, which were defined by OMB on 6-6-03, were obtained from: http://www.census.gov/population/estimates/metro-city/03msa. txt. We chose the time period for the ratio calculation (e.g., spring and summer quarters) and the locations based on the data used in each epidemiological study. Table F-6 presents the 8-hour adjustment used for each study. Tables G-7 through G-9 present supporting documentation for some of the multi-city 8-hour adjustments.

Table F-6. Eight-Hour Adjustments by Study

Effect	Author	Year	Location	Adjustment Factor Location	Quarters	Metric	8-Hour Adj	Notes
Mortality, Non-Accidental	Bell et al.	2004	95 US cities	Nation	2-3	24HourMean	0.67	
Mortality, All Cause	Bell et al.	2005	Meta-analysis	From study. See comment	--	24HourMean	0.53	
HA, All Respiratory	Burnett et al.	2001	Toronto, CAN	Buffalo-Checktowaga-Tonawanda, NY MSA	2-3	1HourMax	1.12	
School Loss Days, All Cause	Chen et al.	2000	Washoe Co, NV	Washoe County	1-4	1HourMax	1.19	
School Loss Days, All Cause	Gilliland et al.	2001	Southern California	Los Angeles-Long Beach-Santa Ana, CA MSA	1-4	8HourMean	0.96	The statewide avg is 0.96
Mortality, Cardiopulmonary	Huang et al.	2005	19 US cities	See below	See below	24HourMean	0.66	
Mortality, Non-Accidental	Ito et al.	2005	6 US cities	See below	See below	24HourMean	0.65	
Mortality, Non-Accidental	Ito et al.	2005	Meta-analysis	From study. See comment.	--	24HourMean	0.67	
Mortality, Non-Accidental	Ito et al.	2006	Meta-analysis	From study. See comment.	--	1HourMax	1.33	
Mortality, Non-Accidental	Levy et al.	2005	Meta-analysis	From study. See comment.	--	1HourMax	1.33	
HA, chronic Lung Disease	Moolgavkar et al.	1997	Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI MSA	1-4	24HourMean	0.70	Data 2004-2007 only
HA, Pneumonia	Moolgavkar et al.	1997	Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI MSA	1-4	24HourMean	0.70	Data 2004-2007 only
Minor Restricted Activity Days	Ostro and Rothschild	1989	Nationwide	Nation	1-4	1HourMax	1.18	

Effect	Author	Year	Location	Adjustment Factor Location	Quarters	Metric	8-Hour Adj	Notes
HA, Chronic Lung Disease (less Asthma)	Schwartz	1994	Detroit, MI	Detroit-Warren-Livonia, MI MSA	1-4	24HourMean	0.62	Data 2006 only
HA, Pneumonia	Schwartz	1994	Detroit, MI	Detroit-Warren-Livonia, MI MSA	1-4	24HourMean	0.62	Data 2006 only
HA, Pneumonia	Schwartz	1994	Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI MSA	1-4	24HourMean	0.70	Data 2004-2007 only
HA, All Respiratory	Schwartz	1995	New Haven, CT	New Haven-Milford, CT MSA	2-3	24HourMean	0.67	
HA, All Respiratory	Schwartz	1995	Tacoma, WA	Seattle-Tacoma-Bellevue, WA MSA	2-3	24HourMean	0.69	
Mortality, Non-Accidental	Schwartz	2004	14 US cities	See below	See below	1HourMax	0.67	

Table F-7. Eight-Hour Adjustment Details – 6-City Study

City/County	CBS As or Counties Used in Ratio Average	Quarters Used	Study Metric	8-Hour Adj
Detroit	Detroit-Warren-Livonia, MI MSA	2-3	24HourMean	0.63
Cook County	Cook County	2-3	24HourMean	0.65
Houston	Houston-Baytown-Sugar Land, TX MSA	2-3	24HourMean	0.59
Minneapolis	Minneapolis-St. Paul-Bloomington, MN-WI MSA	2-3	24HourMean	0.70
Philadelphia	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD MSA	2-3	24HourMean	0.65
St. Louis	St. Louis, MO-IL MSA	2-3	24HourMean	0.64
	Average			**0.64**

Table F-8. Eight-Hour Adjustment Details – 14-City Study

City/County	CBS As or Counties Used in Ratio Average	Quarters Used	Study Metric	8-Hour Adj	Notes
Birmingham	Birmingham-Hoover, AL MSA	2-3	1HourMax	1.15	
Boulder	Boulder, CO MSA	2-3	1HourMax	1.15	
Canton	Canton-Massillon, OH MSA	2-3	1HourMax	1.12	
Chicago	Chicago-Naperville-Joliet, IL-IN-WI MSA	2-3	1HourMax	1.16	
Cincinnati	Cincinnati-Middletown, OH-KY-IN MSA	2-3	1HourMax	1.15	
Colorado Springs	Colorado Springs, CO MSA	2-3	1HourMax	1.11	
Columbus	Columbus, OH MSA	2-3	1HourMax	1.13	
Detroit	Detroit-Warren-Livonia, MI MSA	2-3	1HourMax	1.17	
Houston	Houston-Baytown-Sugar Land, TX MSA	2-3	1HourMax	1.22	
New Haven	New Haven-Milford, CT MSA	2-3	1HourMax	1.17	
Pittsburgh	Pittsburgh, PA MSA	2-3	1HourMax	1.15	
Provo	Provo-Orem, UT MSA	3	1HourMax	1.13	Only quarter 3 available
Seattle	Seattle-Tacoma-Bellevue, WA MSA	2-3	1HourMax	1.15	
Spokane	Spokane, WA MSA	3	1HourMax	1.08	Only quarter 3 available
	Average			**1.15**	

Table F-9. Eight-Hour Adjustment Details – 19-City Study

City/County	CBS As or Counties Used in Ratio Average	Quarters Used	Study Metric	8-Hour Adj	Notes
Atlanta	Atlanta-Sandy Springs-Marietta, GA MSA	2-3	24HourMean	0.59	
Chicago	Chicago-Naperville-Joliet, IL-IN-WI MSA	2-3	24HourMean	0.65	
Cleveland	Cleveland-Elyria-Mentor, OH MSA	2-3	24HourMean	0.69	
Dallas/Ft. Worth	Dallas-Fort Worth-Arlington, TX MSA	2-3	24HourMean	0.67	
Detroit	Detroit-Warren-Livonia, MI MSA	2-3	24HourMean	0.63	
Houston	Houston-Baytown-Sugar Land, TX MSA	2-3	24HourMean	0.59	
Los Angeles	Los Angeles-Long Beach-Santa Ana, CA MSA	2-3	24HourMean	0.59	Los Angeles and Santa Ana/Anaheim have same CBSA
Santa Ana/Anaheim	Los Angeles-Long Beach-Santa Ana, CA MSA	2-3	24HourMean	0.59	
Miami	Miami-Fort Lauderdale-Miami Beach, FL MSA	2-3	24HourMean	0.71	
New York	New York-Newark-Edison, NY-NJ-PA MSA	2-3	24HourMean	0.66	
Philadelphia	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD MSA	2-3	24HourMean	0.65	
Phoenix	Phoenix-Mesa-Scottsdale, AZ MSA	2-3	24HourMean	0.66	
Pittsburgh	Pittsburgh, PA MSA	2-3	24HourMean	0.61	
San Bernardino	Riverside-San Bernardino-Ontario, CA MSA	2-3	24HourMean	0.65	
San Antonio	San Antonio, TX MSA	2-3	24HourMean	0.66	
San Diego	Sand Diego-Carlsbad-San Marcos, CA MSA	2-3	24HourMean	0.70	
Oakland	San Francisco-Oakland-Fremont, CA MSA	2-3	24HourMean	0.70	
San Jose	San Jose-Sunny vale-Santa Clara, CA MSA	2-3	24HourMean	0.64	
Seattle	Seattle-Tacoma-Bellevue, WA MSA	2-3	24HourMean	0.69	
	Average			**0.65**	**Keeping 1 Los Angeles keeps 8-hour adj at 0.65**

Appendix G: Nitrogen Dioxide Health Impact Functions in U.S. Setup

In this Appendix, we present the health impact functions used to estimate NO_2-related adverse health effects. 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.

G.1 Hospital Admissions

Table G-1 summarizes the health impacts functions used to estimate the relationship between nitrogen dioxide 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-1. Health Impact Functions for Nitrogen Dioxide and Hospital Admissions

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
All Respiratory	Fung et al.	2006	Vancouver, Canada	65-99		D24HourMean	0.003285	0.001707	Log-linear	
Asthma	Linn et al.	2000	Metropolitan Los Angeles	0-29		D24HourMean	0.002400	0.000800	Log-linear	
Asthma	Linn et al.	2000	Metropolitan Los Angeles	30-99		D24HourMean	0.001400	0.000500	Log-linear	
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-14		D1HourMax	0.006747	0.003628	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-14		D1HourMax	-0.002878	0.003231	Log-linear	Male
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	15-64		D1HourMax	0.007139	0.004353	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	15-64		D1HourMax	0.000746	0.005879	Log-linear	Male
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	65-99		D1HourMax	0.001238	0.002961	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	65-99		D1HourMax	-0.001435	0.004295	Log-linear	Male
Chronic Lung Disease	Moolgavkar S.H.	2003	Los Angeles County, CA	65-99		D24HourMean	0.001800	0.000188	Log-linear	
Chronic Lung Disease	Moolgavkar S.H.	2003	Cook County, CA	65-99		D24HourMean	0.002400	0.000803	Log-linear	
All Respiratory	Yang et al.	2003	Vancouver, Canada	65-99	SO_2, O_3, CO, COH	D24HourMean	0.008759	0.003069	Logistic	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Form	Notes
Chronic Lung Disease (less Asthma)	Yang et al.	2005	Vancouver, Canada	65-99	O_3	D24HourMean	0.020605	0.006637	Log-linear	

G.1.1 Fung et al. (2006)

Fung et al. (2006) assessed the impact of ambient gaseous pollutants (SO_2, NO_2, CO, and O_3) and particulate matters (PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$) as well as the coefficient of haze (COH) on recurrent respiratory hospital admissions (ICD-9 codes 460-519) among the elderly in Vancouver, Canada, for the period of June 1, 1995, to March 31, 1999, using a new method proposed by Dewanji and Moolgavkar(2000; 2002). The associations were conducted at current day, 3-day, 5-day, and 7-day moving averages. The strongest association between NO_2 and hospital admissions was observed at 3-day lag (RR = 1.018, 95% CI: 1.000-1.037). For SO_2, significant associations were found between admissions and 3-day, 5-day, and 7-day moving averages of the ambient SO_2 concentrations, with the strongest association observed at the 7-day lag (RR = 1.044, 95% CI: 1.018-1.070). The authors found $PM_{10-2.5}$ for 3-day and 5-day lag to be significant, with the strongest association at 5-day lag (RR = 1.020, 95% CI: 1.001-1.039). No significant associations with admission were found with current day exposure.

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

In a single-pollutant model the coefficient and standard error are estimated from the relative risk (1.018) and 95% confidence interval (95% CI: 1.000-1.037) for a 5.431 ppb increase in 3-day moving average of NO_2 (Fung, et al., 2006, Table 4).

G.1.2 Linn et al. (2000)

Linn et al. (2000) evaluated associations between air pollution and hospital admissions for cardiopulmonary illnesses in metropolitan Los Angeles during 1992-1995. In a single-pollutant Poisson regression model, daily average of NO_2 (year-round) was found significantly associated with same-day asthma hospital admissions for both age groups (i.e., 0-29 and 30-99). The results for winter and autumn were also reported but insignificant.

Hospital Admissions, Asthma (ICD-9 code 493)

Linn et al. (2000, Table 9) reported the coefficients and standard error for patients = 30 years of age and Linn et al. (2000, p.432) reported the coefficients and standard error for patients <30 years of age.

G.1.3 Luginaah et al. (2005)

Luginaah et al. (2005) assessed the association between air pollution and daily respiratory hospitalization (ICD-9 codes 460-519) for different age and sex groups from 1995 to 2000. The pollutants included were NO_2, SO_2, CO, O_3, PM_{10}, coefficient of haze (COH), and total reduced sulfur (TRS). The authors estimated relative risks (RR) using both time-series and case- crossover methods after controlling for appropriate confounders (temperature, humidity, and change in barometric pressure). The results of both analyses were consistent.

They found associations between NO_2, SO_2, CO, COH, or PM_{10} and daily hospital admission of respiratory diseases especially among females. For females 0-14 years of age, there was 1-day delayed effect of NO_2 (RR = 1.19, case-crossover method), a current-day SO_2 (RR = 1.11, time series), and current-day and 1- and 2-day delayed effects for CO by case crossover (RR = 1.15, 1.19, 1.22, respectively). Time-series analysis showed that 1-day delayed effect of PM_{10} on respiratory admissions of adult males (15-64 years of age), with an RR of 1.18. COH had significant effects on female respiratory hospitalization, especially for 2-day delayed effects on adult females, with RRs of 1.15 and 1.29 using time-series and case-crossover analysis, respectively. There were no significant associations between O_3 and TRS with respiratory admissions.

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

For different age and sex groups, the coefficient and standard error are estimated from the relative risks and 95% confidence interval reported for single-pollutant models using time-series method for a 16 ppb increase in daily 1-hour max NO_2 levels (Luginaah, et al., 2005, Table 3). The time-series method is chosen based on the discussion in Fung et al. (2003).

G.1.4 Moolgavkar (2003)

Moolgavkar (2003) presented re-analyses of Moolgavkar (2000c; 2000a; 2000b) about the associations between air pollution and daily deaths and hospital admissions in Los Angeles and Cook counties in the United States. The principal reason for conducting these re-analyses was to assess the impact of using convergence criteria that are more stringent than the default criteria used in the S-Plus software package. The author also reported the results of generalized linear model (GLM) analyses using natural splines with the same degree of freedom as the smoothing splines he used in the generalized additive model (GAM) analyses. In single-pollutant Poisson regression models, hospital admissions for COPD (ICD-9 code 490-496) were associated with daily average of NO_2 levels at lags of 0, 1, 2, 3, 4 and 5 days for individuals 65 and older. The association was strongest at lag 0 using both GAM (stringent convergence) and GLM.

Hospital Admissions, Chronic Lung Disease (ICD-9 code 490-496) --- Los Angeles County

Moolgavkar (2003, Table 18) shows the estimated percentage change (log RR x100) in daily COPD admissions associated with increases of 10 ppb NO_2 from single-pollutant models in Los Angeles County. The coefficients can be calculated based on the reported percentage changes, that is, $\beta NO_2 = \log(RR)/10ppb$. Along with the t-statistics reported in the same table, standard errors (SE) are calculated. For Los Angeles County $\beta NO_2 = 0.0018$ and SE = 0.00019.

Hospital Admissions, Chronic Lung Disease (ICD-9 code 490-496) — Cook County

Moolgavkar (2003, Table 20) shows the estimated percentage change (log RR x100) in daily COPD admissions associated with increases of 10 ppb NO_2 from single-pollutant

models in Cook County. The coefficient and standard error are calculated in the same way as described above. For Cook County $\beta NO_2 = 0.0024$ and SE = 0.00080.

G.1.5 Yang et al. (2003)

Yang et al.(2003) examined the impact of ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, and coefficient of haze on daily respiratory admissions (ICD-9 codes 460-519) in both young children (<3 years of age) and the elderly (65-99 years of age) in greater Vancouver, British Columbia during the 13-yr period 1986-1998. Bidirectional case-crossover analysis was used to investigate associations and odds ratios were reported for single-pollutant, two-pollutants and multiple pollutants models. NO_2 was found significantly associated with respiratory admissions in all models for elderly. For children NO_2 effect was found significant in single- pollutant model and two pollutants model with O_3.

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

In a multi-pollutant model with SO_2, O_3, CO, and COH, the coefficient for NO_2 and standard error are estimated for age 65-99 from the relative risks (1.05) and 95% confidence interval (1.01-1.08) for a 5.57 ppb increase in daily average of NO_2 at lag 1 day (Yang, et al., 2003, Table 3).

G.1.6 Yang et al. (2005)

Yang et al. (2005) examined the associations between gaseous pollutants and hospitalization for chronic lung disease less asthma (ICD-9 codes 490-492, 494, and 496) among elderly people living in Vancouver, Canada. The authors regressed the logarithm of daily counts of acute COPD hospitalization during the 5-year period from 1994 to 1998 on the daily mean levels of each pollutant, after accounting for seasonal and subseasonal fluctuations, non-Poisson dispersion, and weather variables. They found that nitrogen dioxide and carbon monoxide were significantly associated with hospitalization for COPD, and the magnitude of effects was increased slightly with increasing days of exposure averaging, with the relative risk for a 7-day average being 1.12 (95% CI: 1.04, 1.20) for nitrogen dioxide in a two-pollutant model with O_3. There was no significant association between either sulfur dioxide or ozone and COPD hospitalization. The combined relative risk for all four gaseous pollutants (CO, O_3, NO_2, and SO_2) on COPD hospitalization was 1.21. The effects of gaseous pollutants on COPD hospitalization were not significant after adjustment for PM_{10}, although its inclusion did not have a marked effect on the point estimates for relative risks.

Hospital Admissions, Chronic Lung Disease less Asthma (ICD-9 codes 490-492, 494, and 496)

In a two-pollutant model with O_3, the coefficient and standard error are estimated from the relative risk (1.12) and 95% confidence interval (1.04-1.20) for a 5.5 ppb increase in 7-day average NO_2 levels (Yang, et al., 2005, Table 5).

G.2 Emergency Room Visits

Table G-2 summarizes the health impacts functions used to estimate the relationship between nitrogen dioxide 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-2. Health Impact Functions for Nitrogen Dioxide and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form
Asthma	Ito et al.	2007	NYC	0-99		D24HourMean	0.005460	0.000933	Log-linear
Asthma	NYDOH	2006	Bronx, NYC	0-99	O_3	D24HourMean	0.002264	0.001178	Log-linear
Asthma	Peel et al.	2005	Atlanta, GA	0-99		D1HourMax	0.002296	0.000901	Log-linear
Asthma	Villeneuve et al.	2007	Edmonton, Canada	75-99		D24HourMean	0.013505	0.005345	Logistic

G.2.1 Ito et al. (2007)

Ito et al. (2007) assessed associations between air pollution and asthma emergency department visits in New York City for all ages. Specifically they examined the temporal relationships among air pollution and weather variables in the context of air pollution health effects models. The authors compiled daily data for $PM_{2.5}$, O_3, NO_2, SO_2, CO, temperature, dew point, relative humidity, wind speed, and barometric pressure for New York City for the years 1999-2002.The authors evaluated the relationship between the various pollutants' risk estimates and their respective concurvities, and discuss the limitations that the results imply about the interpretability of multi-pollutant health effects models.

Emergency Room Visits, Asthma (ICD-9 code 493)

In a single-pollutant model, the coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.09-1.19) for a 24 ppb increase in the average of 0- and 1-day lag of NO_2 (Ito, et al., 2007, p. S52).

G.2.2 NYDOH (2006)

New York State Department of Health (NYDOH) investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in Manhattan and Bronx, NYC and compared the magnitude of the air pollution effect between the two communities. NYDOH(2006) used Poisson regression to test for effects of 14 key air contaminants on daily ED visits, with control for temporal cycles, temperature, and day-of-week effects. The core analysis utilized the average exposure for the zero- to four-day lags. Mean daily NO_2 was found significantly associated with asthma ED visits in Bronx but not Manhattan. Their findings of more significant air pollution effects in the Bronx are likely to relate in part to greater statistical power for identifying effects in the

Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx.

Emergency Room Visits, Asthma (ICD-9 code 493)

In a two-pollutant model with O_3, the coefficient and standard error are estimated from the relative risk (1.08) and 95% confidence interval (1.00-1.17) for a 34 ppb increase in the average of 0- to 4-day lags of NO_2 (NYDOH, 2006, Table 9).

G.2.3 Peel et al. (2005)

Peel et al. (2005) examined the associations between air pollution and respiratory emergency department visits (i.e., asthma (ICD-9 code 493, 786.09), COPD (491,492,496), URI (460- 466, 477), pneumonia (480-486), and an all respiratory-disease group) in Atlanta, GA from 1 January 1993 to 31 August 2000. They used 3-Day Moving Average (Lags of 0, 1, and 2 Days) and unconstrained distributed lag (Lags of 0 to 13 Days) in the Poisson regression analyses. In single-pollutant models, the authors found that positive associations persisted beyond 3 days for several outcomes, and over a week for asthma. Standard deviation increases of O_3, NO_2, CO, and PM_{10} were associated with 1-3% increases in URI visits; a 2 $\mu g/m^3$ increase of $PM_{2.5}$ organic carbon was associated with a 3% increase in pneumonia visits; and standard deviation increases of NO_2 and CO were associated with 2-3% increases in chronic obstructive pulmonary disease visits.

Emergency Room Visits, Asthma (ICD-9 code 493)

In the single-pollutant model using unconstrained distributed lag, the coefficient and standard error are estimated from the relative risk (1.047) and 95% confidence interval (1.011-1.085) for a 20 ppb increase in NO_2 (Peel, et al., 2005, Table 4).

G.2.4 Villeneuve et al. (2007)

Villeneuve et al.(2007) examined the associations between air pollution and emergency department (ED) visits for asthma among individuals two years of age and older in the census metropolitan area of Edmonton, Canada between April 1, 1992 and March 31, 2002 using a time stratified case-crossover design. Daily air pollution levels for the entire region were estimated from three fixed-site monitoring stations. Odds ratios and their corresponding 95% confidence intervals were estimated using conditional logistic regression with adjustment for temperature, relative humidity and seasonal epidemic of viral related respiratory disease. Villeneuve et al. (2007) found positive associations for asthma ED visits with outdoor air pollution levels between April and September, but such associations were absent during the remainder of the year. Effects were strongest among young children (2-4 years of age) and elderly (>75 years of age). Air pollution risk estimates were largely unchanged after adjustment for aeroallergen levels.

Emergency Room Visits, Asthma (ICD-9 code 493)

In a single-pollutant model for all seasons, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.04-1.38) for a 13.5 ppb increase in 5-day average of NO_2 (Villeneuve, et al., 2007, Table 9).

G.3 Asthma Exacerbation

Table G-3 summarizes the health impacts functions used to estimate the relationship between nitrogen dioxide 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-3. Health Impact Functions for Nitrogen Dioxide and Asthma Exacerbation

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
One or More Symptoms	Delfino et al.	2002	Southern California	9-18		D8HourMax	0.019939	0.011443	Logistic	
One or More Symptoms	Delfino et al.	2003	Los Angeles	10-16		D8HourMax	0.240337	0.115256	Logistic	Hispanic asthmatics
One or More Symptoms	Mortimer et al.	2002	7 urban areas (US)	4-12	O_3, SO_2	D4HourMean	0.013501	0.011179	Logistic	Warm season; Adjusted age
One or More Symptoms	O'Connor et al.	2008	7 inner cities	4-12	$PM_{2.5}$, O_3	D24HourMean	0.010545	0.004988	Log-linear*	Adjusted age
Nightime asthma	O'Connor et al.	2008	7 inner cities	4-12	$PM_{2.5}$, O_3	D24HourMean	0.012482	0.006488	Log-linear*	Adjusted age
Slow play	O'Connor et al.	2008	7 inner cities	4-12	$PM_{2.5}$, O_3	D24HourMean	0.013979	0.005609	Log-linear*	Adjusted age
Missed school days	O'Connor et al.	2008	7 inner cities	4-12	$PM_{2.5}$, O_3	D24HourMean	0.013979	0.010534	Logistic	Adjusted age
Cough	Ostro et a.	2001	Central Los Angeles, CA	4-12		D1HourMax	0.000591	0.000595	Logistic	African-Americans; Adjusted age
Cough (New Cases)	Ostro et al.	2001	Central Los Angeles, CA	4-12		D1HourMax	0.002267	0.001098	Logistic	African-Americans; Adjusted age
Shortness of Breath	Ostro et al.	2001	Central Los Angeles, CA	4-12		D1HourMax	0.001539	0.000896	Logistic	African-Americans; Adjusted age
Shortness of Breath (New Cases)	Ostro et al.	2001	Central Los Angeles, CA	4-12		D1HourMax	0.002621	0.001429	Logistic	African-Americans; Adjusted age

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Wheeze	Ostro et al.	2001	Central Los Angeles, CA	4-12		D1Hour Max	0.001539	0.000612	Logistic	African-Americans; Adjusted age
Wheeze (New Cases)	Ostro et al.	2001	Central Los Angeles, CA	4-12		D1Hour Max	0.002444	0.000897	Logistic	African-Americans; Adjusted age
One or More Symptoms	Schildcro ut et al.	2006	Eight U.S. cities	4-12		D24Hour Mean	0.004309	0.001406	Logistic	Adjusted age

*O'Connor et al. (2008) did not specify the functional form. Log-linear assumption is made based on the reported results (Table IV on page 1137).

G.3.1 Delfino et al. (2002)

Delfino et al., (2002) examined the association between air pollution and asthma symptoms among 22 asthmatic children (9-19 years of age) followed March through April 1996 (1,248 person-days) in Southern California. Air quality data for PM_{10}, NO_2, O_3, fungi and pollen were used in a logistic model with control for temperature, relative humidity, day-of-week trends and linear time trends. The odds ratio (95% confidence interval) for asthma episodes in relation to lag0 20 ppb changes in 8-hr max NO_2 is 1.49 (0.95-2.33). The authors also considered subgroups of asthmatic children who were on versus not on regularly scheduled anti-inflammatory medications and found that pollutant associations were stronger during respiratory infections in subjects not on anti-inflammatory medications.

Asthma Exacerbation, One or More Symptoms

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.49) and the 95% confidence interval (0.95-2.33) for a 20 ppb increase in 8-hr max NO_2 (Delfino, et al., 2002, Table 4).

Incidence Rate: Delfino et al., (2002, Table 1) reported asthma episodes in the 22 asthmatic children. Asthma episodes are defined as having asthma symptoms that interfered with daily activities (symptom sore >2). The incidence rate is calculated as the ratio of number of person-days that symptom sore >2 and the total number of person-days, i.e., daily asthma episodes incidence rate = 196/1248=0.157

Population: The study population includes asthmatics from 9 to 18 years of age. We treat these as two groups based on the available information from American Lung Association (2010b, Table 7). Asthmatic population ages 9 to 17 = 10.70% of population ages 9 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).

G.3.2 Delfino et al. (2003)

Delfino et al. (2003) conducted a panel study of 22 Hispanic children with asthma who were 10-16 years old and living in a Los Angeles community with high traffic density. Subjects filled out symptom diaries daily for up to 3 months (November 1999 through January 2000). Pollutants included ambient hourly values of ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO) and 24-hr values of volatile organic compounds (VOCs), particulate matter with aerodynamic diameter < 10 microm (PM_{10}), and elemental carbon (EC) and organic carbon (OC) PM_{10} fractions. Asthma symptom severity was regressed on pollutants using logistic models. The authors found positive associations of symptoms with criteria air pollutants (NO_2, SO_2, O_3, and PM_{10}). Selected adjusted odds ratio for more severe asthma symptoms from interquartile range increases in pollutants was, for 1.4 ppb 8-hr max NO_2, 1.40 [95% confidence interval (CI), 1.02-1.92]. Their findings support the view that air toxins in the pollutant mix from traffic and industrial sources may have adverse effects on asthma in children.

Asthma Exacerbation, One or More Symptoms

In a single-pollutant model the coefficient and standard error are based on the odds ratio (95% CI) for more severe asthma symptoms, i.e., odds ratio of 1.40 [95% confidence interval (CI), 1.02-1.92] for a 1.4 ppb increase in 8-hr max NO_2 (Delfino, et al., 2003, Table 4).

Incidence Rate: Daily asthma symptoms incidence rate = 2.3% (Delfino, et al., 2003, Table 1)

Population: Delfino et al. (2003) examined Hispanic asthmatic population, whose prevalence is not available. We use the prevalence of asthmatic population from the American Lung Association (2010b, Table 7) as a proxy. Asthmatic population ages 10 to 16 = 10.70% of population ages 10 to 16. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 at 10.70% (based on data from the 2008 National Health Interview Survey).

G.3.3 Mortimer et al. (2002)

Mortimer et al. (2002) examined the effect of daily ambient air pollution within a cohort of 846 asthmatic children residing in eight urban areas of the USA between June 1 to August 31, 1993, using data from the National Cooperative Inner-City Asthma Study. Daily air pollution concentrations were extracted from the Aerometric Information Retrieval System database from the Environment Protection Agency in the USA. Logistic models were used to evaluate the effects of several air pollutants (O_3, NO_2, SO_2 and PM_{10}) on peak expiratory flow rate (PEFR) and symptoms in 846 children (ages 4-9 yrs) with a history of asthma. In single pollutant models, each pollutant was associated with an increased incidence of morning symptoms: (odds ratio(OR) =1.16 (95% CI 1.02-1.30) per IQR increase in 4-day average O_3, OR=1.32 (95% CI 1.03- 1.70) per IQR increase in 2-day average SO_2, OR=1.48 (95% CI 1.02-2.16) per IQR increase in 6-day average NO_2 and OR=1.26 (95% CI 1.0-1.59) per IQR increase in two-day average PM_{10}. This longitudinal analysis supports previous time-series findings that at levels below current USA air-quality

standards, summer-air pollution is significantly related to symptoms and decreased pulmonary function among children with asthma.

Asthma Exacerbation, One or More Symptoms

In a three-pollutant model with O_3 and SO_2, the coefficient for NO_2 and standard error are based on the odds ratio (95% CI) for morning asthma symptoms, i.e., odds ratio of 1.31 [95% confidence interval (CI), 0.87-2.09] for a 20 ppb increase in 1-6 day average of NO_2 (Delfino, et al., 2003, Table 4).

Incidence Rate: Daily incidence rate for "any morning symptom" is 11.6% (Mortimer, et al., 2002, Table 1).

Population: Asthmatic population ages 4 to 12 = 10.70% of population ages 4 to 12. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 at 10.70% (based on data from the 2008 National Health Interview Survey). Based on the American Lung Association (2010b, Table 7), the estimated asthma prevalence for children under 5 years old at 6.14% (based on data from the 2008 National Health Interview Survey), which is close to the rate of ages 5-17. Therefore we use the rate of population 5-17 as an estimate for asthma prevalence in population ages 4-9 for simplicity.

G.3.4 O'Connor et al. (2008)

O'Connor et al. (2008) investigated the association between fluctuations in outdoor air pollution and asthma exacerbation among 861 inner-city children (5-12 years of age) with asthma in 7 US urban communities. Asthma symptom data were collected every 2 months during the 2-year study period. Daily pollution measurements were obtained from the Aerometric Information Retrieval System between August 1998 and July 2001. The relationship of symptoms to fluctuations in pollutant concentrations was examined by using logistic models. In single-pollutant models, significant or nearly significant positive associations were observed between higher NO_2 concentrations and each of the health outcomes. Significant positive associations with symptoms but not school absence were observed in the single-pollutant model for CO. The O_3, $PM_{2.5}$, and SO_2 concentrations did not appear significantly associated with symptoms or school absence except for a significant association between $PM_{2.5}$ and school absence. The authors concluded that the associations with NO_2 suggest that motor vehicle emissions may be causing excess morbidity in this population.

Asthma Exacerbation, Missed school

In a three-pollutant model with O_3 and $PM_{2.5}$, the coefficient and standard error are estimated from the odds ratio (1.33) and 95% confidence interval (0.87-2.02) for a 20.4 ppb increase in 19-day average of NO_2 (O'Connor, et al., 2008, Table IV). The independent pollution variable was the mean concentration during the 19 days preceding the interview, that is, the 14 days of the symptom recall period plus a 5-day lag period preceding the symptom recall period.

Incidence Rate: Daily incidence rate for missed school = 5.7% (O'Connor, et al., 2008, Table I).

Population: Same as population used in Mortimer et al. (2002). See description above.

Asthma Exacerbation, One or More Symptoms

O'Connor et al. (2008, Table IV) reported a 24% increase in symptom frequency with a 95% confidence interval of 2% to 52% associated with a 20.4 ppb increase in the 19-day average of NO_2.

Incidence Rate: Daily incidence rate for one or more symptoms = 20.7% (O'Connor, et al., 2008, Table I).

Population: See population description above.

Asthma Exacerbation, Nighttime Asthma

O'Connor et al. (2008, Table IV) reported a 29% increase in symptom frequency with a 95% confidence interval of 0% to 68% associated with a 20.4 ppb increase in the 19-day average of NO_2. Incidence Rate: Daily incidence rate for nighttime asthma = 12.1% (O'Connor, et al., 2008, Table I).

Population: See population description above.

Asthma Exacerbation, Slow Play

O'Connor et al. (2008, Table IV) reported a 33% increase in symptom frequency with a 95% confidence interval of 6% to 66% associated with a 20.4 ppb increase in the 19-day average of NO_2.

Incidence Rate: Daily incidence rate for slow play = 15.7% (O'Connor, et al., 2008, Table I).

Population: See population description above.

G.3.5 Ostro et al. (2001)

Ostro et al. (2001) examined relations between several air pollutants and asthma exacerbation in African-Americans children (8 to 13 years old) in central Los Angeles from August to November 1993. Air quality data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 were used in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day with symptoms followed by a symptom-free day. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among

asthmatics. The authors found both the prevalence and incidence of wheeze associated with $PM_{2.5}$, PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics.

Asthma Exacerbation, Cough

The coefficient and standard error are based on an odds ratio of 1.03 (95% CI 0.97-1.09) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 4, p.204).

Incidence Rate: Use daily prevalence of cough reported on p.202 of Ostro et al. (2001) and then take weighted average with weight being the population in the two study locations, i.e., daily cough rate per person = (0.151*115+0.115*23)/ (115+23) = 0.145

Population: Asthmatic African-American population ages 4 to 12 = 17.76% of African-American population ages 4 to 12. The American Lung Association (2010b, Table 9) estimates asthma prevalence for African-American children ages 5 to 17 at 17.76% (based on data from the 2008 National Health Interview Survey). Based on the American Lung Association (2010b, Table 9), the estimated asthma prevalence for African children under 5 years old is 9.98% (based on data from the 2008 National Health Interview Survey), which is close to the rate of ages 5-17. Therefore we use the rate of population 5-17 as an estimate for asthma prevalence in population ages 4-9 for simplicity.

Asthma Exacerbation, Cough (New Cases)

The coefficient and standard error are based on an odds ratio of 1.12 (95% CI 1.00-1.24) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 5, p.204).

Incidence Rate: daily cough incidence rate = 0.067 (Ostro, et al., 2001, p.202).

Population: Asthmatic African-American population ages 4 to 12 = 7.26% of African-American population ages 4 to 12 (described above).

Asthma Exacerbation, Shortness of Breath

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 0.99-1.18) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 4, p.204).

Incidence Rate: Use daily prevalence of shortness of breath reported on p.202 of Ostro et al. (2001) and then take weighted average with weight being the population in the two study locations, i.e., daily shortness of breath rate per person = (0.075*115+0.081*23)/ (115+23) = 0.074

Population: Asthmatic African-American population ages 4 to 12 = 7.26% of African-American population ages 4 to 12 (described above).

Asthma Exacerbation, Shortness of Breath (New Cases)

The coefficient and standard error are based on an odds ratio of 1.14 (95% CI 0.99-1.31) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 5, p.204).

Incidence Rate: daily shortness of breath incidence rate = 0.037 (Ostro, et al., 2001, p.202)

Population: Asthmatic African-American population ages 4 to 12 = 7.26% of African-American population ages 4 to 12 (described above).

Asthma Exacerbation, Wheeze

The coefficient and standard error are based on an odds ratio of 1.08 (95% CI 1.02-1.15) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 4, p.204).

Incidence Rate: Use daily prevalence of wheeze reported on p.202 of Ostro et al. (2001) and then take weighted average with weight being the population in the two study locations, i.e., daily wheeze rate per person = (0.173*115+0.172*23)/ (115+23) = 0.173

Population: Asthmatic African-American population ages 4 to 12 = 7.26% of African-American population ages 4 to 12 (described above).

Asthma Exacerbation, Wheeze (New Cases)

The coefficient and standard error are based on an odds ratio of 1.13 (95% CI 1.04-1.24) for a 50 ppb increase in 1-h max NO_2 concentration (Ostro, et al., 2001, Table 5, p.204).

Incidence Rate: daily wheeze incidence rate = 0.076 (Ostro, et al., 2001, p.202)

Population: Asthmatic African-American population ages 4 to 12 = 7.26% of African-American population ages 4 to 12 (described above).

G.3.6 Schildcrout et al. (2006)

Schildcrout et al.(2006) investigated the relation between ambient concentrations of the five criteria pollutants (PM_{10}, O_3, NO_2, SO_2, 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, NO_2, PM_{10}, SO_2, and warm-season O_3 were examined in both one-pollutant and two-pollutant

models, using lags of up to 2 days in logistic and Poisson regressions. Lags in CO and NO_2 were positively associated with both measures of asthma exacerbation, and the 3-day moving sum of SO_2 levels was marginally related to asthma symptoms. PM_{10} and O_3 were unrelated to exacerbations. The strongest effects tended to be seen with 2-day lags, where a 1-parts-per-million change in CO and a 20-parts-per-billion change in NO_2 were associated with symptom odds ratios of 1.08 (95% confidence interval (CI): 1.02, 1.15) and 1.09 (95% CI: 1.03, 1.15), respectively.

Asthma Excerbation, One or More Symptoms

In a single-pollutant model, Schildcrout et al.(2006, Figure 1) reported an odds ratio of 1.09 (95% CI: 1.03, 1.15) for daily asthma symptoms associated with 20 ppb change in 24-hr mean of NO_2 at lag 2.

Incidence Rate: Daily incidence rate for one or more symptoms (symptom score>0) = 52% (Schildcrout, et al., 2006, Table 1)

Population: Asthmatic population ages 4 to 12 = 10.70% of population ages 4 to 12. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5-17 at 10.70% (based on data from the 2008 National Health Interview Survey).

G.4 Minor Effects

Table G-4 summarizes the health impacts function used to estimate the relationship between nitrogen dioxide and minor effects. Below, we present a brief summary of the study and any items that are unique to the study.

Table G-4. Health Impact Functions for Nitrogen Dioxide and Minor Effects

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Cough	Schwartz et al.	1994	Six U.S. cities	7-14	PM_{10}	D24Hour Mean	0.015700	0.011232	Logistic	Warm season

G.4.1 Schwartz et al. (1994)

Schwartz et al. (1994) studied the association between ambient air pollution exposures and respiratory illness among 1,844 school children (7-14 years of age) in six U.S. cities during five warm seasons months between April and August. Daily measurements of ambient sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), inhalable particles (PM_{10}), respirable particles ($PM_{2.5}$), light scattering, and sulfate particles were made, along with integrated 24-h measures of aerosol strong acidity. Significant associations in single pollutant models were found between SO_2, NO_2, or $PM_{2.5}$ and incidence of cough, and between sulfur dioxide and incidence of lower respiratory symptoms. Significant associations were also found between incidence of coughing symptoms and

incidence of lower respiratory symptoms and PM_{10}, and a marginally significant association between upper respiratory symptoms and PM_{10}.

Acute Respiratory Symptoms, Cough

In a two-pollutant model with PM_{10}, the coefficient and standard error are based on an odds ratio of 1.17 (95% CI 0.94-1.46) for a 10 ppb increase in the lag 1-4 days average of NO_2 concentration (Schwartz, et al., 1994, Table 4).

Incidence Rate: The proposed incidence rate, 0.416 percent, is based on the percentiles in Schwartz et al. (1994, Table 2). The authors did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated per person per day values are 10th = 0 percent, 25th = 0 percent, 50th = 0.31 percent, 75th = 0.58 percent, and 90th = 0.86 percent. The most conservative estimate consistent with the data is to assume the incidence is zero up to the 50th percentile, a constant 0.31 percent between the 50th and 75th percentiles, and a constant 0.58 percent between the 75th and 90th percentiles, a constant 0.86 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 is 0.461 percent, which is used in this analysis.

Population: Population of ages 7 to 14

Appendix H: Sulfur Dioxide Health Impact Functions in U.S. Setup

In this Appendix, we present the health impact functions used to estimate SO_2-related adverse health effects. 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.

H.1 Hospital Admissions

Table H-1 summarizes the health impacts functions used to estimate the relationship between sulfur dioxide and hospital admissions. Below, we present a brief summary of each of the studies and any items that are unique to the study.

Table H-1. Health Impact Functions for Sulfur Dioxide and Hospital Admissions

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
All Respiratory	Fung et al.	2006	Vancouver, Canada	65-99		D24Hour Mean	0.017224	0.005084	Log-linear	
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-99		D1HourMax	0.002336	0.001671	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-99		D1HourMax	-0.000670	0.001718		Male
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-14		D1HourMax	0.005468	0.002501	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	0-14		D1HourMax	-0.000260	0.002552	Log-linear	Male
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	15-64		D1HourMax	0.003418	0.003118	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	15-64		D1HourMax	0.002336	0.004039	Log-linear	Male
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	65-99		D1HourMax	0.003709	0.003274	Log-linear	Female
All Respiratory	Luginaah et al.	2005	Windsor, Ontario	65-99		D1HourMax	-0.002332	0.003620	Log-linear	Male
All Respiratory	Schwartz et al.	1996	Cleveland, Ohio	65-99		D24Hour Mean	0.000769	0.000453	Log-linear	
Asthma	Sheppard, L.	2003	Seattle, WA	0-64		D24Hour Mean	0.002031	0.002591	Log-linear	
All Respiratory	Yag et al.	2003	Vancouver, Canada	65-99	NO_2, O_3, CO, COH	D24Hour Mean	0.002843	0.003627	Logistic	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Chronic Lung Disease (less Asthma)	Yang et al.	2005	Vancouver, Canada	65-99	O_3	D24Hour Mean	0.024164	0.011938	Log-linear	

H.1.1 Fung et al. (2006)

Fung et al. (2006) assessed the impact of ambient gaseous pollutants (SO_2, NO_2, CO, and O_3) and particulate matters (PM_{10}, $PM_{2.5}$, and PM_{10}-2.5) as well as the coefficient of haze (COH) on recurrent respiratory hospital admissions (ICD-9 codes 460-519) among the elderly in Vancouver, Canada, for the period of June 1, 1995, to March 31, 1999, using a new method proposed by Dewanji and Moolgavkar(2000; 2002). The authors found significant associations between respiratory hospital admissions and 3-day, 5-day, and 7-day moving averages of the ambient SO_2 concentrations, with the strongest association observed at the 7-day lag (RR = 1.044, 95% CI: 1.018-1.070). The authors also found PM_{10}-2.5 for 3-day and 5-day lag to be significant, with the strongest association at 5-day lag (RR = 1.020, 95% CI: 1.001-1.039). No significant associations with admission were found with current day exposure.

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

In a single-pollutant model the coefficient and standard error are estimated from the relative risk (1.044) and 95% confidence interval (95% CI: 1.018-1.070) for a 2.5 ppb increase in 7-day moving average of SO_2 (Fung, et al., 2006, Table 4).

H.1.2 Luginaah et al. (2005)

Luginaah et al. (2005) assessed the association between air pollution and daily respiratory hospitalization (ICD-9 codes 460-519) for different age and sex groups from 1995 to 2000. The pollutants included were NO_2, SO_2, CO, O_3, PM_{10}, coefficient of haze (COH), and total reduced sulfur (TRS). The authors estimated relative risks (RR) using both time-series and case-crossover methods after controlling for appropriate confounders (temperature, humidity, and change in barometric pressure). The results of both analyses were consistent. They found associations between NO_2, SO_2, CO, COH, or PM_{10} and daily hospital admission of respiratory diseases especially among females. For females 0-14 years of age, there was 1-day delayed effect of NO_2 (RR = 1.19, case-crossover method), a current-day SO_2 (RR = 1.11, time series), and current-day and 1- and 2-day delayed effects for CO by case crossover (RR = 1.15, 1.19, 1.22, respectively). Time-series analysis showed that 1-day delayed effect of PM_{10} on respiratory admissions of adult males (15-64 years of age), with an RR of 1.18. COH had significant effects on female respiratory hospitalization, especially for 2-day delayed effects on adult females, with RRs of 1.15 and 1.29 using time-series and case-crossover analysis, respectively. There were no significant associations between O_3 and TRS with respiratory admissions.

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

For different age and sex groups, the coefficient and standard error are estimated from the relative risks and 95% confidence interval reported for single-pollutant models using time-series method for a 19.25 ppb increase in daily 1-hour max SO_2 levels (Luginaah, et al., 2005, Table 3). The time-series method is chosen based on the discussion in Fung et al. (2003)

H.1.3 Schwartz et al. (1996)

Schwartz et al. (1996) is a review paper with an example drawn from hospital admissions of the elderly in Cleveland, Ohio from 1988-1990. The authors argued that the central issue is control for seasonality. They illustrated the use of categorical variables for weather and sinusoidal terms for filtering season in the Cleveland example. After controlling for season, weather, and day of the week effects, hospital admissions of persons aged 65 and older in Cleveland for respiratory illness was associated with ozone (RR = 1.09, 95% CI 1.02, 1.16) and PM_{10} (RR = 1.12, 95% CI 1.01, 1.24), and marginally associated with SO_2 (RR = 1.03, 95% CI = 0.99, 1.06). All of the relative risks are for a 100 micrograms/m^3 increase in the pollutant.

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

Schwartz et al.(1996, Table 2) reported the relative risk (1.03) with the 95% confidence interval (0.99-1.06) from Poisson regression models for a 100µg/ m^3 increase in average of lag0-1 day of SO_2. In order to obtain the coefficient and standard error, a conversion of 0.3846 ppb per µg/m^3 was used, based on http://www.epa.gov/air/criteria.html.

H.1.4 Sheppard (2003)

Sheppard (2003) reanalyzed data from Sheppard et al.(1999) on nonelderly (0-64 years of age) hospital admissions for asthma in Seattle, Washington, to evaluate the effect of the fitting procedure (GAM/Splus issue). The author found that the effect estimates were slightly lower when more stringent convergence criteria were used in the GAM and an additional small reduction in the estimates was found when generalized linear models (GLMs) with natural splines were used instead.

Hospital Admissions, Asthma (ICD-9 code 493)

Sheppard (2003, p. 229) reported the re-calculated relative risk (1.01) and 95% confidence interval (0.98-1.03) for a 4.9 ppb increase in the daily average of same day SO_2 (lag 0).

H.1.5 Yang et al. (2003)

Yang et al. (2003) examined the impact of ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide, and coefficient of haze on daily respiratory admissions (ICD-9 codes 460-519) in both young children (<3 years of age) and the elderly (65-99 years of age)

in greater Vancouver, British Columbia during the 13-yr period 1986-1998. Bidirectional case-crossover analysis was used to investigate associations and odds ratios were reported for single-pollutant, two-pollutant and multiple-pollutant models. Sulfur dioxide was found marginally significant in all models for elderly.

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

In a multi-pollutant model with NO_2, O_3, CO, and COH, the coefficient for SO_2 and standard error are estimated for age 65-99 from the relative risk (1.01) and 95% confidence interval (0.98-1.03) for a 5.57 ppb increase in daily average of same day SO_2 (Yang, et al., 2003, Table 3).

H.1.6 Yang et al. (2005)

Yang et al. (2005) examined the associations between gaseous pollutants and hospitalization for chronic lung disease less asthma (ICD-9 codes 490-492, 494, and 496) among elderly people living in Vancouver, Canada. The authors regressed the logarithm of daily counts of acute COPD hospitalization during the 5-year period from 1994 to 1998 on the daily mean levels of each pollutant, after accounting for seasonal and subseasonal fluctuations, non-Poisson dispersion, and weather variables. They found that nitrogen dioxide and carbon monoxide were significantly associated with hospitalization for COPD, and the magnitude of effects was increased slightly with increasing days of exposure averaging, with the relative risk for a 7-day average being 1.12 (95%CI: 1.04, 1.20) for nitrogen dioxide in a two-pollutant model with O_3. Sulfur dioxide was found marginally significant in a two-pollutant model with ozone. The combined relative risk for all four gaseous pollutants (CO, O_3, NO_2, and SO_2) on COPD hospitalization was 1.21. The effects of gaseous pollutants on COPD hospitalization were not significant after adjustment for PM_{10}, although its inclusion did not have a marked effect on the point estimates for relative risks.

Hospital Admissions, Chronic Lung Disease less Asthma (ICD-9 codes 490-492, 494, and 496)

In a two-pollutant model with O_3, the coefficient and standard error are estimated from the relative risk (1.07) and 95% confidence interval (1.00-1.14) for a 2.8 ppb increase in 7-day average of SO_2 (Yang, et al., 2005, Table 5).

H.2 Emergency Room Visits

Table H-2 summarizes the health impacts functions used to estimate the relationship between sulfur dioxide 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 H-2. Health Impact Functions for Sulfur Dioxide and Emergency Room Visits

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Asthma	Ito et al.	2007	NYC	0-99		D24Hour Mean	0.030692	0.005439	Log-linear	Warm season
Asthma	Ito et al.	2007	NYC	0-99		D24Hour Mean	0.004372	0.001702	Log-linear	Allyear
Asthma	Michaud et al.	2004	Hilo, Hawaii	0-99		D24Hour Mean	0.002956	0.002386	Log-linear	
Asthma	NYDOH	2006	Bronx, NYC	0-4		D24Hour Mean	0.011111	0.005129	Log-linear	
Asthma	NYDOH	2006	Bronx, NYC	0-99		D24Hour Mean	0.006996	0.003641	Log-linear	Male
Asthma	NYDOH	2006	Bronx, NYC	0-99		D24Hour Mean	0.011912	0.003450	Log-linear	Female
Asthma	NYDOH	2006	Bronx, NYC	0-99	$PM_{2.5}$	D24Hour Mean	0.009487	0.002929	Log-linear	
Asthma	NYDOH	2006	Bronx, NYC	0-99	NO_2	D24Hour Mean	0.009487	0.002732	Log-linear	
Asthma	NYDOH	2006	Bronx, NYC	0-99	O_3	D24Hour Mean	0.009487	0.002510	Log-linear	
Asthma	NYDOH	2006	Bronx, NYC	35-64		D24Hour Mean	0.015047	0.004515	Log-linear	
Asthma	Peel et al.	2005	Atlanta, GA	0-99		D1HourMax	0.000744	0.001030	Log-linear	
Asthma	Villeneuve et al.	2007	Edmonton, Canada	2-99		D24Hour Mean	-0.006734	0.002617	Logistic	1-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	2-4		D24Hour Mean	0.000000	0.008510	Logistic	0-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	5-14		D24Hour Mean	-0.006734	0.006945	Logistic	1-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	15-44		D24Hour Mean	-0.006734	0.004317	Logistic	1-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	45-64		D24Hour Mean	-0.003350	0.007697	Logistic	1-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	65-74		D24Hour Mean	0.003317	0.012590	Logistic	0-day lag
Asthma	Villeneuve et al.	2007	Edmonton, Canada	75-99		D24Hour Mean	0.019423	0.027751	Logistic	5-day average
Asthma	Wilson et al.	2005	Portland, ME	0-14		D24Hour Mean	0.005000	0.009000	Log-linear	
Asthma	Wilson et al.	2005	Manchester, NH	0-14		D24Hour Mean	0.018000	0.011000	Log-linear	
Asthma	Wilson et al.	2005	Portland, ME	15-64		D24Hour Mean	0.011000	0.005000	Log-linear	
Asthma	Wilson et al.	2005	Manchester, NH	15-64		D24Hour Mean	0.003000	0.006000	Log-linear	
Asthma	Wilson et al.	2005	Portland, ME	65-99		D24Hour Mean	0.011000	0.014000	Log-linear	

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Asthma	Wilson et al.	2005	Manchester, NH	65-99		D24Hour Mean	0.011000	0.023000	Log-linear	
Asthma	Wilson et al.	2005	Portland, ME	0-99		D24Hour Mean	0.010000	0.009000	Log-linear	
Asthma	Wilson et al.	2005	Manchester, NH	0-99		D24Hour Mean	0.006000	0.011000	Log-linear	

H.2.1 Ito et al. (2007)

Ito et al. (2007) assessed associations between air pollution and asthma emergency department visits in New York City for all ages. Specifically they examined the temporal relationships among air pollution and weather variables in the context of air pollution health effects models. The authors compiled daily data for $PM_{2.5}$, O_3, NO_2, SO_2, CO, temperature, dew point, relative humidity, wind speed, and barometric pressure for New York City for the years 1999-2002.The authors evaluated the relationship between the various pollutants' risk estimates and their respective concurvities, and discuss the limitations that the results imply about the interpretability of multi-pollutant health effects models.

Emergency Room Visits, Asthma (ICD-9 code 493)

In a single-pollutant model for warm season, the coefficient and standard error are estimated from the relative risk (1.20) and 95% confidence interval (1.13-1.28) for a 6 ppb increase in the average of 0- and 1-day lag of SO_2 (Ito, et al., 2007, through contacting the author).

H.2.2 Michaud et al. (2004)

Michaud et al. (2004) examined the association of emergency department (ED) visits in Hilo, Hawai'i, from January 1997 to May 2001 with volcanic fog, or "vog", measured as sulfur dioxide (SO_2) and submicrometer particulate matter (PM1). Log-linear regression models were used with robust standard errors. The authors studied four diagnostic groups: asthma/COPD; cardiac; flu, cold, and pneumonia; and gastroenteritis. Before adjustments, highly significant associations with vog-related air quality were seen for all diagnostic groups except gastroenteritis. After adjusting for month, year, and day of the week, only asthma/COPD had consistently positive associations with air quality. They found that the strongest associations were for SO_2 with a 3-day lag (6.8% per 10 ppb; P=0.001) and PM1, with a 1-day lag (13.8% per 10 $\mu g/m^3$; P=0.011).

Emergency Room Visits, Asthma (ICD-9 codes 493 and 495)

In a single-pollutant model adjusted for month, year and day of the week, the coefficient and standard error are estimated from the relative risks (1.03) and 95% confidence interval (1.02- 1.12) for a 10 ppb increase in daily average of SO_2 at lag 3-day (Michaud, et al., 2004, Table 3).

H.2.3 NYDOH (2006)

New York State Department of Health (NYDOH) investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in Manhattan and Bronx, NYC and compared the magnitude of the air pollution effect between the two communities. NYDOH (2006) used Poisson regression to test for effects of 14 key air contaminants on daily ED visits, with control for temporal cycles, temperature, and day-of-week effects. The core analysis utilized the average exposure for the 0- to 4-day lags. Mean daily SO_2 was found significantly associated with asthma ED visits in Bronx but not Manhattan. Their findings of more significant air pollution effects in the Bronx are likely to relate in part to greater statistical power for identifying effects in the Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx.

Emergency Room Visits, Asthma (ICD-9 code 493) ---Bronx

Function for Children (0-4 years of age)

In a single pollutant model, the coefficient and standard error are estimated from the relative risk (1.13) and 95% confidence interval (1.01-1.26) for a 11 ppb increase in the average of 0- to 4-day lags of SO_2 (NYDOH, 2006, Table 13).

Function for Adults (35-64 years of age)

In a single pollutant model, the coefficient and standard error are estimated from the relative risk (1.18) and 95% confidence interval (1.07-1.30) for a 11 ppb increase in the average of 0- to 4- day lags of SO_2 (NYDOH, 2006, Table 13).

Function for Females (All ages)

In a single pollutant model, the coefficient and standard error are estimated from the relative risk (1.14) and 95% confidence interval (1.06-1.23) for a 11 ppb increase in the average of 0- to 4- day lags of SO_2 (NYDOH, 2006, Table 12).

Function for Males (All ages)

In a single pollutant model, the coefficient and standard error are estimated from the relative risk (1.08) and 95% confidence interval (1.00-1.17) for a 11 ppb increase in the average of 0- to 4-day lags of SO_2 (NYDOH, 2006, Table 12).

Functions for All Population

We use results from three different two-pollutant models for all population.

In a two-pollutant model with O_3, the coefficient and standard error are estimated from the relative risk (1.11) and 95% confidence interval (1.05-1.17) for a 11 ppb increase in the average of 0- to 4-day lags of SO_2 (NYDOH, 2006, Table 9).

In a two-pollutant model with $PM_{2.5}$, the coefficient and standard error are estimated from the relative risk (1.11) and 95% confidence interval (1.04-1.18) for a 11 ppb increase in the average of 0- to 4-day lags of SO_2 (NYDOH, 2006, Table 9).

In a two-pollutant model with NO_2, the coefficient and standard error are estimated from the relative risk (1.11) and 95% confidence interval (1.04-1.17) for a 11 ppb increase in the average of 0- to 4-day lags of SO_2 (NYDOH, 2006, Table 9).

H.2.4 Peel et al. (2005)

Peel et al. (2005) examined the associations between air pollution and respiratory emergency department visits (i.e., asthma (ICD-9 code 493, 786.09), COPD (491,492,496), URI (460- 466, 477), pneumonia (480-486), and an all respiratory-disease group) in Atlanta, GA from 1 January 1993 to 31 August 2000. They used 3-Day Moving Average (Lags of 0, 1, and 2 Days) and unconstrained distributed lag (Lags of 0 to 13 Days) in the Poisson regression analyses. In single-pollutant models, positive associations persisted beyond 3 days for several outcomes, and over a week for asthma. The effects of NO_2, CO or PM_{10} on asthma ED visits were found significant but SO_2 or O_3 were not significantly associated with asthma ED visits.

Emergency Room Visits, Asthma (ICD-9 code 493)

In the single-pollutant model using unconstrained distributed lag, the coefficient and standard error are estimated from the relative risk (1.015) and 95% confidence interval (0.975-1.057) for a 20 ppb increase in 1-hour max SO_2 (Peel, et al., 2005, Table 4).

H.2.5 Villeneuve et al. (2007)

Villeneuve et al.(2007) examined the associations between air pollution and emergency department (ED) visits for asthma among individuals two years of age and older in the census metropolitan area of Edmonton, Canada between April 1, 1992 and March 31, 2002 using a time stratified case-crossover design. Daily air pollution levels for the entire region were estimated from three fixed-site monitoring stations. Odds ratios and their corresponding 95% confidence intervals were estimated using conditional logistic regression with adjustment for temperature, relative humidity and seasonal epidemic of viral related respiratory disease. Villeneuve et al. (2007) found positive associations for asthma ED visits with outdoor air pollution levels between April and September, but such associations were absent during the remainder of the year. Effects were strongest among young children (2-4 years of age) and elderly (>75 years of age). Air pollution risk estimates were largely unchanged after adjustment for aeroallergen levels.

Emergency Room Visits, Asthma (ICD-9 code 493)

In single-pollutant models for all seasons, the coefficients and standard errors for different age groups are estimated from the relative risks and 95% confidence intervals for a 3 ppb increase in daily average of SO_2 (Villeneuve, et al., 2007, Table3-9).

H.2.6 Wilson et al. (2005)

Daily emergency room (ER) visits for all respiratory (ICD-9 codes 460-519) and asthma (ICD-9 code 493) were compared with daily SO_2, O_3, and weather variables over the period 1998- 2000 in Portland, Maine and 1996-2000 in Manchester, New Hampshire. Seasonal variability was removed from all variables using nonparametric smoothed function (LOESS). Wilson et al. (2005) used generalized additive models to estimate the effect of elevated levels of pollutants on ER visits. Relative risks of pollutants were reported over their inter-quartile range (IQR, the 75th - 25th percentile pollutant values). In Portland, an IQR increase in SO_2 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 O_3 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 SO_2 and O_3 have a significant impact on public health in Portland, Maine.

Emergency Room Visits, Asthma

The coefficients and standard errors for different age groups in the two cities are taken from Wilson et al. (2005, Table 5).

H.3 Asthma Exacerbation

Table H-3 summarizes the health impacts functions used to estimate the relationship between sulfur dioxide and asthma exacerbation. Below, we present a brief summary of each of the studies and any items that are unique to the study.

Table H-3. Health Impact Functions for Sulfur Dioxide and Asthma Exacerbation

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
One or More Symptoms	Delfino et al.	2003	Los Angeles	10-15		D8Hour Max	0.122994	0.046891	Logistic	Hispanic children
One or More Symptoms	Mortimer et al.	2002	7 urban areas (US)	4-12	O_3, NO_2	D3Hour Mean	0.008698	0.003515	Logistic	Warm season; Adjusted age
Missed school days	O'Connor et al.	2008	7 inner cities	4-12		D24Hour Mean	0.009856	0.015289	Logistic	Adjusted age
One or More Symptoms	O'Connor et al.	2008	7 inner cities	4-12		D24Hour Mean	0.004699	0.008263	Log-linear*	Adjusted age
Nighttime asthma	O'Connor et al.	2008	7 inner cities	4-12		D24Hour Mean	0.010567	0.010042	Log-linear*	Adjusted age
Slow play	O'Connor et al.	2008	7 inner cities	4-12		D24Hour Mean	0.005456	0.009517	Log-linear*	Adjusted age
One or More Symptoms	Schildcrout et al.	2006	Eight U.S. cities	4-12		D24Hour Mean	0.003922	0.001963	Logistic	Adjusted age

H.3.1 Delfino et al. (2003)

Delfino et al.(2003) conducted a panel study of 22 Hispanic children with asthma who were 10-16 years old and living in a Los Angeles community with high traffic density. Subjects filled out symptom diaries daily for up to 3 months (November 1999 through January 2000). Pollutants included ambient hourly values of ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO) and 24-hr values of volatile organic compounds (VOCs), particulate matter with aerodynamic diameter < 10 microm (PM_{10}), and elemental carbon (EC) and organic carbon (OC) PM_{10} fractions. Asthma symptom severity was regressed on pollutants using logistic models. The authors found positive associations of symptoms with criteria air pollutants (NO_2, SO_2, O_3,and PM_{10}). Selected adjusted odds ratio for more severe asthma symptoms from interquartile range increases in pollutants was, for 2.5 ppb 8-hr max SO_2, 1.36 [95% confidence interval (CI), 1.08-1.71]. Their findings support the view that air toxins in the pollutant mix from traffic and industrial sources may have adverse effects on asthma in children.

Asthma Exacerbation, One or More Symptoms

In a single-pollutant model, the coefficient and standard error are based on the odds ratio (95% CI) for more severe asthma symptoms, i.e., odds ratio of 1.36 [95% confidence interval (CI), 1.08-1.71] for a 2.5 ppb increase in 8-hr max SO_2 (Delfino, et al., 2003, Table 4).

Incidence Rate: Daily asthma symptoms incidence rate = 2.3% (Delfino, et al., 2003, Table 1)

Population: Delfino et al.(2003) examined Hispanic asthmatic population, whose prevalence is not available. We use the prevalence of asthmatic population from the American Lung Association (2010b, Table 7) as a proxy. Asthmatic population ages 10 to 16 = 10.70% of population ages 10 to 16. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 at 10.70% (based on data from the 2008 National Health Interview Survey).

H.3.2 Mortimer et al. (2002)

Mortimer et al. (2002) examined the effect of daily ambient air pollution within a cohort of 846 asthmatic children residing in eight urban areas of the USA between June 1 to August 31, 1993, using data from the National Cooperative Inner-City Asthma Study. Daily air pollution concentrations were extracted from the Aerometric Information Retrieval System database from the Environment Protection Agency in the USA. Logistic models were used to evaluate the effects of several air pollutants (O_3, NO_2, SO_2 and PM_{10}) on peak expiratory flow rate (PEFR) and symptoms in 846 children (ages 4-9 yrs) with a history of asthma. In single pollutant models, each pollutant was associated with an increased incidence of morning symptoms: (odds ratio(OR) =1.16 (95% CI 1.02-1.30) per IQR increase in 4-day average O_3, OR=1.32 (95% CI 1.03-1.70) per IQR increase in 2-day average SO_2, OR=1.48 (95% CI 1.02-2.16) per IQR increase in 6-day

average NO_2 and OR=1.26 (95% CI 1.0-1.59) per IQR increase in two-day average PM_{10}. This longitudinal analysis supports previous time-series findings that at levels below current USA air-quality standards, summer-air pollution is significantly related to symptoms and decreased pulmonary function among children with asthma.

Asthma Exacerbation, One or More Symptoms

In a three-pollutant model with O_3 and NO_2, the coefficient for SO_2 and standard error are based on the odds ratio (95% CI) for morning asthma symptoms, i.e., odds ratio of 1.19 [95% confidence interval (CI), 1.04-1.37] for a 20 ppb increase in the average of lag 1-2 day SO_2 (Delfino, et al., 2003, Table 4).

Incidence Rate: Daily incidence rate for "any morning symptom" is 11.6% (Mortimer, et al., 2002, Table 1).

Population: Asthmatic population ages 4 to 12 = 10.70% of population ages 4 to 12. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 at 10.70% (based on data from the 2008 National Health Interview Survey). Based on the American Lung Association (2010b, Table 7), the estimated asthma prevalence for children under 5 years old at 6.14% (based on data from the 2008 National Health Interview Survey), which is close to the rate of ages 5-17. Therefore we use the rate of population 5-17 as an estimate for asthma prevalence in population ages 4-9 for simplicity.

H.3.3 O'Connor et al. (2008)

O'Connor et al. (2008) investigated the association between fluctuations in outdoor air pollution and asthma exacerbation (wheeze-cough, nighttime asthma, slow play and school absence) among 861 inner-city children (5-12 years of age) with asthma in 7 US urban communities. Asthma symptom data were collected every 2 months during the 2-year study period. Daily pollution measurements were obtained from the Aerometric Information Retrieval System between August 1998 and July 2001. The relationship of symptoms to fluctuations in pollutant concentrations was examined by using logistic models. In single-pollutant models, significant or nearly significant positive associations were observed between higher NO_2 concentrations and each of the health outcomes. Significant positive associations with symptoms but not school absence were observed in the single-pollutant model for CO. The O_3, $PM_{2.5}$, and SO_2 concentrations did not appear significantly associated with symptoms or school absence except for a significant association between $PM_{2.5}$ and school absence. The authors concluded that the associations with NO_2 suggest that motor vehicle emissions may be causing excess morbidity in this population.

Asthma Exacerbation, Missed school

In a single-pollutant model, the coefficient and standard error are estimated from the odds ratio (1.13) and 95% confidence interval (0.78-1.64) for a 12.4 ppb increase in 19-day average of SO_2 (O'Connor, et al., 2008, Table IV). The independent pollution variable was the mean concentration during the 19 days preceding the interview, that is, the 14

days of the symptom recall period plus a 5-day lag period preceding the symptom recall period. Incidence Rate: Daily incidence rate for missed school = 5.7% (O'Connor, et al., 2008, Table I).

Population: Same as population used in Mortimer et al. (2002). See description above.

Asthma Exacerbation, One or More Symptoms (Wheeze-cough)

O'Connor et al. (2008, Table IV) reported a 6% increase in symptom frequency with a 95% confidence interval of -13% to 30% associated with a 12.4 ppb increase in the 19-day average of SO_2.

Incidence Rate: Daily incidence rate for one or more symptoms = 20.7% (O'Connor, et al., 2008, Table I).

Population: See population description above.

Asthma Exacerbation, Nighttime Asthma

O'Connor et al. (2008, Table IV) reported a 14% increase in symptom frequency with a 95% confidence interval of -11% to 45% associated with a 12.4 ppb increase in the 19-day average of SO_2.

Incidence Rate: Daily incidence rate for nighttime asthma = 12.1% (O'Connor, et al., 2008, Table I).

Population: See population description above.

Asthma Exacerbation, Slow Play

O'Connor et al. (2008, Table IV) reported a 7% increase in symptom frequency with a 95% confidence interval of -15% to 35% associated with a 12.4 ppb increase in the 19-day average of SO_2.

Incidence Rate: Daily incidence rate for slow play = 15.7% (O'Connor, et al., 2008, Table I).

Population: See population description above.

H.3.4 Schildcrout et al. (2006)

Schildcrout et al. (2006) investigated the relation between ambient concentrations of the five criteria pollutants (PM_{10}, O_3, NO_2, SO_2, 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, NO_2, PM_{10}, SO_2, and warm-season O_3 were examined in both one-pollutant and two-pollutant models, using lags of up to 2 days in logistic and Poisson regressions. Lags in CO and

NO_2 were positively associated with both measures of asthma exacerbation, and the 3-day moving sum of SO_2 levels was marginally related to asthma symptoms. PM_{10} and O_3 were unrelated to exacerbations. The strongest effects tended to be seen with 2-day lags, where a 1-parts-per-million change in CO and a 20-parts-per-billion change in NO_2 were associated with symptom odds ratios of 1.08 (95% confidence interval (CI): 1.02, 1.15) and 1.09 (95% CI: 1.03, 1.15), respectively.

Asthma Exacerbation, One or More Symptoms

In a single-pollutant model, Schildcrout et al., (2006, Figure 1) reported an odds ratio of 1.04 (95% CI: 1.00, 1.08) for daily asthma symptoms associated with 10 ppb change in 24-hr mean of SO_2.

Incidence Rate: Daily incidence rate for one or more symptoms (symptom score>0) = 52% (Schildcrout, et al., 2006, Table 1)

Population: Asthmatic population ages 4 to 12 = 10.70% of population ages 4 to 12. The American Lung Association (2010b, Table 7) estimates asthma prevalence for children 5- 17 at 10.70% (based on data from the 2008 National Health Interview Survey).

H.4 Minor Effects

Table H-4 summarizes the health impacts function used to estimate the relationship between sulfur dioxide and minor effects. Below, we present a brief summary of the study and any items that are unique to the study.

Table H-4. Health Impact Functions for Sulfur Dioxide and Minor Effects

Effect	Author	Year	Location	Age	Co-Poll	Metric	Beta	Std Err	Functional Form	Notes
Cough	Schwartz et al.	1994	Six U.S. cities	7-14	PM_{10}	D24Hour Mean	0.008618	0.007429	Logistic	Warm season

H.4.1 Schwartz et al. (1994)

Schwartz et al. (1994) studied the association between ambient air pollution exposures and respiratory illness among 1,844 school children (7-14 years of age) in six U.S. cities during five warm season months between April and August. Daily measurements of ambient sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), respirable particles ($PM_{2.5}$), inhalable particles (PM_{10}), light scattering, and sulfate particles were made, along with integrated 24-hour measures of aerosol strong acidity. Significant associations in single pollutant models were found between SO_2, NO_2, or $PM_{2.5}$ and incidence of cough, and between sulfur dioxide and incidence of lower respiratory symptoms. Significant associations were also found between incidence of coughing symptoms and incidence of lower respiratory symptoms and PM_{10}, and a marginally significant association between upper respiratory symptoms and PM_{10}.

Acute Respiratory Symptoms, Cough

In a two-pollutant model with PM_{10}, the coefficient and standard error are based on an odds ratio of 1.09 (95% CI 0.94-1.3) for a 10 ppb increase in the lag 1-4 days average of SO_2 concentration (Schwartz, et al., 1994, Table 4).

Incidence Rate: The proposed incidence rate, 0.416 percent, is based on the percentiles in Schwartz et al. (1994, Table 2). The authors did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated per person per day values are 10th = 0 percent, 25th = 0 percent, 50th = 0.31 percent, 75th = 0.58 percent, and 90th = 0.86 percent. The most conservative estimate consistent with the data is to assume the incidence is zero up to the 50th percentile, a constant 0.31 percent between the 50th and 75th percentiles, and a constant 0.58 percent between the 75th and 90th percentiles, a constant 0.86 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 is 0.461 percent, which is used in this analysis.

Population: Population of ages 7 to 14

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

This appendix presents the unit values that are available in BenMAP for each of the health endpoints included in the current suite of health impact functions. 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 is given in Table I-1.

Table I-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 = logeX, then X is lognormally distributed. Equivalently, X is lognormmaly distributed if X = eY, where Y is noramally distributed.

*** The Weibull distribution has the following probability density function:

$$\left(\frac{\beta}{\alpha}\right)\left(\frac{x}{\alpha}\right)^{\beta-1} e^{-(x/\alpha)^{\beta}}$$

I.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.

I.1.1 Value of a Statistical Life Based on 26 Studies

One unit value available in BenMAP is $6.3 million. 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.

The VSL approach and the set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria as Viscusi in his review of value-of-life studies. The $6.3 million estimate is consistent with Viscusi's conclusion (updated to 2000$) that "most of the reasonable estimates of the value of life are clustered in the $3.8 to $8.9 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.

I.1.2 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, we have included three alternatives based loosely on the results of recent work by Mrozek and Taylor (2002) and Viscusi and Aldy (2003). Each of the four alternatives has a mean value of $5.5 million (2000$), but with a different distributions: normal, uniform, triangular, and beta. Table I-2 presents the distribution parameters for the suite of mortality valuations currently available in BenMAP.

Table I-2. Unit Values Available for Mortality

Basis for Estimate *	Age Range at Death		Unit Value (VSL) (20005)	Distribution of Unit Value	Parameters of Distribution	
	Min	Max			P1	P2
VSL, based on 26 value-of-life studies	0	99	$6,324,101	Weibull	5.32E-6	1.509588
VSL, based on range from $1 million to $10 million – 95% CI of assumed normal distribution	0	99	$5,500,000	Normal	2,295,960.54	–
VSL based on range from $1 million to $10 million – assumed uniform distribution	0	99	$5,500,000	Uniform	1,000,000	10,000,000
VSL based on range from $1 million to $10 million – assumed triangular distribution	0	99	$5,500,000	Triangular	1,000,000	10,000,000

* The original value of a statistical life was calculated in 1990 $. We have used a factor of 1.3175, based on the All-Items CPI-U.

I.2 Chronic Illness

This sub-section presents the unit values developed for chronic bronchitis, chronic asthma, and non-fatal myocardial infarctions.

I.2.1 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.2.1.1 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 Viscusis study, and WTP to avoid a less severe case. The estimated relationship (see Table 4 in Krupnick and Cropper) can be written as:

$$1n(WTP) = \alpha + \beta \times sev$$

where a denotes all the other variables in the regression model and their coefficients, ß 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

$$1n(WTP_{13}) = \alpha + \beta \times 13$$

and

$$1n(WTP_x) = \alpha + \beta \times x$$

Subtracting one equation from the other,

$$1n(WTP_{13}) - 1n(WTP_x) = \beta \times (13 - x)$$

or

$$1n\left(\frac{WTP_{13}}{WTP_x}\right) = \beta \times (13 - x)$$

Exponentiating and rearranging terms,

$$WTP_x = WTP_{13} \times e^{-\beta \bullet (13-x)}$$

There is uncertainty surrounding the exact values of WTP13; x, and ß, 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 ß 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 $340,000 (in $2000, 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.2.1.2 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 2000$ using the CPI-U for medical care; lost earnings (opportunity costs) were inflated to 2000$ 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 are shown in Table I-3 below.

Table I-3. 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	$340,482	Custom
COI: med costs + wage loss, 3% DR	27	44	$18,960	$135,463	$154,422	None
	45	64	$23,759	$76,029	$99,788	None
	65	99	$11,088	$0	$11,088	None
COI: med costs + wage loss, 7% DR	27	44	$7,886	$80,444	$88,331	None
	45	64	$14,390	$59,577	$73,967	None
	65	99	$9,030	$0	$9,030	None

I.2.2 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 $150,221.

I.2.3 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 $47,637 (in 2000$).

O'Conor 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'Conor 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 $30,257 (in 2000$). A unit value, based on a three percent discount rate, is the average of the two estimates, or $38,947. 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. 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	$38,947	Triangular	$30,257	$47,637
WTP: 7% DR	27	99	$25,357	Triangular	$19,699	$31,015

I.2.4 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. 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: $8,774 for someone between the ages of 25 and 44, $12,932 for someone between the ages of 45 and 54, and $74,746 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings using a seven percent discount rate are $7,855, $11,578, and $66,920, 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 possible 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 $109,474 in year 2000$. 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 $49,651 in 2000$ for MI patients, using statistical prediction (regression) models to estimate inpatient costs. Only inpatient costs (physician fees and hospital costs) were included.

- Russell et al. (1998) estimated first-year direct medical costs of treating nonfatal MI of $15,540 (in 1995$), and $1,051 annually thereafter. Converting to year 2000$, that would be $18,880 for a 5-year period, using a three percent discount rate, or $17,850, 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 x 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-5 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-5. 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	$109,474	$0	$109,474
	25	44	$109,474	$9,033	$118,507
	45	54	$109,474	$13,313	$122,787
	55	65	$109,474	$76,951	$186,425
	66	99	$109,474	$0	$109,474
COI: 10 yrs med, 5 yrs wages, 3% DR, Eisenstein (2001)	0	24	$49,651	$0	$49,651
	25	44	$49,651	$9,033	$58,683
	45	54	$49,651	$13,313	$62,964
	55	65	$49,651	$76,951	$126,602
	66	99	$49,651	$0	$49,651
COI: 5 yrs med, 5 yrs wages, 3% DR, Russell (1998)	0	24	$22,331	$0	$22,331
	25	44	$22,331	$9,033	$31,363
	45	54	$22,331	$13,313	$35,644
	55	65	$22,331	$76,951	$99,281
	66	99	$22,331	$0	$22,331
COI: 5 yrs med, 5 yrs wages, 7% DR, Wittels (1990)	0	24	$109,474	$0	$109,474
	25	44	$109,474	$8,087	$117,561
	45	54	$109,474	$11,919	$121,393
	55	65	$109,474	$68,894	$178,368
	66	99	$109,474	$0	$109,474
COI: 10 yrs med, 5 yrs wages, 7% DR, Eisenstein (2001)	0	24	$49,651	$0	$49,651
	25	44	$49,651	$8,087	$57,738
	45	54	$49,651	$11,919	$61,570
	55	65	$49,651	$68,894	$118,545
	66	99	$49,651	$0	$49,651
COI: 5 yrs med, 5 yrs wages, 7%	0	24	$21,113	$0	$21,113

Basis of Estimate	Age Range		Medical Cost *	Opportunity Cost **	Total Cost
	Min	Max			
DR, Russell (1998)	25	44	$21,113	$8,087	$29,200
	45	54	$21,113	$11,919	$33,032
	55	65	$21,113	$68,894	$90,007
	66	99	$21,113	$0	$21,113

* From Cropper and Krupnick (1999). Present discounted value of 5 yrs of lost earnings, at 3% and 7% discount rate, adjusted from 1977$ to 2000$ using CPI-U "all items".

** 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 $109,474; Russell et al. estimated first- year direct medical costs and annual costs thereafter. The resulting 5-year cost is $22,331, using a 3% discount rate, and $21,113, using a 7% discount rate. Medical costs were inflated to 2000$ using CPI-U for medical care.

1.3 Hospital Admissions & Emergency Room Visits

This section presents the 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.

I.3.1 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 (2007). The NIS is the largest inpatient care database in the United States, and it is the only national hospital database containing charge information on all patients. It contains data from a very large nationally representative sample of about eight million hospital discharges, and therefore provides the best estimates of mean hospital charges and mean lengths of stay available, with negligible standard errors. The sampling frame for the 2007 NIS is a sample of hospitals that comprises approximately 90 percent of all hospital discharges in the United States. Since the NIS is based on discharge samples, the discharge-level weight was used to weight discharges in order to produce national estimates. The principle diagnoses 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 2010 American Community Survey (ACS). ACS provided data for median annual earnings of full-time males and females at least 16 years old in 800 counties. For these 800 counties, the average of full-time male and female earnings was used to estimate the county-specific median annual income. For all other counties, the ratio of state-specific median annual income in 2010 to 2000 was calculated. This ratio was then applied to the 2000 county-specific median annual income to obtain the estimate for 2010 county-specific income (the source for 2000 county-specific income is Geolytics, 2001). 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 charge are from year 2007 and median income data are from 2010, the default hospital admission unit values in BenMAP are in year 2000 dollars to be consistent with the unit values of other health endpoints in BenMAP. This was done by deflating the medical costs (2007 dollars) and median income (2010 dollars) to 2000 dollars using BenMAP's inflation index. Then when BenMAP estimates year 2007 dollars, the answer will be the same as if 2007 NIS and 2010 ACS data were used.

The hospital admission outcomes for which unit values are available in BenMAP are given in Table I-6. 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 for the table below, to give a general idea of the cost of illness estimates for the different hospital admissions endpoints.

Table I-6. Unit Values Available for Hospital Admissions

Endpoint	ICD Codes	Age Range		Mean Hospital Charge (2000 $)	Mean Length of Stay (days)	Total Cost of Illness (Unit Value in 2000 $)*
		Min	Max			
HA, All Cardiovascular	390-429	18	64	$26,654	4.12	$27,119
HA, All Cardiovascular	390-429	65	99	$24,893	4.88	$25,444
HA, All Cardiovascular	390-429	0	99	$25,605	4.59	$26,123
HA, Congestive Heart Failure	428	65	99	$19,693	5.32	$20,293
HA, Dysrhythmia	427	65	99	$19,560	3.98	$20,008
HA, Dysrhythmia	427	0	99	$19,301	3.72	$19,720
HA, Ischemic Heart Disease	410-414	65	99	$32,452	4.61	$32,972
HA, All Respiratory	460-519	0	1	$9,883	3.19	$10,242
HA, All Respiratory	460-519	65	99	$20,667	6.07	$21,351
HA, All Respiratory	460-519	0	99	$19,009	5.35	$19,612
HA, Asthma	493	0	64	$9,723	3.00	$10,061
HA, Astham	493	65	99	$15,267	4.79	$15,808
HA, Asthma	493	0	99	$10,852	3.37	$11,232
HA, Chronic Lung Disease	490-496	18	64	$12,836	3.90	$13,276
HA, Chronic Lung Disease	490-496	65	99	$14,835	4.79	$15,375
HA, Chronic Lung Disease	490-496	0	99	$13,025	4.10	$13,487
HA, Chronic Lung Disease (less Asthma)	490-492, 494-496	18	64	$13,999	4.23	$14,475
HA, Chronic Lung Disease (less Asthma)	490-492, 494-496	65	99	$14,742	4.79	$15,282
HA, Chronic Lung Disease (Less Asthma)	490-492, 494-496	0	99	$14,497	4.59	$15,015
HA, Pneumonia	480-487	65	99	$17,647	5.77	$18,297
HA, Pneumonia	480-487	0	99	$16,956	5.25	$17,548

* 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 ($695 in 2007 dollar or $564.3 in 2000 dollar) by 5. The median weekly wages for 2007 was obtained from the U.S. Census Bureau, Statistical Abstract of the United States: 2009, Section 12, T able 626: "Full-Time Wage and Salary Workers - Numbers and Earnings: 2000 to 2007."

I.3.2 Emergency Room Visits for Asthma

Two unit values are currently available for use in BenMAP for asthma emergency room (ER) visits. One is $311.55, 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 $311.55 in 2000 $ (using the CPI-U for medical care to adjust to 2000$). 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 $311.55, on the interval [$230.67, $430.93].

A second unit value is $260.67 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 I-7.

Table I-7. Unit Values Available for Asthma-Related ER Visits

Basis for Estimate	Age Range		Unit Value	Distribution of Unit Value	Parameters of Distribution	
	Min	Max			P1	P2
COI: Smith et al. (1997)	0	99	$312	Triangular	$231	$431
COI: Standford et al. (1999)	0	99	$261	Normal	5.22	--

I.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 each of these acute symptoms and illnesses. Tables J-8 and J-9 summarize the values.

Table I-8. Unit Values Available for Acute Symptoms and Illnesses (in 2000 $)

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	$59	Uniform	17.51	101.11
	WTP: 6 day illness, CV studies	0	17	$356	Uniform	105.06	606.64
	WTP: 28 symptom-days, Dickie and Ulery	0	17	$374	Lognormal	5.947	0.0907
Any of 19 Respiratory Symptoms	WTP: 1 day illness, CV studies	1	65	$24	Uniform	0	48.25
Minor Restricted Activity Days	WTP: 1 day, CV studies	18	99	$51	Triangular	20.71	80.37
	WTP: 3 symptoms 1 day, Dickie and Ulery (2002)	18	99	$98	Lognormal	4.6088	0.0649
Lower Respiratory Symptoms	WTP: 1 day, CV studies	0	17	$16	Uniform	6.94	24.47
	WTP: 2 symptoms 1 day, Dickie and Ulery (2002)	0	17	$187	Lognormal	5.2556	0.07048
	WTP: 2x1 day, CV studies	0	17	$31	uniform	13.89	48.93
School Loss Days	Described in text	0	17	$75	None	N/A	N/A
Upper Respiratory Symptoms	WTP: 1 day, CV studies	0	17	$25	Uniform	9.22	43.11
	WTP: 2 symptoms 1 day, Dickie and Ulery (2002)	0	17	$187	Lognormal	5.2556	0.07048
	WTP: 2x1 day, CV studies	0	17	$49	Uniform	18.45	86.22
Work Loss Days **	Median daily wage, county-specific	18	65	$115	None	N/A	N/A

* 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.

** 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.

Table I-9. 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	$43	Uniform	15.56	70.88
	1 symptom-day, Dickie and Ulery (2002)	18	99	$74	Lognormal	4.321	0.0957
	Bad asthma day, Rowe and Chestnut (1986)	0	17	$43	Uniform	15.56	70.88
	2 x bad asthma day, Rowe and Chestnut (1986)	0	17	$86	Uniform	31.12	141.77
	1 symptom-day, Dickie and Ulery (2002)	0	17	$156	Lognormal	5.074	0.0925

* 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.4.1 Acute Bronchitis in Children

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore must be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

Three unit values are available in BenMAP for acute bronchitis in children. In previous benefit analyses, EPA used a unit value of $59.31. 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. More typically, it can last for 6 or 7 days. A simple adjustment, then, would be to multiply the original unit value of $59.31 by 6 or 7. A second unit value of $356 (=$59.31 x 6) was therefore derived.

Finally, as noted above, 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 $374. 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.

I.4.2 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-10 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-10. Median WTP Estimates and Derived Midrange Estimates (in 1999 $)

Symptom*	Dickie et al.	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	4.81	20.84	-	12.75
Head/sinus congestion	5.61	22.45	10.45	12.75
Coughing	1.61	17.65	6.35	8.93
Eye irritation	-	20.03	-	20.03
Headache	1.61	32.07	-	12.75
Shortness of breath	0.00	-	13.47	6.37
Pain upon deep inhalation (PDI)	5.63	-	-	5.63
Wheeze	3.21	-	-	3.21
Coughing up phlegm	3.51**	-	-	3.51
Chest tightness	8.03	-	-	8.03

* 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, $24.64, is just an average of the seven estimates of mean WTP for the different URS complexes.

The WTP estimates on which the first unit value is based were elicited from adults, whereas the health endpoint associated with air pollution in the epidemiological study is in children. As noted above, 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. We therefore derived a second unit value of $49.28 (=2 x $24.64) from the first unit value.

A third 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 $187.

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.

I.4.3 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 I-10), 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 original unit value for LRS, $15.57, is just an average of the eleven estimates of mean WTP for the different LRS symptom clusters.

A second unit value is twice the original unit value, or $31.15, 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 $187.

I.4.4 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:

$$(0.40)(\$24.64) + (0.40)(\$15.57) + (0.20)(\$24.64 + \$15.57) = \$24.12.$$

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.

I.4.5 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.

I.4.6 Minor Restricted Activity Days (MRADs)

Two unit values are currently available in BenMAP for MRADs. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEc (1993) derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is $38.37 (1990 $). 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.

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 2000 $, this is a triangular distribution centered at $50.55, ranging from $21 to $80.

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 $98.

I.4.7 Asthma Exacerbation

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.

Two unit values are currently available in BenMAP for asthma exacerbation in adults, and three are currently available for asthma exacerbation in children. In past benefit analyses, EPA based willingness to pay to avoid an asthma exacerbation on four WTP estimates from Rowe and Chestnut (1986) for avoiding a "bad asthma day." The mean of the four average WTPs is $32 (1990 $), or $43 in 2000$. 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 [$16, $71] in 2000 $. This unit value is available for both adults and children.

A 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 $86, 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 $156.

I.4.8 School Loss Days

There is currently one unit value available in BenMAP for school loss days, based on (1) the probability that, if a school child stays home from school, a parent will have to stay home from work to care for the child, and (2) the value of the parent's lost productivity. We first estimated the proportion of families with school-age children in which both parents work, and then valued a school loss day as the probability of a work loss day resulting from a school loss day (i.e., the proportion of households with school-age children in which both parents work) times a measure of lost wages.

From the U.S. Bureau of the Census (2002) we obtained (1) the numbers of single, married, and "other" (i.e., widowed, divorced, or separated) women with children in the workforce, and (2) the rates of participation in the workforce of single, married, and "other" women with children. From these two sets of statistics, we calculated a weighted average participation rate of 72.85 percent, as shown in Table I-11.

Our estimated daily lost wage (if a mother must stay at home with a sick child) is based on the median weekly wage among women age 25 and older in 2000. This median weekly wage is $551. Dividing by 5 gives an estimated median daily wage of $103. The expected loss in wages due to a day of school absence in which the mother would have to stay home with her child is estimated as the probability that the mother is in the workforce times the daily wage she would lose if she missed a day = 72.85% of $103, or $75. We currently have insufficient information to characterize the uncertainty surrounding this estimate.

Table I-11. Women with Children: Number and Percent in the Labor Force, 2000, and Weighted Average Participation Rate

Category	Women in Labor Force (millions)* (1)	Population Rate (%)* (2)	Implied Total Number in Population (in millions) (3) = (1)/(2)	Implied Percent in Population (4)	Population-Weighted Average Participation Rate [=sum (2)x(4) over rows]
Single	3.1	73.9%	4.19	11.84%	--
Married	18.2	70.6%	25.78	72.79%	--
Other**	4.5	82.7%	5.44	15.36%	--
Total	--	--	35.42	--	72.85%

*Source: U.S. Bureau of the Census (2002).
** Widowed, divorced, or separated.

A unit value based on the approach described above is likely to understate the value of a school loss day in two ways. First, it omits WTP to avoid the symptoms/illness which resulted in the school absence. Second, it effectively gives zero value to school absences which do not result in a work loss day. The unit value of $75 is therefore considered an "interim" value until such time as alternative means of estimating this unit value become available.

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

This section describes the population and monitor data in the United States setup.

- 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, $PM_{2.5}$, PM_{10}, lead, NO_2, and SO_2 monitor data for the years 2000-2007. Data for CO are available at the BenMAP website: http://www.epa.gov/air/benmap/.

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 developed by Abt Associates, 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. If you are interested in PopGrid, please email: benmap@epa.gov.

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 desired by you.

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:

$$age_{3\text{–}12} = \frac{1}{2} \times age_{1\text{–}4} + age_{5\text{–}9} + \frac{3}{5} \times age_{10\text{–}14}$$

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 (2012) provides the county-level population forecasts used to calculate the scaling ratios; these data are discussed in detail below.

In the simplest case, where one is forecasting a single population variable, say, children ages 4 to 9 in the year 2020, BenMAP calculates:

$$age_{4\text{–}9,g,2020} = age_{4\text{–}9,g,2010} \times \frac{age_{4\text{–}9,county,2020}}{age_{4\text{–}9,county,2010}}$$

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:

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

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:

$$age_{4\text{–}9,g\ in\ county_c,2010} = age_{4\text{–}9,g,2010} \times fraction\ \ age_{4\text{–}9,g\ in\ county_c}$$

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:

$$age_{4-9,\,g\ in\ county_c,\,2020} = age_{4-9,g\ in\ county_c,2010} \times \frac{age_{4-9,\,county_c,2020}}{age_{4-9,\,county_c,2010}}$$

Combining all these steps for "n" counties within the gth grid-cell, we forecast the population of persons ages 4 to 9 in the year 2020 as follows:

$$age_{4-9,g,2020} = \sum_{c=1}^{n} age_{4-9,g,2010} \times \frac{total\ pop_{g\,in\,county_c}}{total\ pop_g} \times \frac{age_{4-9,\,county_c,2020}}{age_{4-9,\,county_c,2010}}$$

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

$$age_{4-9,g,2020} = \sum_{c=1}^{n} age_{4-9,g,2010} \times \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \times \frac{age_{4-9,\,county_c,2020}}{age_{4-9,\,county_c,2010}}$$

$$age_{10-14,g,2020} = \sum_{c=1}^{n} age_{10-14,g,2010} \times \frac{total\ pop_{g\ in\ county_c}}{total\ pop_g} \times \frac{age10-14_{10-14,\,county_c,2020}}{age_{10-14,\,county_c,2010}}$$

$$age_{4-12,g,2020} = age_{4-9,\,g,2020} + \frac{3}{5} \times age_{10-14,\,g,2020}$$

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, 2011-2040) 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-levelSF1 file. (For the sake of completeness, we note that there exists a county-level Census 2000 MARS file; however, due to major population count discrepancies between the county-level MARS file and block-level SF1 file, we used only the nation-level summary table. Tables in MARS documentation file did not have the discrepancies that the county-level file had. We were unable to get an adequate explanation of this from the U. S. Census.)

The steps in preparing the data are as follows:

1. Adjust Age-classifications:

We combined some age groups in the block-level SF1 data to match the age groups wanted for BenMAP. For example, we combined age groups 15-17 and 18-19 to create the 15-19 age group used in BenMAP. Then, in the case of the 0-4 age group, we split it

into <1 and 1-4 using the tract-level SF1 data, which gave us the fraction of 0-4 year-olds who are <1.

2. Fill in Missing Racial-Ethnic Interactions:

We used the tract-level SF1 data to calculate the fraction of Hispanics in each ethnically-aggregated subpopulation from the block-level data, by age and sex. We used these fractions to distribute each age-sex-race-block-level datum into Hispanics and non-Hispanics.

3. Assign "Other" and "Multi-Racial" to the Remaining Four Racial Categories:

We assign the "Other" race category in two steps. First, based on the national MARS data, we estimated how many people in the "multi-racial" category checked off "some other race" as one of their races, for Hispanics and non-Hispanics separately. In each age-sex-race-block-level datum, we added those people to "other race" category to create the re-distribution pool, analogously to the method implemented by Census while creating MARS data (see U.S. Census Bureau, 2002a, Table 1, below). Second, based on the national re-allocation fractions for Hispanics and non-Hispanics (derived from the MARS data), we assigned the "Other" race into the four races of interest and "multi-race".

After the assignment of the "Other" race category, we then assigned "multi-racial" category to the four racial categories, using state fractions of these races in each age-sex-race-block- level datum.

Exhibit J-3: Summary of Modified Race and Census 2000 Race Distributions for the United States

Subject	Modified Race		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 (2012) developed county-level forecasts for each year from 2000 through 2040, 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/CED12.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 2000-2040, 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.

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 2000

There are a small number of cases were the 2010 county population for a specific demographic group is zero, so the ratio of any future year to the year 2010 data is undefined. In these relatively rare cases, we prepared statewide and national totals and used ratios at the higher levels of geographic aggregation when the more local ratios caused divide-by-zero errors.

J.1.3 PopGrid

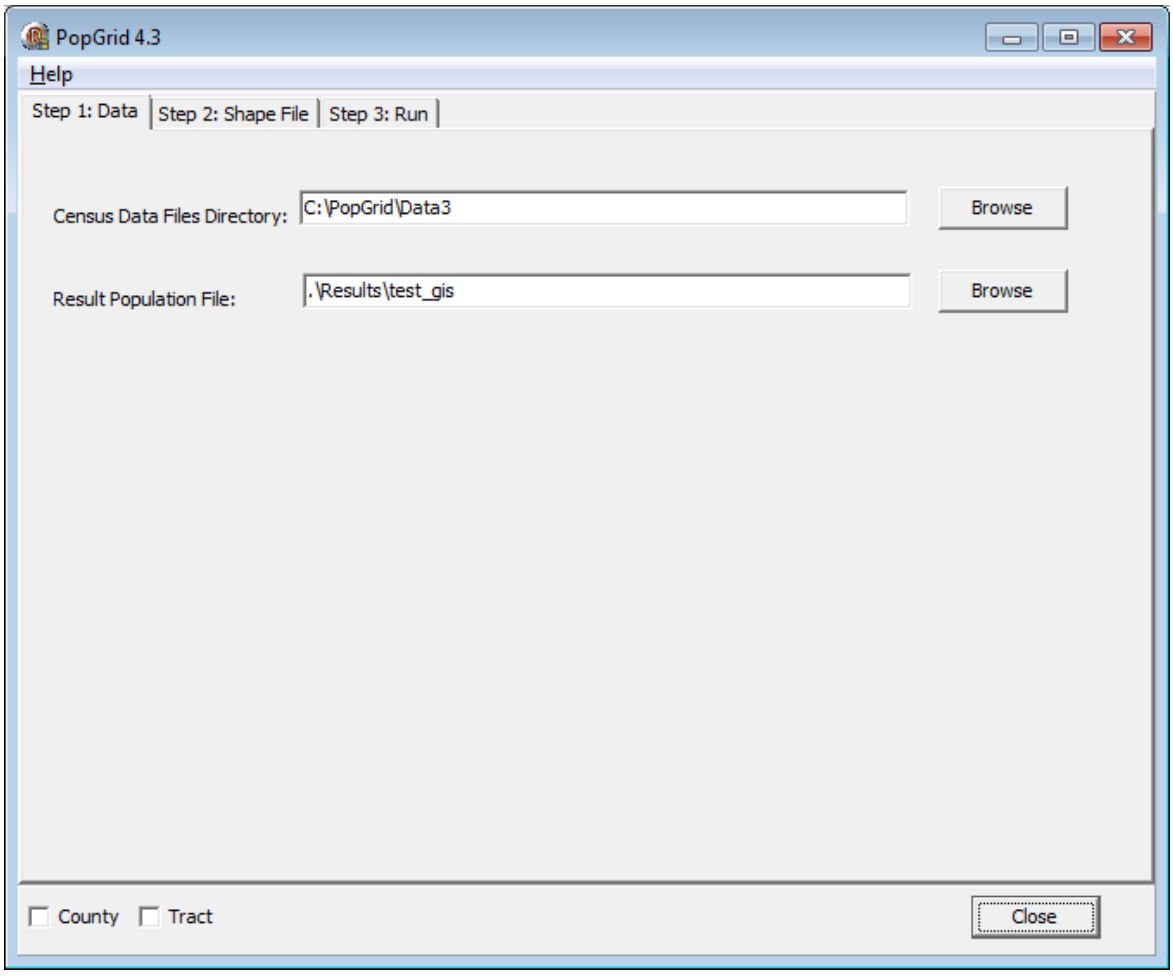
If the geographic center of a Census block falls within a population grid-cell, PopGrid assigns the block population to this particular population grid-cell. Note that the grid-cells in an air quality model, such as CMAQ, may cross multiple county boundaries. PopGrid keeps track of the total number of 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.

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

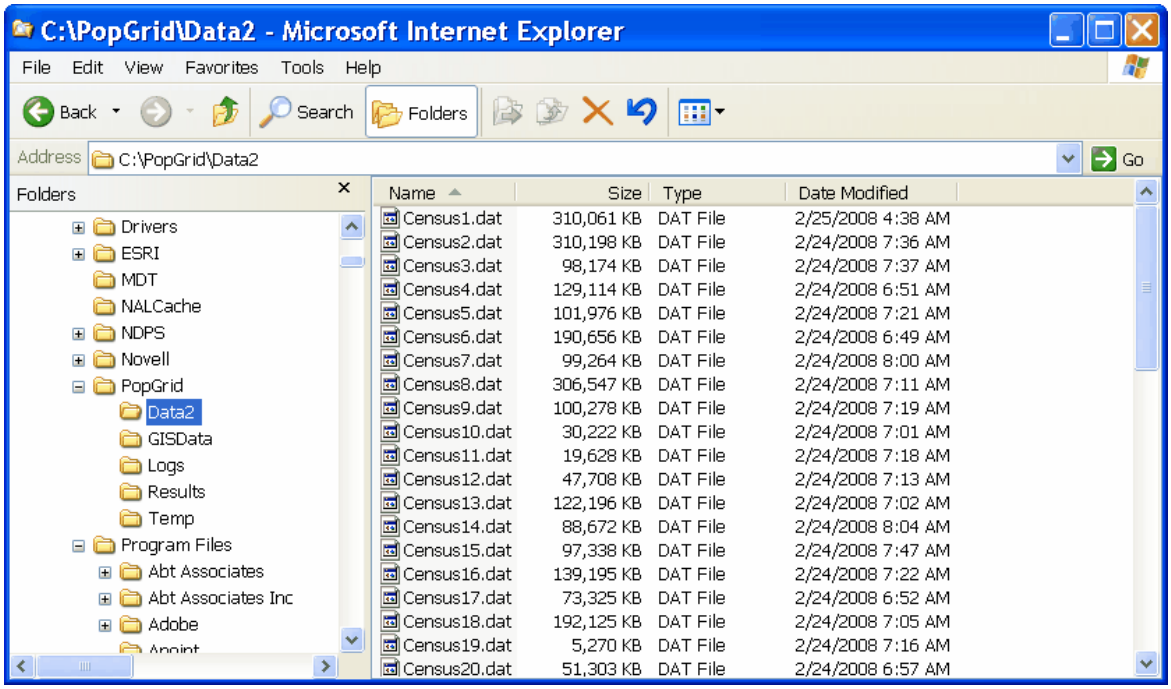
age-gender groups within a given race-ethnic group have the same geographic distribution.

J.1.3.1 How to Use PopGrid

After installing PopGrid, double-click on the PopGrid executable "PopGrid4.exe." The following screen will appear:

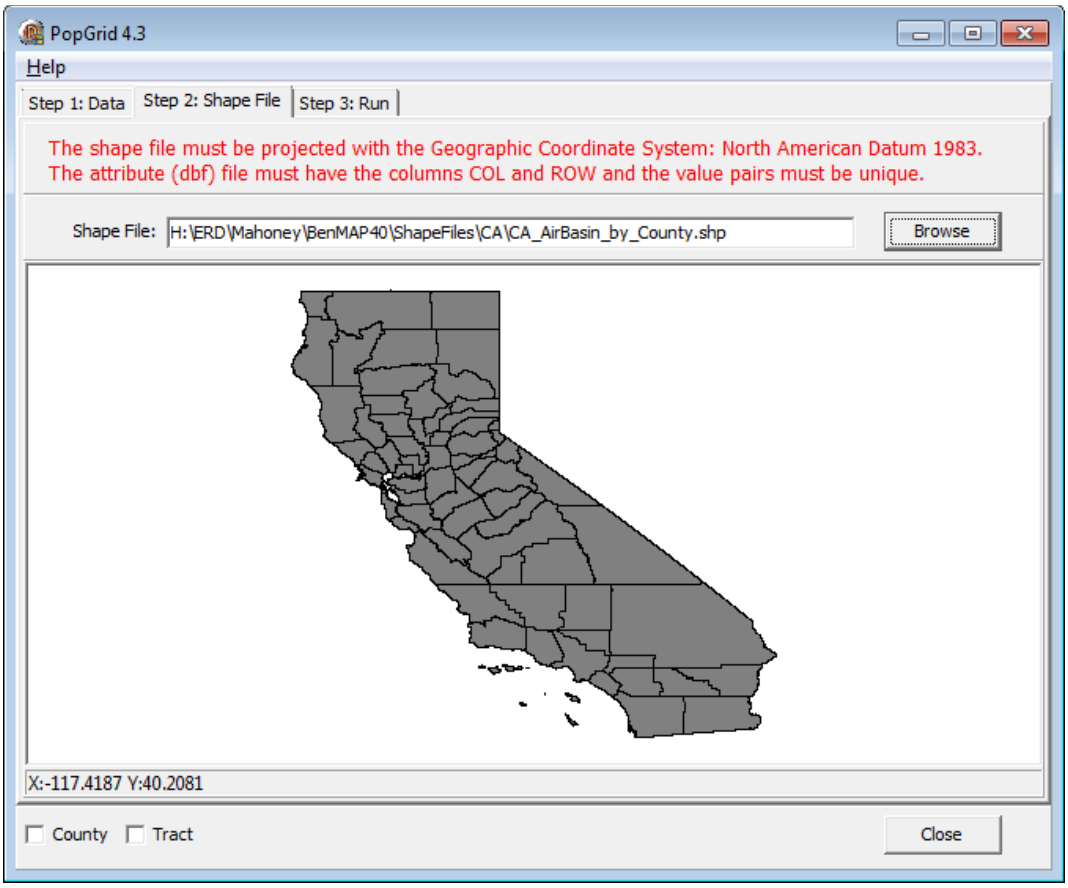


The **Census Data Files Directory** box points PopGrid to where the block data are located that PopGrid uses. Make sure that the files in this directory are unzipped. This data folder should look something like the following:

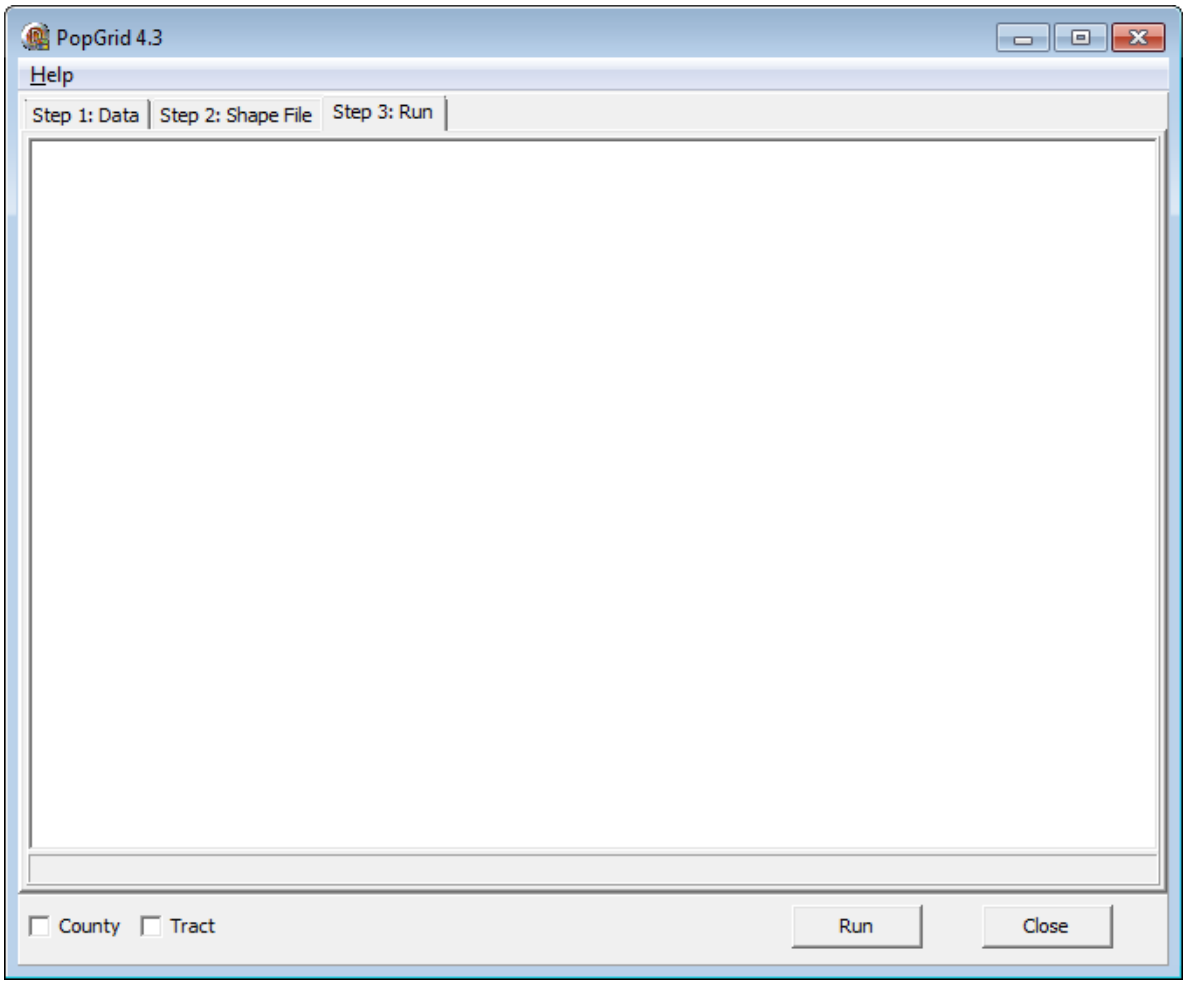


The **Result Population File** box provides the path and the name of the file that you want to create. In the example above, PopGrid is being used to estimate population for the intersection of air basins and counties in California (CA_AirBasin_by_County).

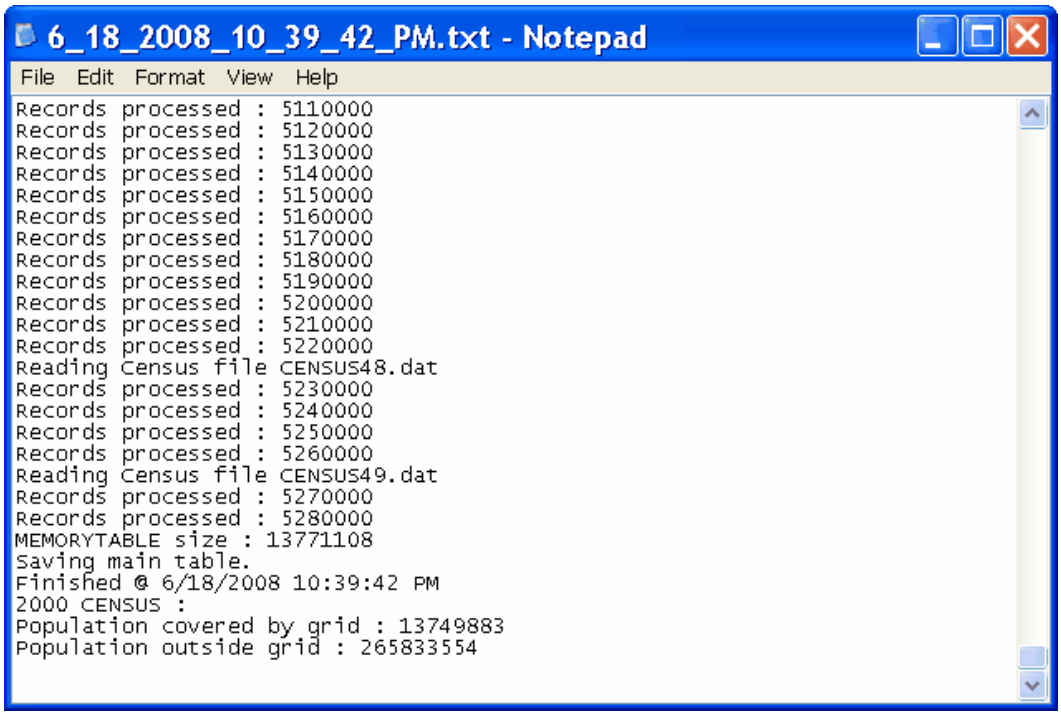
Click on the **Step 2: Shape File** tab. Choose the shapefile that you want to use. The example for air basins and counties in California looks as follows:



After choosing your shapefile, 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").



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-4 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-4. Population File Fragment from PopGrid

Row	Column	Year	Population	Race	Ethnicity	Gender	AgeRange
58	81	2000	1.54	WHITE	HISPANIC	MALE	0TO0
58	81	2000	0.03	BLACK	HISPANIC	MALE	0TO0
58	81	2000	0.01	NATAMER	HISPANIC	MALE	0TO0
58	81	2000	0.01	ASIAN	HISPANIC	MALE	0TO0
58	81	2000	4.86	WHITE	HISPANIC	MALE	1TO4
58	81	2000	0.12	BLACK	HISPANIC	MALE	1TO4
58	81	2000	0.03	NATAMER	HISPANIC	MALE	1TO4
58	81	2000	0.03	ASIAN	HISPANIC	MALE	1TO4
58	81	2000	6.79	WHITE	HISPANIC	MALE	5TO9
58	81	2000	0.21	BLACK	HISPANIC	MALE	5TO9
58	81	2000	0.05	NATAMER	HISPANIC	MALE	5TO9
58	81	2000	0.05	ASIAN	HISPANIC	MALE	5TO9
58	81	2000	0.90	WHITE	HISPANIC	MALE	10TO14
58	81	2000	0.04	BLACK	HISPANIC	MALE	10TO14

Row	Column	Year	Population	Race	Ethnicity	Gender	AgeRange
58	81	2000	0.01	NATAMER	HISPANIC	MALE	10TO14
58	81	2000	0.01	ASIAN	HISPANIC	MALE	10TO14
58	81	2000	3.44	WHITE	HISPANIC	MALE	15TO19
58	81	2000	0.15	BLACK	HISPANIC	MALE	15TO19
58	81	2000	0.04	NATAMER	HISPANIC	MALE	15TO19
58	81	2000	0.04	ASIAN	HISPANIC	MALE	15TO19
58	81	2000	1.49	WHITE	HISPANIC	MALE	20TO24
58	81	2000	0.06	BLACK	HISPANIC	MALE	20TO24
58	81	2000	0.02	NATAMER	HISPANIC	MALE	20TO24
58	81	2000	0.03	ASIAN	HISPANIC	MALE	20TO24
58	81	2000	1.93	WHITE	HISPANIC	MALE	25TO29
58	81	2000	0.04	BLACK	HISPANIC	MALE	25TO29
58	81	2000	0.01	NATAMER	HISPANIC	MALE	25TO29
58	81	2000	0.01	ASIAN	HISPANIC	MALE	25TO29
58	81	2000	1.87	WHITE	HISPANIC	MALE	$30TO_34$
58	81	2000	0.08	BLACK	HISPANIC	MALE	$30TO_34$

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-2 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-5, 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-5. 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 2007 data, as reported to the U.S. Environmental Protection Agency's (EPA) Air Quality System (AQS), for $PM_{2.5}$, PM_{10} STP and LC, lead TSP, ozone, NO_2, SO_2, and CO. Table J-6 summarizes the data sources and vintage of the processed data.

Table J-6. Underlying Data Sources for BenMAP Air Quality Data File

Pollutant	AQS Parameter Code	Year	Data Source	Date Acquired	Documented Vintage
$PM_{2.5}$	88101	2000-2006	http://www.epa.gov/ttn/airs/airsaqs/detailedata/downloadaqsdata.htm	5/22/2008	4/10/2008
		2007	Requested from AQS representative	6/4/2008	
Ozone	44201	2000-2006	http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm	5/22/2008	4/14/2008-4/15/2008
		2007	Requested from AQS representative	6/4/2008	
Lead TSP	12128	2000	http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm	5/29/2008	6/20/2007
		2001-2006	http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm	5/29/2008	4/9/2008
		2007	http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm	6/4/2008	
PM_{10} STP	81102	2000-2006	http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm	5/22/2008	4/10/2008
		2007	Requested from AQS representative	6/4/2008	
PM_{10} LC	85101	2000-2007	Requested from AQS representative	6/4/2008	

The AQS data were uploaded to the STI Air Quality Archive (AQA) Oracle database. The AQA database performs additional quality control (QC) checks against the AQS data, such as uniqueness by AQS site, method, parameter occurrence code (POC), and duration codes; checks of minimum and maximum values; and maximum rate of change between consecutive data values (where appropriate). The specific QC checks imposed on the BenMAP data are outlined in Table J-7. No maximum value filters were applied to the concentration data. High aerosol concentration values caused by dust storms or other exceptional events are included in the BenMAP-ready data files.

Table J-7. Pollutant-Specific QC Checks Performed in AQA

Pollutant	AQS Parameter Code	Acceptable Concentration Range	Maximum Rate of Change
$PM_{2.5}$	88101	> = 0 µg/m3	–
Ozone	44201	> = 0 ppb	60 ppb
Lead	12128	> = 0 µg/m3	–

Pollutant	AQS Parameter Code	Acceptable Concentration Range	Maximum Rate of Change
PM_{10}	81102 and 85101	> = 0 µg/m3	–
CO	42101	> = 0 ppm	–
NO_2	42602	> = 0 ppb	50 ppb
SO_2	42401	> = 0 ppb	–

J.2.1 Data Processing

STI developed data processing procedures consistent with those used in the past by Abt Associates to create air quality data for files for use in the BenMAP model. Critical data processing rules implemented in the deliverable data are listed below:

- Data delivered by STI are reported with consistent units: µg/m^3 for aerosols; ppb for ozone, NO_2, and SO_2; and ppm for CO.
- The "monitor name" field is populated by concatenating the AQS site, parameter, and POC codes.
- The "monitor description" field is populated with the following metadata: method code, land use, location setting, POC, and AQS parameter code. The AQS probe location and monitoring objective code fields are left blank in STI-processed data.
- The data were formatted with one record per site, pollutant, POC, and year for use in the BenMAP program. Data for 365 days, or 8,760 hourly values, are expected per record. This format is satisfied regardless of leap years; an average of February 28 and 29 data are reported.
- The monitoring method is allowed to change over the course of a year. To provide a more complete record, data with multiple method codes for a given site, parameter, POC, and year were combined and the first reported method code was reported in the BenMAP-ready data files.
- Aerosol data collected with 24-hr sample durations were used before data collected with underlying 1-hr sample durations.One-hour sampling duration data are used for ozone, NO_2, SO_2, and CO.

J.2.2 Output Files

Table J-8 lists the number of monitors by pollutant and year, represented in the resulting BenMAP-ready data files.

Table J-8. Number of Monitors by Pollutant, AQS Parameter Code, and Year Included in the BenMAP-Ready Data Files

Pollutant	AQS Parameter Code	Number of Monitors by Year							
		2000	2001	2002	2003	2004	2005	2006	2007
$PM_{2.5}$	88101	1,311	1,339	1,328	1,316	1,226	1,260	1,197	1,144
PM_{10} STP	81102	1,415	1,388	1,320	1,240	1,211	1,199	1,164	1,111
PM_{10} LC	85101	868	757	714	694	688	748	558	502
Lead TSP	12128	209	232	253	250	231	247	204	175
Ozone	44201	1,138	1,184	1,191	1,210	1,206	1,194	1,199	1,217
NO_2	42602	444	458	445	444	446	437	428	423
CO	42101	523	518	498	482	452	429	413	389
SO_2	42401	613	605	580	561	557	534	518	520

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 0.5^{th}, 1.5^{th}, ..., and 99.5^{th} 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.1.3 Characterization of Uncertainty Surrounding QALY Estimates

The uncertainty distribution of the QALY estimates associated with a given health effect is similar to that for dollar benefits. That is, it 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 QALYs per case avoided. The derivation of the uncertainty distribution for incidence change is described above. The distributions used to characterize the uncertainty surrounding QALYs 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 that was described in the previous section for the distribution of □. That is, the
the 0.5th, 1.5th, ..., and 99.5th percentile values of its distribution.

A distribution of the uncertainty surrounding the QALYs 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 QALY distribution. To derive this new distribution, each of the 100 QALY weights is multiplied by each of the N incidence change values. These values are sorted low to high and binned down to a final distribution of QALY 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 or QALYs) 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 ith study providing an estimate β_i with variance v_i (I = 1, ..., n). Let

$$S = \sum \frac{1}{v_i},$$

denote the sum of the inverse variances. Then the weight, w_i, given to the ith 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, vfe, is the inverse of the sum of the variances, or 0.00197. (The sums of the βi and vi are not shown, since they are of no importance. The sum of the 1/vi is S, used to calculate the weights. The sum of the weights, wi, i=1, ..., 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 βi 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, Qw, the weighted sum of squared differences of the separate study estimates from the pooled estimate based on the fixed effects model, is calculated as:

$$Q_w = \sum_i \frac{1}{v_i}\left(\beta_{fe} - \beta_i\right)^2$$

Under the null hypothesis that there is a single underlying parameter, β, of which all the βi's are estimates, Qw has a chi-squared distribution with n-1 degrees of freedom. (Recall that n is the number of studies in the meta-analysis.) If Qw 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 Qw , 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 Qw < n-1), then η2 is a negative number, and it is not possible to calculate a random effects estimate. In this case, however, the small value of Qw 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 η2 is positive.

Given a value for η2 , 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 (vi) and the between-study variance (η2). Whereas the weights implied by the fixed effects model used only vi, the within-study variance, the weights implied by the random effects model use vi + η2.

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 ith health impact function for βi and the variance of incidence change for the ith health impact function for vi.

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 ith valuation for βi and the variance of dollar benefits for the ith valuation for vi.

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). Additional white space is ignored, including newline characters. To include white space in a parameter value, you must enclose the parameter in double quotes. The double quotes will not be included in the parameter value in this case (If you wish to include beginning and trailing double quotes in a parameter value, put two in a row at the beginning and end - e.g. ""Look at all those double quotes."").

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.

Supported Pollutant values are:

-Ozone

-PM_{10}

-$PM_{2.5}$

These values are also found on 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 Model Relative

This section initiates the creation of a monitor model relative air quality grid. This creation type has all the same required and optional parameters as the MonitorDirect creation type. In addition, it has two/three new required parameters.

Required Parameters:

-ScalingMethod <scaling method>

Supported scaling methods are Spatial, Temporal, and Both.

-BaseYearFilename <filename>

Specifies the base year adjustment file to use in monitor scaling.

-BaseYearDSNName <ODBC DSN Name>

Supported -BaseYearDSNName values are

"Excel Files"	Excel Spreadsheet (.xls)
"Text Files"	Comma-delimited (.csv) files
"MS Access Database"	Access Database (.mdb)

When the ScalingMethod is Temporal or Both, the FutureYearFileName and FutureYearDSNName parameters are required. These specify the future year adjustment file to use in monitor scaling.

L.3.2.4 Monitor Rollback

// MonitorRollback

SpatialScaling = '-SpatialScaling';

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 .cfg files, both because this would be quite cumbersome to do in plain text, and because it probably is not needed. Slight modifications of existing .cfg 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 .cfg file to run.

-ResultsFilename <filename>

Specifies the .cfgr 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 .cfg file.

-ControlAQG <filename>

Specifies the control air quality grid file to use when running the configuration - overrides whatever value is present in the .cfg file.

-Year <Integer>

Year in which to run the configuration (this will affect the population numbers used) - overrides whatever value is present in the .cfg 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 .cfg file.

-Threshold <real>

Threshold to use when running the configuration - overrides whatever value is present in the .cfg file.

L.3.4 Run APV

The command line version of BenMAP does not support creation of new .apv files, both because this would be quite cumbersome to do in plain text, and because it probably is not needed. Slight modifications of existing .apv 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 .apv file to run.

-ResultsFilename <filename>

Specifies the .apvr file to save the results in.

Optional Parameters

-CFGRFilename <filename>

Specifies the .cfgr file to use when running the APV configuration - note that this file must contain the same set of results which the .cfgr 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 .cfgr files; and APV Configuration Results Reports, which can be generated from .apvr 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. 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, Standard Deviation, Variance, and Latin Hypercube Points. If this parameter is not present, all fields will be included in the report.

-Grouping <grouping method>

Specifies the grouping for the results - Gridcell, then C-R Function, or C-R Function, then Gridcell. Supported values are GridcellFirst, GridcellLast. The default value is GridcellFirst.

-DecimalDigits <integer>

Specifies the number of digits after the decimal point to include in the report. Supported values are zero to eight. The default value is four.

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, AggregatedIncidence, PooledIncidence,Valuation, AggregatedValuation, PooledValuation, QALYValuation, AggregatedQALYValuation and PooledQALYValuation.

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).

L.4 Example 1

VARIABLES

%CFG%	C:\BenMAP\CommandLine\Configurations\PM25 Wizard.cfg
%APV%	C:\BenMAP\CommandLine\Configuations\PM25 Wizard.apv
%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.aqg
-GridType	"CMAQ 12km"
-Pollutant	$PM_{2.5}$

MonitorDirect

-InterpolationMethod	VNA_Alt
-MonitorDataType	Library
-MonitorDataSet	"EPA Standard Monitors"
-MonitorYear	2002
-MaxDistance	50

CREATE AQG

-Filename	%AQG%\PM25_2002Control_50km.aqg
-GridType	"CMAQ 12km"
-Pollutant	$PM_{2.5}$

MonitorRollback

-InterpolationMethod	VNA_Alt
-MonitorDataType	Library
-MonitorDataSet	"EPA Standard Monitors"

-MonitorYear	2002
-RollbackGridType	State
-MaxDistance	50

RollbackToStandardOptions

-Stanard	65
-Metric	D24HourMean
-InterdayRollbackMethod	Quadratic

RUN CFG

-CFGFilename	%CFG%
-ResultsFilename	%RESULTSDIR%\PM25_2002_50km.aqg
-BaselineAQG	%AQG%\PM25_2002Baseline_50km.aqg
-ControlAQG	%AQG%\PM25_2002Control_50km.aqg

RUN APV

-APVFilename	%APV%
-ResultsFilename	%RESULTSDIR%\PM25_2002_50km.apvr
-CFGRFilename	%RESULTSDIR%\PM25_2002_50km.cfgr
-IncidenceAggregation	Nation
-ValuationAggregation	Nation

GENERATE REPORT APVR

-InputFile	%RESULTSDIR%\PM25_2002_50km.apvr
-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.apvr
-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.cfg
%APV%	C:\BenMAP\CommandLine\Configurations\PM25 Wizard.apv
%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.aqg
-GridType	"County"
-Pollutant	$PM_{2.5}$

MonitorDirect

-InterpolationMethod	VNA_Alt
-MonitorData Type	Library
-MonitorDataSet	"EPA Standard Monitors"
-MonitorYear	2004

CREATE AQG

-Filename	%AQG%\PM25_2004_Control.aqg
-GridType	"County"
-Pollutant	$PM_{2.5}$

MonitorRollback

-InterpolationMethod	VNA_Alt
-MonitorDataType	Library
-MonitorDataSet	"EPA Standard Monitors"
-MonitorYear	2004
-RollbackGridType	State
-MaxDistance	50

RollbackToStandardOptions

-Standard	35
-Metric	D24HourMean
-InterdayRollbackMethod	Quadratic

RUN CFG

-CFGFilename	%CFG%
-ResultsFilename	%RESULTSDIR%\PM25_2004.cfgr
-BaselineAQG	%AQG%\PM25_2004Baseline.aqg
-ControlAQG	%AQG%\PM25_2004Control.aqg

RUN APV

-APVFilename	%APV%
-ResultsFilename	%RESULTSDIR%\PM25_2004.apvr
-CFGRFilename	%RESULTSDIR%\PM25_2004.cfgr
-IncidenceAggregaton	Nation
-IncidenceAggregation	Nation

GENERATE REPORT APVR

-InputFile	%RESULTSDIR%\PM25_2004.apvr
-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.apvr
-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×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<>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(sqr(X)+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 PM_{10} 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].

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.

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.

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.

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.

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.

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.

Collet, D. 1994. Modelling Survival Data in Medical Research. Chapman & Hall: New York.

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.

Decisioneering. 1996. Crystal Ball: Forecasting and Risk Analysis for Spreadsheet Users: User Manual. Version 4.0. Denver, CO. www.decisioneering .com.

Delfino, R. J., H. Gong, Jr., W. S. Linn, E. D. Pellizzari and Y. Hu. 2003. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. Environ Health Perspect. Vol. 111 (4): 647-56.

Delfino, R. J., R. S. Zeiger, J. M. Seltzer, D. H. Street and C. E. McLaren. 2002. Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. Environ Health Perspect. Vol. 110 (10): A607-17.

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.

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 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.

ESEERCO. 1994. New York State Environmental Externalities Cost Study. Report 2: Methodology. Empire State Electric Energy Research Corporation.

Fung, K. Y., S. Khan, D. Krewski and Y. Chen. 2006. Association between air pollution and multiple respiratory hospitalizations among the elderly in Vancouver, Canada. Inhal Toxicol. Vol. 18 (13): 1005-11.

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.

GeoLytics Inc. 2001. CensusCD® 1990 + Maps, Release 4.1. CD-ROM. GeoLytics, Inc.

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.

Greene, W. H. 1997. Econometric Analysis. Prentice Hall: Upper Saddle River, NJ.

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. and G. D. Thurston. 1996. Daily PM_{10}/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., 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.

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.

Kennedy, P. 1990. A Guide to Econometrics. MIT Press: Cambridge, MA.

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.

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.

Linn, W. S., Y. Szlachcic, H. Gong, Jr., P. L. Kinney and K. T. Berhane. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. Environ Health Perspect. Vol. 108 (5): 427-34.

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. Fishe, 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.

Luginaah, I. N., K. Y. Fung, K. M. Gorey, G. Webster and C. Wills. 2005. Association of ambient air pollution with respiratory hospitalization in a government-designated "area of concern": the case of Windsor, Ontario. Environ Health Perspect. Vol. 113 (3): 290-6.

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.

Michaud, J. P., J. S. Grove and D. Krupitsky. 2004. Emergency department visits and "vog"-related air quality in Hilo, Hawai'i. Environ Res. Vol. 95 (1): 11-9.

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.

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.

Nelder, J. A. and R. Mead. 1965. A simplex algorithm for function minimization. Computer Journal. Vol. 7: 308-313.

New York Department of Health (NYDOH). 2006. A Study of Ambient Air Contaminants and Asthma in New York City. New York State Department of Health Center for Environmental Health.

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.

NYDOH. 2006. A Study of Ambient Air Contaminants and Asthma in New York City. New York State Department of Health Center for Environmental Health.

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.

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. 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.

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 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 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 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 PM_{10} Pollution - a Daily Time Series Analysis. American Review of Respiratory Disease. Vol. 144 (3): 668-674.

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.

Ransom, M. R. and C. A. Pope. 1992. Elementary school absences and PM_{10} 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.

Rowe, R. D. and L. G. Chestnust. 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.

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.

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.

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.

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.

Tolley, G. S. and et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. Prepared for U.S. Environmental Protection Agency. January.

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.

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.

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, Assesssment and Standards Division. Washington, DC. EPA420-R-08-001. March. http://www.epa.gov/otaq/locomotv.htm.

Villeneuve, P. J., L. Chen, B. H. Rowe and F. Coates. 2007. Outdoor air pollution and emergency department visits for asthma among children and adults: a case-crossover study in northern Alberta, Canada. Environ Health. Vol. 6 (1): 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.

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.

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., 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.

Yang, Q., Y. Chen, D. Krewski, R. T. Burnett, Y. Shi and K. M. McGrail. 2005. Effect of short-term exposure to low levels of gaseous pollutants on chronic obstructive pulmonary disease hospitalizations. Environ Res. Vol. 99 (1): 99-105.

Yang, Q., Y. Chen, Y. Shi, R. T. Burnett, K. M. McGrail and D. Krewski. 2003. Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada. Inhal Toxicol. Vol. 15 (13): 1297-308.

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. and Schwartz, J. (2006). "Air pollution and emergency admissions in Boston, MA." J Epidemiol Community Health 60 (10): 890-5.