IEc

APPENDIX A: ELICITATION PROTOCOL

Elicitation Protocol
$PM_{2.5}$

Expert (circle one): A B C D E F
G H I J K L

Elicitors _____

1. Introduction (20 minutes)

1.1 Objectives of the Study

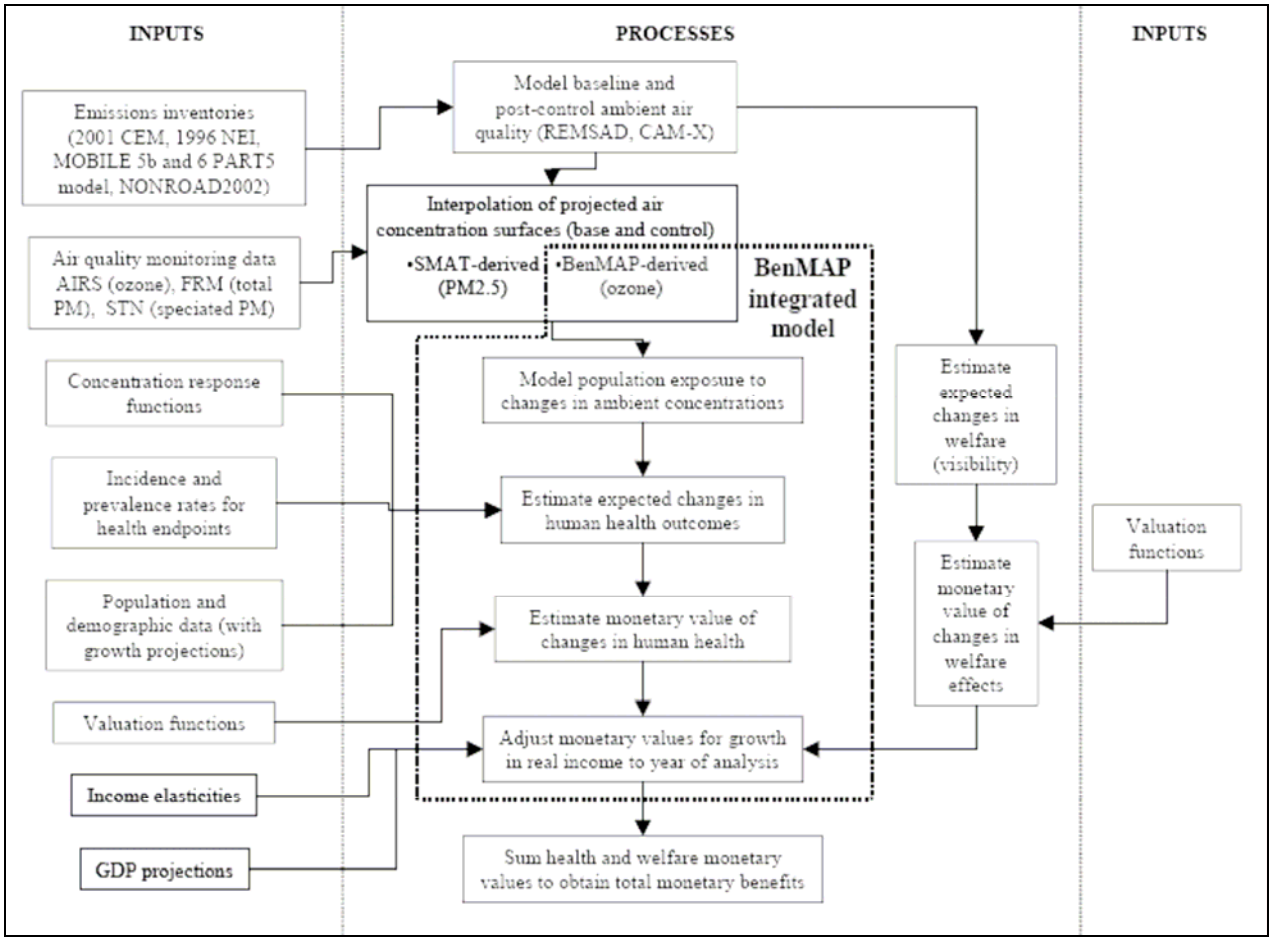
In response to recommendations made in the 2002 National Academy of Sciences (NAS) report, "Estimating the Public Health Benefits of Proposed Air Pollution Regulations," EPA is exploring ways to improve the characterization of uncertainty in its analyses of the health benefits of proposed or existing regulations affecting air quality. The purpose of this project is to provide a more complete characterization, both qualitative and quantitative, of the uncertainties associated with the relationship between reductions in ambient $PM_{2.5}$ (measured as total gravimetric mass) and mortality. The results can assist EPA in preparing future benefit analyses.

To clarify our objectives for this elicitation, it may be helpful to examine briefly the current EPA methodology for estimating the potential benefits (change in mortality) associated with reductions in ambient $PM_{2.5}$ concentrations. Figure 1, taken from the Regulatory Impact Analysis for the Clean Air Interstate Rule, illustrates the overall process and elements of a cost-benefit analysis for air pollutants, of which the health benefits analysis is one part. To estimate the health benefits associated with possible regulatory changes, EPA uses concentration-response (C-R) functions that relate proposed reductions in community level ambient concentrations of $PM_{2.5}$ to changes in mortality (and other adverse health outcomes). Although both long-term and short-term exposures to ambient levels of $PM_{2.5}$ have been associated with an increased risk of mortality, cohort studies are thought to better capture the full public health impact of exposure to air pollution over time, because they capture the effects of long-term exposures and possibly some component of short-term exposures (Kunzli et al., 2001; NRC, 2002). EPA currently employs a C-R function based on the published results of the American Cancer Society (ACS) cohort study (Pope et al., 2002) to estimate mortality changes associated with reductions in annual average $PM_{2.5}$ concentrations.[1] EPA does

[1] See section on key assumptions for an explanation of the relationship between the C-R function and the relative risks reported in epidemiological studies.

not currently estimate separate mortality changes associated with reductions in daily concentrations of $PM_{2.5}$ in its primary estimate of mortality benefits. Thus, EPA's primary estimate effectively assumes that the ACS cohort study captures all or most mortality impacts due to reductions in both daily levels of $PM_{2.5}$ and long-term average $PM_{2.5}$ concentrations.

Figure 1: Key Steps in Air Quality Based Benefits Analysis



Source: USEPA, Regulatory Impact Analysis for the Clean Air Interstate Rule. EPA-452/R-05-002. March 2005. http://www.epa.gov/cair/pdfs/finaltech08.pdf

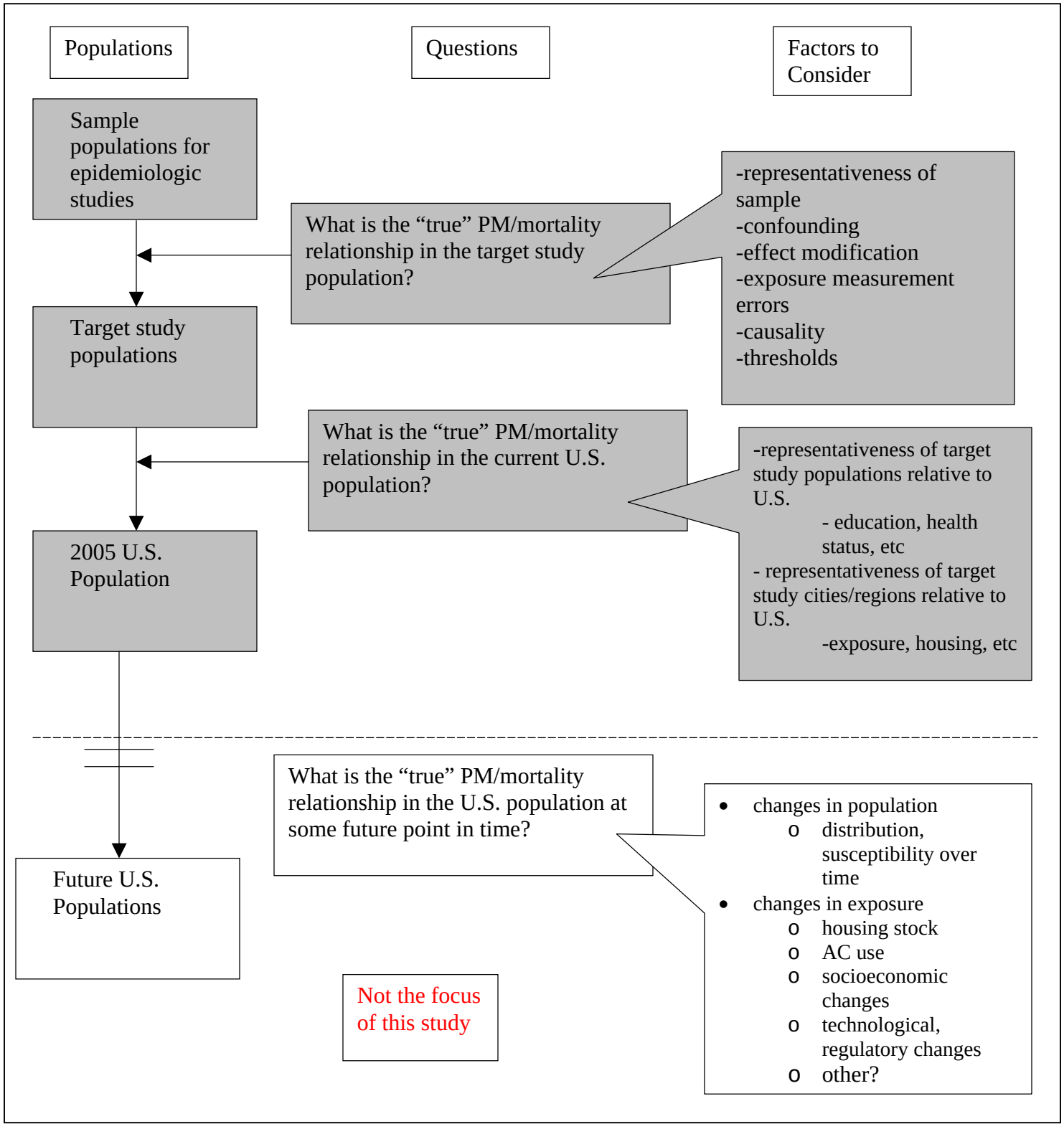
EPA currently quantifies uncertainty in the PM/mortality C-R function using both sensitivity analysis, in which the implications of changing a single parameter are investigated, and a probability distribution based on the central relative risk estimate from the key epidemiological study and the standard error derived from the published confidence interval around that estimate. These parameters are used in benefits models to generate a *distribution* of possible changes in mortality incidence, rather than a single point estimate. Until recently, the standard error was the only quantitative representation of uncertainty in the C-R function captured in these probabilistic analyses.

In its 2002 report, the NAS panel questioned the value of generating distributions based solely on the statistical or stochastic uncertainty represented by published confidence intervals in epidemiological studies. The NAS panel encouraged EPA to attempt to characterize uncertainties in the C-R relationship that may not be captured in those confidence intervals. One suggested method for doing so was through the use of expert judgment.

The expert elicitation process can help generate a more comprehensive assessment of uncertainty because it encourages experts to carefully consider and integrate information and uncertainties from different kinds of scientific studies that inform our understanding of the C-R relationship between $PM_{2.5}$ exposure and mortality. For example, epidemiological studies provide information on the potential magnitude and direction of an association between $PM_{2.5}$ exposures and mortality in large study populations, data which could form the basis of a C-R function. However, epidemiological studies alone cannot provide a complete understanding of the $PM_{2.5}$/mortality relationship. Toxicological studies conducted in laboratory animals, clinical studies of human subjects, and in vitro studies may also be valuable for evaluating the biological plausibility of the epidemiological results and identifying steps in a potential mechanism linking $PM_{2.5}$ exposures with mortality.

None of these study designs provide a perfect measurement of the $PM_{2.5}$/mortality relationship, for a variety of reasons. For example, the accuracy of epidemiologic results may depend on a number of factors including sample selection, the control of confounding, and the quality of exposure measurements. Furthermore, the existing scientific evidence for a PM/mortality relationship may be based on studies conducted in particular regions of the U.S. or in other countries where differences may exist in the underlying populations, historic PM levels, PM composition, and other potentially important factors (see Figure 2). On the other hand, laboratory studies typically employ much smaller sample sizes than epidemiological studies, and thus must expose subjects to much higher concentrations than the ambient levels typically found in the U.S. In addition, toxicological studies often assess effects of exposure on healthy subjects and thus may not reflect the impacts of exposure on susceptible subpopulations (EPA, 2004). Extrapolation of animal toxicity results to humans is also a challenging task. We will be asking you today to discuss and synthesize all of the relevant uncertainties in the evidence from epidemiology, toxicology, and clinical studies as you estimate a $PM_{2.5}$/mortality C-R function to be applied to the entire adult U.S. population.

Figure 2: Inferences about Populations



In 2003 and 2004, EPA conducted a pilot elicitation, the primary objective of which was to explore whether expert judgment, informed by scientific evidence, can yield a more comprehensive estimate of the uncertainty in the C-R function relating mortality to reductions in $PM_{2.5}$ concentrations. Through a sequence of structured discussions and questions, we sought to obtain PM experts' quantitative characterization of uncertainty in the relationship between both long-term and short-term ambient exposure to $PM_{2.5}$ and mortality, in the form of a probability distribution for the percent change in all-cause mortality per 1 $\mu g/m^3$ reduction in $PM_{2.5}$. The goal was to characterize a distribution that would reflect not just sampling error, but other potentially important sources of bias and uncertainty. The peer review of the pilot elicitation was generally favorable, and the results have been used to inform the current elicitation. As noted above, the purpose of the current project is to provide a more complete characterization, both qualitative and quantitative, of the uncertainties associated with the relationship between reductions in ambient $PM_{2.5}$ and mortality.

The elicitation protocol for this study reflects information obtained during the pilot study. For example, while the pilot study asked separate questions about mortality associated with long-term exposures and short-term exposures, and directed experts to provide an estimate for each that was exclusive of the other, it was not always clear from the expert responses that they were able to do so. Because of the difficulties in disentangling mortality impacts associated with reductions in long-term exposures from those associated with short-term exposures, in the current elicitation we are asking that experts provide an estimate of the distribution of the total annualized mortality impact that would occur when annual average $PM_{2.5}$ levels are reduced. We recognize that reductions in annual average $PM_{2.5}$ will in part be comprised of reductions in short-term exposures. We will ask you to walk us through your interpretation of the evidence from whatever kinds of studies you consider to be informative for this question, which may include cohort and time-series epidemiology studies, as well as toxicological and clinical studies.

1.2 Methodology

We plan to elicit your judgments in a series of steps:

1. We will discuss the primary quantitative question about the $PM_{2.5}$/mortality C-R function and the detailed assumptions describing the scenario on which it is based.
2. We will ask you to provide a conceptual model, in words or using diagrams, of the evidence and/or issues that you think will be important in developing your quantitative assessment of the C-R function.
3. Through a series of initial questions, we will document your views on the evidence available to make inferences about the nature and magnitude of potential relationships between exposures to $PM_{2.5}$ and mortality in the U.S., the strengths and weaknesses of that evidence, and the major factors that may be responsible for or could modify the relationships observed.

4. We will elicit a preliminary set of probabilistic judgments from you about the percent change in total all-cause mortality for adults associated with a permanent 1 $\mu g/m^3$ reduction in ambient annual average $PM_{2.5}$ concentrations.
5. We will explore the major issues you have raised, examining their collective influence on your quantitative judgments.

If, in the course of discussing your conceptual approach, a different ordering of the questions suggests itself, the order of the elicitation questions can change.

It is important to recognize that your probabilistic judgments should ultimately reflect your "state of knowledge" about each of these quantities; they should be a function both of what you know and what you do not know as a result of underlying uncertainties in the available evidence.

Note that because the elicitation is often an iterative process, some topics may be discussed more than once.

1.3 Confidentiality Agreement

We will take steps to preserve the confidentiality of your judgments. While your name, a summary of our discussions with you, and your quantitative judgments will all be publicly available, neither the summary nor your quantitative judgments will be associated with your name. Instead, you will be assigned a letter at random that will be used in the documentation and reporting of your judgments.

1.4 Use of Expert Elicitations

Your probabilistic judgments about each of these values will be presented individually by expert (anonymously). In addition, we are exploring whether judgments from this elicitation will be combined and if so, which method is most appropriate for combining judgments.

Part 2. Quantitative Elicitation Question: Preview (20 minutes)

In Part 4 of this protocol, you will be asked the following question:

What is your estimate of the true percent change in annual, all-cause mortality in the adult U.S. population resulting from a permanent 1 μg/m^3 reduction in annual average ambient $PM_{2.5}$ across the U.S.? In formulating your answer, please consider mortality effects of reductions in both long-term and short-term exposures. To characterize your uncertainty in the C-R relationship, please provide the 5th, 25th, 50th, 75th, and 95th percentiles of your estimate.

For the purpose of this elicitation, we are assuming that the "true" percent change in mortality per unit reduction in annual average ambient $PM_{2.5}$ for the adult U.S. population could be known exactly if the ambient $PM_{2.5}$ exposures and mortality experience of all U.S. residents, across all regions, were to be measured perfectly and followed for an appropriate period of time. In essence, this relationship might be considered as a single, national average C-R function that could be applied throughout the U.S. in a benefits analysis.

In the scenario we are focusing on today, the reduction in $PM_{2.5}$ resulting from regulatory action is assumed to be immediate and permanent (see Figure 3). We recognize that some of the change in annual all-cause mortality resulting from a reduction of $PM_{2.5}$ might take several years or more to appear. We are asking about the percent change in mortality that might be expected if the U.S. population, remaining similar in every respect to the current U.S. population, were to reach a new steady state baseline risk of mortality (see schematic representation in Figure 4). We are not asking you to characterize quantitatively the time sequence of any changes although we may be asking your qualitative views about it.

Figure 3: Schematic Depiction of an Immediate and Permanent Reduction in $PM_{2.5}$

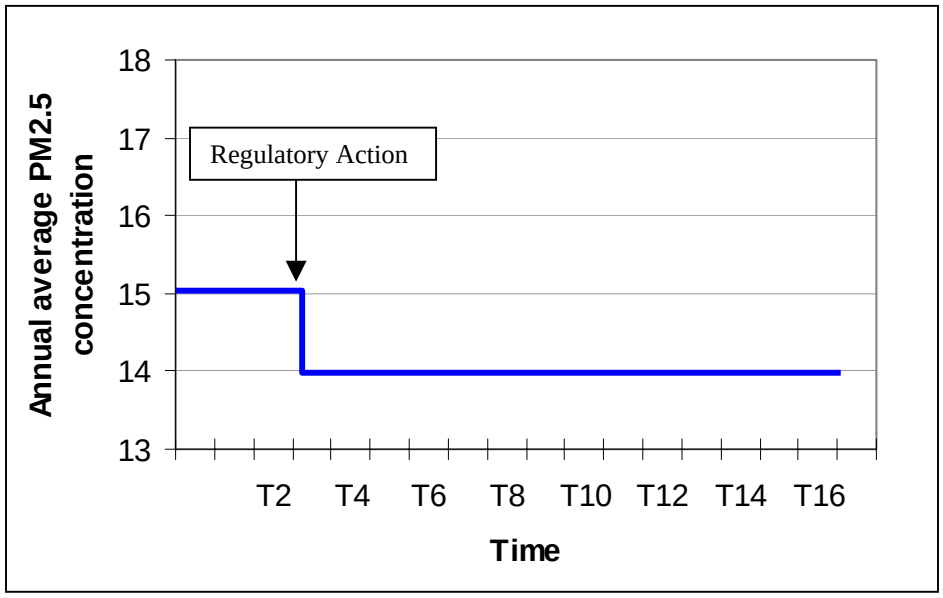
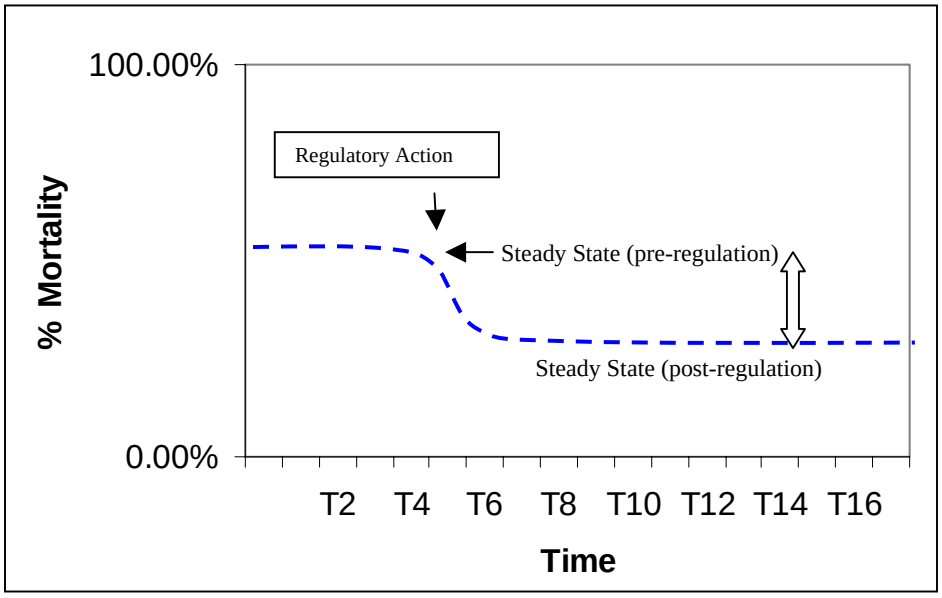


Figure 4: Schematic Depiction of the Reduction in Mortality with a Reduction in Long-term $PM_{2.5}$ Exposure



Assumptions on which your judgments should be conditioned:

- Baseline exposure conditions:
    - The range of baseline annual average $PM_{2.5}$ concentrations is typical of that observed across U.S. metropolitan areas in recent years (i.e., from about 4 to 30 $\mu g/m^3$).
    - The variation in the mix of $PM_{2.5}$ component species is also typical of that observed across U.S. metropolitan areas in recent years (see EPA's *Particle Pollution Report*, 2004 for characterization of the mix of $PM_{2.5}$ components across the U.S.).
    - Baseline concentration distributions of other pollutants, such as nitrogen dioxide, sulfur dioxide, ozone, and carbon monoxide, and other pollutants, are as they currently exist (i.e. as observed in recent years in U.S. metropolitan areas) (see EPA's Air Quality and Trends Report, 2002 for characterization of levels of these other pollutants).
    - Temperature and relative humidity conditions are the same as those that typically occur currently throughout the U.S.
    - Baseline patterns of air conditioning use are represented by recent data (see the US Department of Energy's *Trends in Residential Air Conditioning Usage from 1978-1997*, 2000).
    - The age of the housing stock in the U.S. is represented by estimates from the U.S. Census American Housing Survey from 2003 (see Background Technical Information Pages, Elicitation Aids for information on the median age of housing across the U.S.).[2]
- Historical exposure conditions:
    - Ambient concentration levels of $PM_{2.5}$ and other pollutants reflect historical patterns of pollution across U.S. metropolitan areas (see EPA's *Air Quality and Trends Report*, 2002).
    - Historical trends in housing stock characteristics and air conditioning use are reflected by existing data.
- Regulatory implementation:
    - The reduction in $PM_{2.5}$ resulting from the regulatory action is assumed to be immediate and permanent (see Figure 2).

[2] The age of housing stock may be an indicator of the correlation between indoor and ambient $PM_{2.5}$ levels.

- The reduction will affect all areas, not just non-attainment areas.

- The regulatory strategies implemented to achieve this reduction in $PM_{2.5}$ could include a variety of regulatory measures that could affect levels of precursors and/or primary particles. Such measures could include regulations on motor vehicles, control of emissions from major industrial sources or utilities, or local measures to comply with fine particle standards.

- We cannot specify which $PM_{2.5}$ components will be reduced (e.g. reductions may occur in any or all of $PM_{2.5}$ components such as nitrates, sulfates, organic or elemental carbon, metals, or primary particles). Please assume for the purpose of this exercise that the regulatory action will achieve proportional reductions in all $PM_{2.5}$ components.

- The impact of the regulatory action on co-pollutant concentrations is not known/specified and thus remains a source of uncertainty.

- The temporal pattern of concentration changes producing the specified 1 $\mu g/m^3$ reduction in annual average ambient $PM_{2.5}$ is uncertain, but likely reflects a combination of reductions in peak daily (24-hour) $PM_{2.5}$ exposures and longer-term reductions in $PM_{2.5}$ levels. Both of these effects should be considered when evaluating the mortality effects of a 1 $\mu g/m^3$ reduction in annual average ambient $PM_{2.5}$.

- U.S. Population characteristics:

  - U.S. adult population (25 years and older).

  - The distribution of susceptible individuals across the U.S. population reflects current patterns of susceptibility. These patterns are assumed to remain constant.

  - The impact of projected changes in the age distribution in the population over time on mortality will be incorporated directly into modeling of benefits.

- Time course of mortality changes: Assumptions about the time course of mortality changes, sometimes referred to as the "cessation lag," are incorporated as separate assumptions into EPA's benefits assessments and are not the focus of this elicitation.

Do you have any questions or concerns at this point regarding the specification of this problem?

Part 3. Conditioning Step: Preliminary Questions (3 hours, 20 minutes)

To assist you in providing quantitative probability judgments, we want to help you bring to mind the relevant evidence so that you may consider it systematically. You will need to identify the relevant evidence (including evidence from epidemiological, clinical, toxicological, and exposure studies and theories regarding biological and toxicological mechanism), and consider any sources of uncertainty, error or bias that might influence your interpretation of the evidence.

We have identified several factors that you may like to discuss:

- **scientific (epidemiological, clinical, toxicological, etc.) evidence for physiological mechanisms and causes of death**
- **role of study design in capturing effects of annual average $PM_{2.5}$ exposures**
- **scientific evidence on the magnitude of the $PM_{2.5}$/mortality relationship**
- **confounding**
- **effect modification**
- **exposure issues**
- **evidence for a causal relationship**
- **thresholds**
- **other influential factors (e.g., selection bias, statistical methodology, publication bias)**

We would like you to describe the importance of these factors, and any others that you would like to add, in terms of their contribution to your understanding of and uncertainty about the mortality effects of $PM_{2.5}$. The order here is not intended to indicate relative importance.

The following set of preliminary questions was designed to provide an explicit and orderly consideration of the evidence that might factor into your judgments about the $PM_{2.5}$ mortality relationship. We recognize that individual experts may have other logical approaches for developing his or her judgments. **If you would like to discuss the questions in the protocol in a different order, please let us know.**

3.1 Mechanisms for Effects from Exposure to $PM_{2.5}$ (20 minutes)

In this section, we would like you to discuss the scientific evidence on the possible mechanisms underlying the mortality effects of both short-term and long-term exposures to $PM_{2.5}$ on mortality. While the protocol asks about mechanisms for long- and short-term exposures separately, it can also accommodate a discussion of the effects of both types of exposures together, if you believe the evidence is more consistent with that approach.

Please indicate which approach you wish to take:

3.1.1 Mechanisms for Effects from Long-term Exposures to $PM_{2.5}$

Please state what you believe to be the most compelling current theory or theories concerning potential causes of death and the possible biological or physiological mechanisms for those causes of death resulting from long-term exposures to $PM_{2.5}$. Please discuss the major causes of death in order of their importance (e.g., contribution to total mortality). What existing studies and/or evidence are most influential in informing your views? (Please fill out the table on the next page.)

Causes of Death	Study(ies) (Author(s), Date)	Major Findings	Strengths/Limitations
1.			
2.			
3.			
4.			

3.1.2 Mechanisms for Effects from Short-term Exposures to $PM_{2.5}$

Please state what you believe to be the most compelling current theory or theories concerning potential causes of death and the possible biological or physiological mechanisms for those causes of death resulting from short-term exposures to $PM_{2.5}$. Please discuss the major causes of death in order of their importance (e.g., contribution to total mortality). What existing studies and/or evidence are most influential in informing your views?

Causes of Death	Study(ies) (Author(s), Date)	Major Findings	Strengths/Limitations
1.			
2.			
3.			
4.			

3.2 Conceptual Framework for Mortality Effects of Short-term and Long-Term $PM_{2.5}$ Exposures (20 minutes)

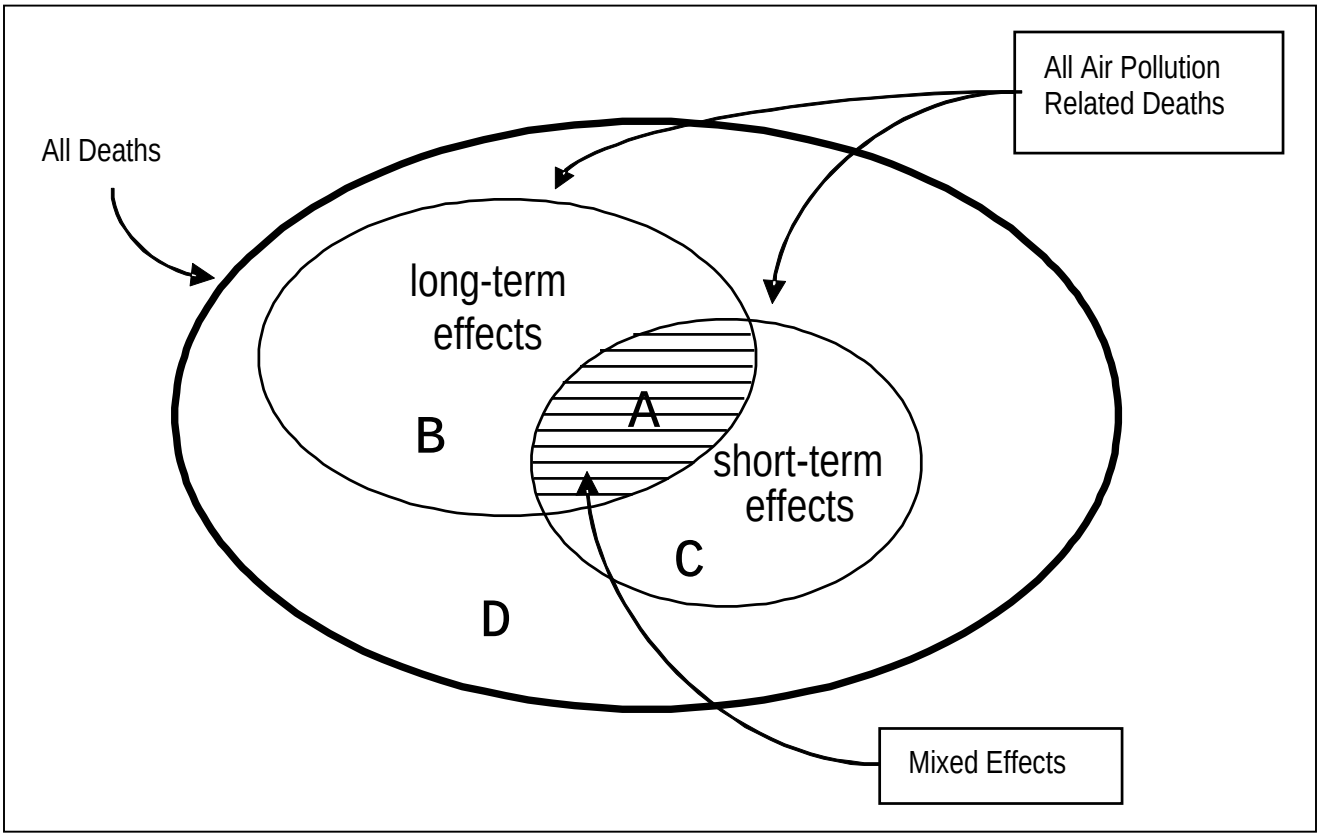
Because the goal of this elicitation is ultimately to obtain your quantitative estimate for the total annualized mortality effect of changes resulting from both short-term and long-term exposures to $PM_{2.5}$, we would like to understand the relationship between the two types of effects with the help of a conceptual framework.

It is difficult to derive an estimate of the total annualized mortality effect of reductions in ambient annual average $PM_{2.5}$ that may reflect reductions in both short-term peak and long-term average exposures to $PM_{2.5}$. Cohort studies focus primarily on analyzing the impact of long-term exposures to $PM_{2.5}$ but may also capture some of the impact of short-term variations in exposure during the cohort follow-up period. Time-series studies analyze the impacts of daily or short-term variations in PM concentrations and can characterize the cumulative impact of exposure over a few days, but not over a longer period of time. Those who rely exclusively on cohort studies may not account for all of the mortality impacts of short-term exposures, and therefore, may underestimate total mortality impacts. Those who rely on a sum of effects estimated in both cohort and time-series studies may overestimate mortality impacts. One approach to sorting out the total benefits of a reduction in annual average $PM_{2.5}$ is to consider carefully the relevant information provided by each type of study and to then combine them. This process itself may introduce uncertainty into your eventual quantification of benefits.

We will explore this question in greater detail momentarily. But first, Kunzli et al. (2001) presents a conceptual framework using Venn diagrams to describe the relationship between the deaths attributable to long-term exposures and those attributable to short-term exposures to fine particles (see Figure 5).

Your own views on the interaction between the mortality effects of short-term and long-term PM exposures may differ from Kunzli et al. (2001). If so, we encourage you to discuss and use alternate frameworks that more closely match your own ideas.

Figure 5: Conceptual Framework for Short-Term and Long-Term Mortality Effects



Graphic illustration of deaths due to ambient air pollution in a population, including cases related to both long-term and short-term air pollution. Exposure may affect the occurrence (event) of death ("short-term effects") and/or increase the underlying frailty in the population ("long-term effects"), leading to a shortening of lifetime. The four different types of cases, A, B, C, and D correspond to the categories given on page 17 of the protocol. Circle sizes do not reflect relative effects. (Adapted from Kunzli et al., 2001).

Kunzli et al. (2001) defined four categories of deaths attributable to air pollution:

Category of Cases	Impact of Air Pollution	
	Underlying frailty due to air pollution	Occurrence of death (event) triggered by air pollution
A	Yes	Yes
B	Yes	No
C	No	Yes
D	No	No

Where:

A: Air pollution increases both the risk of underlying diseases leading to frailty and the short-term risk of death among the frail. For example, patients with chronic bronchitis that has been enhanced by long-term air pollution exposure may be hospitalized with an acute air pollution-related exacerbation of their illness leading to death shortly afterward.

B: Air pollution increases the risk of chronic diseases leading to frailty but is unrelated to timing of death. For example, a person's suffering from chronic bronchitis may be enhanced by long-term ambient air pollution exposure but the person may die due to acute pneumonia acquired during a clean air period.

C: Air pollution is unrelated to risk of chronic disease but short-term exposure increases mortality among persons who are frail. For example, a person with diabetes mellitus may be susceptible to heart attacks due to long-standing coronary disease; in such a case, an air pollution episode may trigger the fatal infarction leading to death.

D: Neither underlying chronic disease nor the event of death is related to exposure to air pollution.

3.2.1 Do the categories A-D make sense to you as a way of defining the effects of long- and short-term exposures to $PM_{2.5}$ on health? **Yes/No**

3.2.2 If not, how would you alter them?

A

B

C

D

3.2.3 Does a Venn diagram adequately represent the relationships between these types of cases? **Yes/ No**

3.2.4 If yes, please draw for us the representation that best represents your views.

3.2.5 If not, please describe your views on these relationships schematically or mathematically.

3.3. The Role of Epidemiological Study Design in Characterizing the Total Impacts of $PM_{2.5}$ Exposures on Mortality (20 minutes)

For the purpose of policy analysis and regulation, the impact of exposures to $PM_{2.5}$ would ideally be separable into those impacts due solely to short-term fluctuations and those due solely to long-term exposures. However, we recognize that current epidemiological study designs (e.g., time-series and cohort studies) may not completely distinguish between these two types of effects. Bearing in mind your earlier discussion of the mechanisms underlying effects of long-term and short-term exposures and the conceptual framework you specified in Question 3.2, we would now like you to discuss the kinds of PM-related mortality likely to be captured by different epidemiological study designs.

3.3.1 Tell us which epidemiological study design(s), either alone or in combination, are most useful for estimating the total annual mortality change related to a permanent reduction in ambient $PM_{2.5}$ concentrations (taking into account both reductions in peak and annual average concentrations, as you prefer). Please list types of studies in the table below in order of preference.

3.3.2 Please discuss the extent to which you feel each of these study designs captures effects from short-term and long-term exposures. For example, what proportion, if any, of the mortality effects identified in cohort studies do you believe may represent the influence of short-term exposures? Please discuss your rationale and any evidence you may have.

Study Design	Type of Effects Captured (e.g., short-term, long-term, or both)

3.4 Epidemiologic Evidence for the Impact of Exposures to $PM_{2.5}$ on Mortality (20 minutes)

We would like to focus now on the epidemiological evidence from U.S. or international studies that you find most informative for your initial quantitative judgments about the estimated percent change in all-cause mortality related to a reduction in annual average ambient $PM_{2.5}$ concentrations.

3.4.1 Before moving on to discussions of particular studies, please list the characteristics of an ideal epidemiological study or studies that would most accurately characterize the change in total annual all-cause mortality in the U.S. population related to a reduction in annual average ambient $PM_{2.5}$ concentrations.

3.4.2 Next, we would like you to review briefly the epidemiological studies that you think are most informative about the percent change in all-cause mortality related to a reduction in annual average ambient $PM_{2.5}$ concentrations. *We are interested in what you believe to be the strongest evidence for as well as against a relationship between $PM_{2.5}$ and mortality.*

Discuss the reasons for your choices, the strengths and the limitations of each study. For example, how do these studies compare to your "ideal" study?

Study (author, date)	Key findings	Strengths	Limitations

In the next three questions (on confounding, effect modification, and exposure issues), we would like you to evaluate the impact of various sources of bias/uncertainty on the particular study or studies (from Section 3.4) that will inform your quantitative estimates about the $PM_{2.5}$ mortality relationship for the U.S. population.

We recognize that each of these questions may require thinking both about how well each study estimates the true relative risks in its target population as well as about the generalizability of each study's results to the U.S. population.

3.5 Confounding (20 Minutes)

We want to understand the influence, if any, of potential confounding in the specific studies (identified in Section 3.4) that you will use to inform your quantitative estimates. In particular, we are interested in understanding what influence they may have on your judgments concerning the form, magnitude, and uncertainty in the C-R function for mortality related to ambient $PM_{2.5}$ exposure.

3.5.1 Using cards available at the interview please create a set of cards identifying what you believe to be the most influential confounders of the relationship between exposure to $PM_{2.5}$ and mortality. As an example, some variables that have been included in epidemiological analyses are listed on cards on page 27.

3.5.2 Define each confounder and discuss the theoretical rationale (e.g., biological or toxicological mechanism) or empirical evidence (e.g., clinical, epidemiological, animal, or exposure studies) for the impact of each potential confounder on the $PM_{2.5}$/mortality effect.

3.5.3 Separate the cards into groups depending on whether you think they have been adequately controlled for in the studies that inform your quantitative estimates (e.g., definitely have, definitely have not, and don't know). Discuss the evidence on which you are relying for your judgments.

3.5.4 Take the cards for those confounders that you do not believe have been adequately controlled for in the studies that inform your quantitative estimates. We would like to understand how you believe that the particular treatment of each confounder could have affected the relative risks (RRs) reported in these studies. Please separate into groups according to whether you think each confounder is likely to produce 1) an upward bias (e.g., resulting in an overestimate of the RR), 2) a downward bias (e.g., resulting in an underestimate of the RR), or 3) an bias of uncertain direction.

3.5.5 Taking into account all of the evidence you have discussed with us, please assign each of the potential confounders you placed in the "overestimate" group a score on a scale of 1 to 3, where a score of 1 indicates minimal effect of the variable in question on the mortality RR estimate and a score of 3 indicates a major upward bias. If you feel that you cannot assign a score to a specific confounder, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that confounder and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.5.6 Taking into account all of the evidence you have discussed with us, please assign each of the potential confounders you placed in the "underestimate" group a score on a scale of 1 to 3, where a score of 1 indicates minimal effect of the variable in question on the mortality relative risk estimate and a score of 3 indicates a major downward bias. If you feel that you cannot assign a score to a specific confounder, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that confounder and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.5.6 Finally, please discuss briefly the importance of confounders for which you are uncertain about the direction of influence.

SUMMARY OF KEY POTENTIAL CONFOUNDERS IN PM MORTALITY EPIDEMIOLOGICAL STUDIES

	Potential Confounders	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	

3.6 Effect Modification (20 minutes)

We want to understand the influence, if any, of effect modification in the studies that are most likely to inform your quantitative estimates (identified in Section 3.4). In particular, we are interested in understanding what influence effect modifiers in these studies may have on your judgments concerning the form, magnitude, and uncertainty in the C-R functions for annual all-cause mortality in the U.S. that would result from a 1 $\mu g/m^3$ reduction in annual average ambient $PM_{2.5}$ exposure.

3.6.1 Using cards available at the interview, please create a set of cards identifying what you believe to be the most influential effect modifiers of the relationship between exposure to $PM_{2.5}$ and mortality. As an example, some variables that have been included in epidemiological analyses are listed on cards on page 27.

3.6.2 Define each effect modifier and discuss the theoretical rationale (e.g., biological or toxicological mechanism) or empirical (e.g., clinical, epidemiological, animal, or exposure studies) evidence for the impact of each potential effect modifier on the $PM_{2.5}$/mortality effect.

3.6.3 Please separate the cards into groups depending on how you believe each effect modifier could affect the relative risks (RRs) from the studies on which you are relying for your judgments, as compared to the hypothetical effect for the full adult U.S. population. For example, do you think the RRs reported in the study are likely to be underestimates or overestimates of the adult U.S. effect? Or are you uncertain of the direction of bias?

3.6.4 Taking into account all of the evidence you have discussed with us, please assign each of the potential effect modifiers you placed in the “overestimate” group a score on a scale of 1 to 3, where a score of 1 indicates a minimal overestimate of effect on extrapolation of RRs to the full adult U.S. population and a score of 3 indicates a major overestimate resulting from extrapolation without adjustment for the effect modification. Use the table below to record your answers. If you feel that you cannot assign a score to a specific effect modifier, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that effect modifier and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.6.5 Taking into account all of the evidence you have discussed with us, please assign each of the potential effect modifiers you placed in the "underestimate " group a score on a scale of 1 to 3, where a score of 1 indicates a minimal underestimate of effect on extrapolation of RRs to the full adult U.S. population and a score of 3 indicates a major underestimate resulting from extrapolation without adjustment for the effect modification. Use the table below to record your answers. If you feel that you cannot assign a score to a specific effect modifier, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that effect modifier and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.6.6 Finally, please discuss briefly the importance of effect modifiers for which you are uncertain about the direction of influence.

SUMMARY OF KEY POTENTIAL EFFECT MODIFIERS IN PM MORTALITY EPIDEMIOLOGICAL STUDIES

	Potential Effect Modifiers	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	

Potential Confounders and/or Effect Modifiers:

Alcohol Use	Smoking History
Diet	SES Variables (specify which ones you think are important)
Occupational History	Pre-Existing Health Status
Gaseous co-pollutants (specify)	Temporal Trends
Weather	Other (specify):

3.7 Exposure Issues (20 minutes)

We want to understand the influence, if any, of potential exposure misclassification or exposure error in the studies that inform your quantitative estimates (identified in Section 3.4). In particular, we are interested in understanding what influence they may have on your judgments concerning the form, magnitude and uncertainty in the C-R functions for mortality related to ambient $PM_{2.5}$ exposure.

3.7.1 Using cards available at the interview, please create a set of cards identifying what you believe to be the most influential exposure issues. As an example, some variables that have been included in epidemiological analyses are listed on cards on page 31.

3.7.2 Define each exposure issue and discuss the theoretical rationale (e.g., biological or toxicological mechanism) or empirical (e.g., clinical, epidemiological, animal, or exposure studies) evidence for the impact of each potential exposure issue on the $PM_{2.5}$/mortality effect. In addition, please state whether the exposure issue affects the internal validity of the study, or the generalizability of the study results to the U.S. population.

3.7.3 Please separate the exposure issues into groups based on whether you think an individual exposure issue is likely to result in 1) an upward bias resulting in an overestimate of the relative risk, 2) a downward bias resulting in an underestimate of the relative risk, or 3) a bias of uncertain direction.

3.7.4 Taking into account all of the evidence you have discussed with us, please assign each of the potential exposure issues you placed in the "overestimate" group a score on a scale of 1 to 3, where a score of 1 indicates minimal effect of the variable in question on the mortality relative risk estimate and a score of 3 indicates a major upward bias. Use the table below to record your answers. If you feel that you cannot assign a score to a specific exposure issue, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that exposure issue and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.7.5 Taking into account all of the evidence you have discussed with us, please assign each of the potential exposure issues you placed in the "underestimate" group a score on a scale of 1 to 3, where a score of 1 indicates minimal effect of the variable in question on the mortality relative risk estimate and a score of 3 indicates a major downward bias. Use the table below to record your answers. If you feel that you cannot assign a score to a specific exposure issue, you may choose to leave that column blank. However, please explain why you feel you cannot assign a score to that exposure issue and describe what information would be needed to be able to better determine the likely magnitude of its impact.

3.7.6 Finally, please discuss briefly the importance of exposure issues for which you are uncertain about the direction of influence.

SUMMARY OF KEY POTENTIAL EXPOSURE ISSUES IN PM-MORTALITY EPIDEMIOLOGICAL STUDIES

	Potential Effect Modifiers	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	
Study (author, date):	**Overestimated RR**	**Score (1-3)**
	Underestimated RR	**Score (1-3)**
	Uncertain Direction of Bias	

Potential Exposure Issues:

Central site vs. individual site monitors	Time course of relevant exposures
Particle Size (e.g., PM_{10} vs. $PM_{2.5}$)	Concentrations vs. Exposure
Measurement error (specify)	Regional Housing Differences (specify)
Differences in PM composition or sources (regional, temporal, etc.)	Other (Specify):

3.8 Causality (20 minutes)

We want to understand whether and how your views on the strength of the evidence supporting a causal relationship between reductions in annual average exposures to ambient $PM_{2.5}$ and changes in annual all-cause mortality may factor into your C-R function.

The goal of this section is to ask you for your assessment of the overall probability of a causal relationship between reductions in annual average exposures to ambient $PM_{2.5}$ and changes in annual all-cause mortality. One way to conceptualize your response is that it represents the probability of there being one or more causal relationships linking reductions in annual average $PM_{2.5}$ levels with changes in mortality. This probability includes the following:

- the likelihood that there is a causal relationship with short-term exposure reductions only;
- the likelihood that there is a causal relationship with long-term exposure reductions only; and
- the likelihood that both short-term and long-term causal relationships are responsible for the observed mortality results.

This overall probability of a causal relationship is consistent with the C-R function describing the relationship between reductions in annual average exposures to ambient $PM_{2.5}$ and changes in annual all-cause mortality that we are eliciting in Section 4 of the protocol.

3.8.1 Please specify what types of scientific evidence you believe are required to support the conclusion that this is a causal relationship.

3.8.2 What evidence is available, either to support or counter a conclusion of a causal relationship between reductions in annual average $PM_{2.5}$ exposure (including reductions in short- and/or long-term exposures) and changes in mortality at the $PM_{2.5}$ levels currently experienced in the U.S. (e.g., annual averages of 4-30 µg/m)?

To the extent that your depiction of the relationship between annual average $PM_{2.5}$ exposures and mortality are associated with both reductions in peak exposures and reductions in long-term exposures, please describe how your views about causality differ for short-term vs. long-term exposures:

Do you wish to make such a distinction? **Yes/No.**

If so, please make any distinctions clear.

Study (author, date)	**Type of evidence**	**Strengths**	**Limitations**

3.8.3 Given all the evidence we have discussed, we would like you to characterize the likelihood that there is a causal relationship between reductions in annual average exposures to $PM_{2.5}$ (including reductions in short- and/or long-term exposures) and changes in mortality at $PM_{2.5}$ levels currently experienced in the U.S. (e.g., annual averages of 4-30 µg/m^3). Please express your answer as a range of probabilities.

Range: Min___ Max ____

Do you think there is a value that is more likely than any other in this range?

Please discuss your rationale for the probability value(s) you give.

3.9 Thresholds (20 minutes)

We want to understand whether and how thresholds may factor into your C-R function for annual all-cause mortality related to reductions in annual average exposures to ambient $PM_{2.5}$.

Thresholds may be defined for individuals (i.e., the concentration below which a particular individual would experience no increased risk of death), for a population (i.e., the concentration below which no member of the population would experience an increased risk of death), and for a particular study (i.e., the concentration below which no increased risk of death is detected). For this analysis, we are interested in population thresholds. Recall that in Part 2 of the protocol, we assume the following about the US population: 1) the population is 25 years of age or older, 2) the distribution of susceptible individuals across population reflects current patterns of susceptibility, and 3) this pattern will remain the same. The impact of projected changes in the age distribution on mortality, however, is incorporated directly into EPA's benefits model.

3.9.1 What are the conceptual and scientific bases (epidemiological, clinical, toxicological, etc.) *for* or *against* the hypothesis of population thresholds in the range of 4-30 $\mu g/m^3$ annual average $PM_{2.5}$?

To the extent that your depiction of the relationship between reductions in annual average $PM_{2.5}$ exposures and mortality is based on both reductions in peak and long-term exposures, do your views about thresholds differ for short-term vs. long-term exposures? **Yes/No.**

If so, please make any distinctions clear.

3.9.2 On a conceptual basis, do you think it is likely that a population threshold for mortality related to reductions in annual average $PM_{2.5}$ exist in the exposure range(s) typical of the U.S.? **Yes/No**

3.9.3 What types of studies do you believe are most appropriate for determining values for potential population thresholds for $PM_{2.5}$ and mortality (e.g., epidemiological, clinical, toxicological)?

3.9.4 In practice, do you believe that a population threshold for annual all-cause mortality related to $PM_{2.5}$ exposures is detectable in any of these studies that are currently available? **Yes/No**

If so, please list the studies that you feel provide evidence for specific population thresholds in the following table:

Study (Author, date)	Type of Study	Threshold ($\mu g/m^3$)	Strengths/Limitations

3.9.5 The quantitative elicitation of your judgments in the next section of the protocol will focus on the total mortality that might result from a permanent reduction in annual average $PM_{2.5}$ concentrations, including reductions due to both long-term and short-term exposures. Thus, at this point we would like you to begin integrating your thinking about both long-term and short-term exposure effects in developing your answers.

On the basis of your interpretation of the scientific evidence, as you have just discussed, do you want to incorporate a threshold into your characterization of the concentration response relationship? **Yes/No.**

Explain your choice briefly.

If you answered yes to Question 3.9.5, please continue to Question 3.9.6. If you answered no, please proceed to Section 3.10 of the protocol.

3.9.6 If you do want to incorporate a threshold please specify the method you would like to use. The following are some examples of ways to quantify a population threshold.

> **Option 1:** Create a probability density function for the location of a population threshold level for mortality related to $PM_{2.5}$ exposures in the 4-30 $\mu g/m^3$ range that is the focus of our analysis. This would include specifying one or more PM ranges and your assessment of the probability that the population threshold falls within that interval. The probabilities for all of the ranges selected should sum to one.
>
> **Option 2:** Specify a specific type of distribution (e.g., uniform, triangular) and its parameters to express your uncertainty in the threshold level.

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3.10 Other Influential Factors (20 minutes)

We want to understand whether and how any other sources of uncertainty, error, or bias, other than the ones we have discussed so far in Part 3 of the protocol, might influence your interpretation of the evidence (e.g., selection bias, statistical methodology, differential impacts of $PM_{2.5}$ sources and/or $PM_{2.5}$ components, publication bias). Please define each factor and discuss the theoretical rationale or empirical evidence for the impact of each on the $PM_{2.5}$/mortality effect. In addition, describe the magnitude and direction of the bias, if known.

PART 4. ELICITATION OF QUANTITATIVE JUDGMENTS (3 hours)

What is your estimate of the true percent change in annual, all-cause mortality in the adult U.S. population resulting from a permanent 1 μg/m^3 reduction in annual average ambient $PM_{2.5}$ across the U.S.? In formulating your answer, please consider mortality effects of both reductions in long-term and short-term exposures. To characterize your uncertainty in the C-R relationship, please provide the 5th, 25th, 50th, 75th, and 95th percentiles of your estimate.

Minimum	5th %ile	25th %ile	50th %ile	75th %ile	95%ile	Maximum

You will have the opportunity to specify whether and how the $PM_{2.5}$/mortality C-R function differs across the concentration range we are asking about (4-30 μg/m^3). You may chose to specify a single C-R function across the entire study range, or you may specify multiple C-R functions for specific segments of the study range.

4.1 Shape of the C-R Function (20 minutes)

4.1.1 Before we work on your quantitative response, we would like to begin by having you describe your C-R function in specific terms, either using a particular mathematical form or by sketching the general shape of the C-R function for the range of $PM_{2.5}$ we have specified in this question. For instance, do you think the function is the same over the whole range, or does it differ depending on the range?

It is critical in answering this question that the basis for your function be made clear. For example, please state whether you are assuming an underlying log-linear or other C-R function, and if you prefer to think initially about changes in mortality rates, relative risks, or percent changes in excess mortality.

Please specify your assumptions:

4.1.2 Please discuss the conceptual basis for your response as well as identify any evidence (epidemiological, clinical, toxicological, statistical, etc.) on which you are relying on to make your judgment about the shape of the C-R function:

Study (author, date)	Type of evidence	Strengths/Limitations

4.2 Causal Relationship and the Structure of the Elicitation (20 minutes)

If you have expressed the view that there is a non-zero probability of a causal relationship between mortality and reductions in annual average exposures to $PM_{2.5}$, it is important to understand how you will factor this into your quantitative characterization of the C-R function below. For example, you could reflect your views on the causal relationship directly in the percentiles of your uncertainty distribution. Alternatively, you may provide a C-R distribution conditional on the existence of a causal relationship. We would then combine that distribution with your probability estimate for the existence of a causal relationship (please refer to the Background Technical Information Pages, Elicitation Aids, for examples of how to incorporate causality into the C-R function).

Please describe for us how you intend to incorporate your view about causality into your quantitative judgments:

4.3 Threshold and the Structure of the Elicitation (20 minutes)

If you have expressed the view that there is a non-zero probability of a threshold within the concentration range of interest, it is important to understand how you will factor this into your quantitative characterization of the C-R function below. For example, you could reflect your views on the threshold directly in the percentiles of your uncertainty distribution. Alternatively, you may provide a C-R distribution conditional on the existence of a threshold. We will then combine that distribution with your probabilities of a threshold (please refer to the Background Technical Information Pages, Elicitation Aids, for examples of how to incorporate threshold into the C-R function)

Please describe for us how you intend to incorporate your view about a threshold into your quantitative judgments.

4.4 Elicitation of Specific Percentiles (90 minutes)

As you develop your quantitative responses to this question, it is very important that we understand the basis for each numerical value you give at each percentile of the uncertainty distribution.

Earlier in the protocol (Section 3) we asked specific questions designed to help think about the theories, relationships, and evidence as well as the methodological limitations of existing studies that may have informed your conceptual and quantitative judgments about uncertainty.

In this section, we will build on your earlier responses as you describe:

- any quantitative factors, including particular statistical values, that inform your judgments about magnitude and uncertainty in the CR relationship
- the studies or other evidence on which you base your judgments
- the process by which you arrive at particular quantitative judgments

To help structure this process, we will ask you to consider the following questions:

- ***What do you think the plausible upper bound might be for the C-R relationship and why? What data might you use to bound the relationship?***

    - ***Assuming the mean $PM_{2.5}$ concentration in the U.S., what does your maximum estimate imply for the total number of annual deaths from all causes in the U.S. attributable to $PM_{2.5}$?***

- ***Similarly, how might you approach estimating the 95%ile? What evidence or theory guides your thinking?***

    - ***How do your beliefs about confounding, effect modification, exposure issues, etc. inform your thinking?***

- ***What do you think the plausible lower bound for this relationship might be? What evidence or theory guides your thinking about this value?***

    - ***For example, how do your beliefs about confounding, effect modification, exposure issues, thresholds or causal relationships inform your thinking?***

- ***Similarly, how might you approach estimating the 5%ile? What evidence or theory guides your thinking?***

    - ***For example, how do your beliefs about thresholds or causal relationships inform your thinking?***

- ***How do you approach developing an interquartile range? What evidence or theory guides your thinking about these values?***

- ***What is the evidence or theory that guides your thinking about the likely median of your distribution for the CR relationship?***

In particular, we will be working with the C-R function that you specified in Section 4.1 above. It is important that we are clear about the form of that function, the parameter(s) you are using to describe it and the range over which it applies. If you have decided to characterize the C-R relationship separately for specific $PM_{2.5}$ ranges, we will ask you to quantify the uncertainty in the C-R relationship for each range.

4.5 Collective Impact of Potential Sources of Bias and Uncertainty (30 minutes)

Now we would like to understand more explicitly how each of the factors (confounding, effect modification, exposure issues) discussed in sections 3.4-3.6 might have influenced your quantitative judgments.

We will begin by recalling the top 5 (or however many you identified previously as influential) cards you identified earlier in the protocol in response to our questions about confounding, effect modification, exposure issues, etc. We will arrange them all on a new chart with respect to their independent influence on estimating the $PM_{2.5}$/mortality relationship, as you indicated in our earlier discussions. As you array the factors in order of magnitude within each category (for example, all those that you designated as "likely to overestimate the ***true, but unknown*** PM/mortality response"), please discuss how you have taken these factors into account in your probability distribution.

This is an appropriate time to adjust your C-R function if you feel that it does not adequately account for either the magnitude or direction of the influence of the confounders, effect modifiers, and exposure issues, etc. that you think are most important.

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APPENDIX B: BRIEFING BOOK MATERIALS

Click on citation to open file.

DRAFT BRIEFING BOOK CONTENTS

Long-Term Mortality Cohort

U.S. Studies

Abbey, D. E., N. Nishino, et al. (1999). "Long-term inhalable particles and other air pollutants related to mortality in nonsmokers." American Journal of Respiratory and Critical Care Medicine 159(2): 373-382.

Abbey, D. E., P. K. Mills, et al. (1991). "Long-term ambient concentrations of total suspended particulates and oxidants as related to incidence of chronic disease in California 7th-Day-Adventists." Environmental Health Perspectives 94: 43-50.

Dockery, D. W., C. A. Pope, et al. (1993). "An association between air-pollution and mortality in 6 United States cities." New England Journal of Medicine 329(24): 1753-9.

Enstrom, J.E. (2005) "Fine particulate air pollution and total mortality among elderly Californians, 1973-2002." Inhalation Toxicology 17: 803-816.

Lipfert, F. W., H. M. Perry, et al. (2000). "The Washington University-EPRI veterans' cohort mortality study: Preliminary results." Inhalation Toxicology 12: 41-73.

McDonnell, W. F., N. Nishino-Ishikawa, et al. (2000). "Relationships of mortality with the fine and coarse fractions of long-term ambient PM_{10} concentrations in nonsmokers." Journal of Exposure Analysis and Environmental Epidemiology 10(5): 427-36.

Pope, C. A., M. J. Thun, et al. (1995). "Particulate air-pollution as a predictor of mortality in a prospective-study of US adults." American Journal of Respiratory and Critical Care Medicine 151(3): 669-74.

Pope, C. A., R. T. Burnett, et al. (2002). "Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." Journal of the American Medical Association 287(9): 1132-41.

International Studies

Filleul, L, V. Rondeau, et al. (2005) "Twenty five year mortality and air pollution: results from the French PAARC survey." Occupational and Environmental Medicine 62(7): 453-60.

Hoek, G.B., B. Brunekreef, et al. (2002). "Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study." Lancet 360(9341): 1203-9.

Cohort Reanalysis

Abrahamowicz, M., T. Schopflocher, et al. (2003) "Flexible modeling of exposure-response relationship between long-term average levels of particulate air pollution and mortality in the American Cancer Society Study." Journal of Toxicology and Environmental Health 10(16-19): 1625-54.

Cakmak, S., R.T. Burnett, et al. (2003) "Spatial regression models for large-cohort studies linking community air pollution and health." Journal of Toxicology and Environmental Health 66(16-19): 1811-23.

Hoover, B.K., D.E. Foliart, et al. (2003). "Retrospective data quality audits of the Harvard Six Cities and American Cancer Society studies." Journal of Toxicology and Environmental Health 66(16-19): 1553-61.

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Krewski, D. R.T. Burnett, et al. (2004). "Validation of the Harvard Six Cities Study of particulate air pollution and mortality." New England Journal of Medicine 350(2): 198-9.

Krewski D., R.T. Burnett, et al. (2005a). "Reanalysis of the Harvard Six Cities Study, part I: validation and replication." Inhalation Toxicology 17(7-8): 335-42.

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Villeneuve, P.J., M.S. Goldberg, et al. (2002). "Fine Particulate Air Pollution and All-Cause Mortality within the Harvard Six-Cities Study: Variations in Risk by Period of Exposure." Annals of Epidemiology 12(8): 568-76.

Short-Term Mortality

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Daniels, M.J., F. Dominici, et al. (2004). "The National Morbidity, Mortality, and Air Pollution Study Part III: PM_{10} concentration-response curves and thresholds for the 20 largest US cities." Research Report 94, Health Effects Institute, Boston, MA.

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Stieb, D.M., S. Judek, et al. (2002). "Meta-analysis of time-series studies of air pollution and mortality: Effects of gases and particles and the influence of cause of death, age, and season." Journal of the Air and Waste Management Association 52(4): 470-84.

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Zanobetti, A., J. Schwartz, et al. (2003). "The temporal pattern of respiratory and heart disease mortality in response to air pollution." Environmental Health Perspectives 111(9): 1188-93.

Zeka, A., A. Zanobetti, et al. (2005). "Short term effects of particulate matter on cause specific mortality: effects of lags and modification by city characteristics." Occupational and Environmental Medicine 62(10): 718-25.

International Studies

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BACKGROUND TECHNICAL INFORMATION PAGES

TABLE OF CONTENTS

Elicitation Aids

Emissions and Air Quality Data

Population Health Data

Policy/Regulatory Information

Population Demographics

BACKGROUND TECHNICAL INFORMATION PAGES

ELICITATION AIDS

BACKGROUND INFORMATION

EXCESS RELATIVE RISKS OF MORTALITY IN KEY COHORT STUDIES FOR A 10 μg/m^3 INCREASE IN $PM_{2.5}$

Study	PM[1]	Total Mortality		Cardiopulmonary Mortality		Lung Cancer Mortality	
		Ex RR[2]	95% CI	Ex RR	95% CI	Ex RR	95% CI
Six City[3]	$PM_{2.5}$	13%	(4.2, 23%)	18%	(6.0, 32%)	18%	(-11, 57%)
Six City New[4]	$PM_{2.5}$	14%	(5.4, 23%)	19%	(6.5, 33%)	21%	(-8.4, 60%)
ACS[5]	$PM_{2.5}$	6.6%	(3.5, 9.8%)	12%	(6.7, 17%)	1.2%	(-8.4, 60%)
ACS New[6]	$PM_{2.5}$	7.0%	(3.9, 9.8%)	12%	(7.4, 17%)	0.8%	(-8.7, 11%)
ACS Extend.[7]	$PM_{2.5}$ 1979-1983	4.1%	(0.8, 4.1%)	5.7%	(2.5, 9.0%)	-1.6%	(-9.1, 6.4%)
ACS Extend.	$PM_{2.5}$ 1999-2000	5.9%	(2.0, 9.9%)	7.9%	(2.3, 14%)	12.7%	(4.1, 22%)
ACS Extend.	$PM_{2.5}$ Avg.	6.2%	(1.6, 11%)	9.3%	(3.3, 16%)	13.5%	(4.4, 22%)
AHSMOG[8]	$PM_{2.5}$	8.5%	(-2.3%, 21%)	23%	(-3.0, 55%)	39%	(-21, 150%)
VA[9]	$PM_{2.5}$	0.3%	NS[10]				
	$PM_{2.5}$	-10%	SS[11]				

[1] Increments are 10 μg/m^3 for $PM_{2.5}$
[2] Ex. RR (excess relative risk, percent) = 100 * (RR-1) where the RR has been converted from the highest-to-lowest range to the standard increment by the equation RR=exp(log(RR for range) x (standard increment)/range).
[3] From Dockery et al. (1993); Krewski et al. (2000), Part II, Table 21a, original model.
[4] From Krewski et al. (2000), Part I, Table 21c.
[5] From Krewski et al. (2000), Part I, Table 25a.
[6] From Krewski et al. (2000), Part I, Table 25c.
[7] From Pope et al. (2002).
[8] From McDonnell et al. (2000), two-pollutant (fine and coarse) models; males only
[9] From Lipfert et al. (2000), Males only, exposure period 1979-1981 from Table 7. Standard errors not provided.
[10] Reported by author to be nonsignificant
[11] Reported to be statistically significant

*Source: USEPA, October 2004, Air Quality Criteria for Particulate Matter Volume II. EPA/600/P-99/002bF. (http://cfpub2.epa.gov/ncea/cfm/partmatt.cfm)

BACKGROUND INFORMATION

EXCESS RELATIVE RISKS OF MORTALITY IN COHORT STUDIES FOR A 1 μg/m^3 INCREASE IN $PM_{2.5}$

Study	PM[1]	Total Mortality		Cardiopulmonary Mortality		Lung Cancer Mortality	
		Ex RR[2]	95% CI	Ex RR	95% CI	Ex RR	95% CI
Six City[3]	$PM_{2.5}$	1.23%	(0.41, 2.09%)	1.67%	(0.58, 2.82%)	1.67%	(-1.16, 4.61%)
Six City New[4]	$PM_{2.5}$	1.32%	(0.53, 2.09%)	1.75%	(0.63, 2.89%)	1.92%	(-0.87, 4.81%)
ACS[5]	$PM_{2.5}$	0.64%	(0.34, 0.94%)	1.14%	(0.65, 1.58%)	0.12%	(-0.91, 1.14%)
ACS New[6]	$PM_{2.5}$	0.68%	(0.38, 0.96%)	1.14%	(0.72, 1.58%)	0.08%	(-0.91, 1.05%)
ACS Extend.[7]	$PM_{2.5}$ 1979-1983	0.40%	(0.08, 0.73%)	0.57%	(0.15, 0.96%)	0.79%	(0.11, 1.50%)
ACS Extend.	$PM_{2.5}$ 1999-2000	0.57%	(0.20, 0.95%)	0.76%	(0.23, 1.32%)	1.20%	(0.40, 2.01%)
ACS Extend.	$PM_{2.5}$ Avg.	0.60%	(0.16, 1.05%)	0.89%	(0.33, 1.50%)	1.27%	(0.43, 2.09%)
AHSMOG[8]	$PM_{2.5}$	0.82%	(-0.23, 0.44%)	2.09%	(-0.30, 4.48%)	3.35%	(-2.33, 9.60%)
VA[9]	$PM_{2.5}$	0.03%	NS[10]				
	$PM_{2.5}$	-1.05%	SS[11]				

[1] Increments are 1 μg/m^3 for $PM_{2.5}$
[2] Ex. RR (excess relative risk, percent) = 100 * (RR-1) where the RR has been converted from the highest-to-lowest range to the standard increment by the equation RR=exp(log(RR for range) x (standard increment)/range).
[3] From Dockery et al. (1993); Krewski et al. (2000), Part II, Table 21a, original model.
[4] From Krewski et al. (2000), Part I, Table 21c.
[5] From Krewski et al. (2000), Part I, Table 25a.
[6] From Krewski et al. (2000), Part I, Table 25c.
[7] From Pope et al. (2002).
[8] From McDonnell et al. (2000), two-pollutant (fine and coarse) models; males only
[9] From Lipfert et al. (2000), Males only, exposure period 1979-1981 from Table 7. Standard errors not provided.
[10] Reported by author to be nonsignificant
[11] Reported to be statistically significant

BACKGROUND INFORMATION

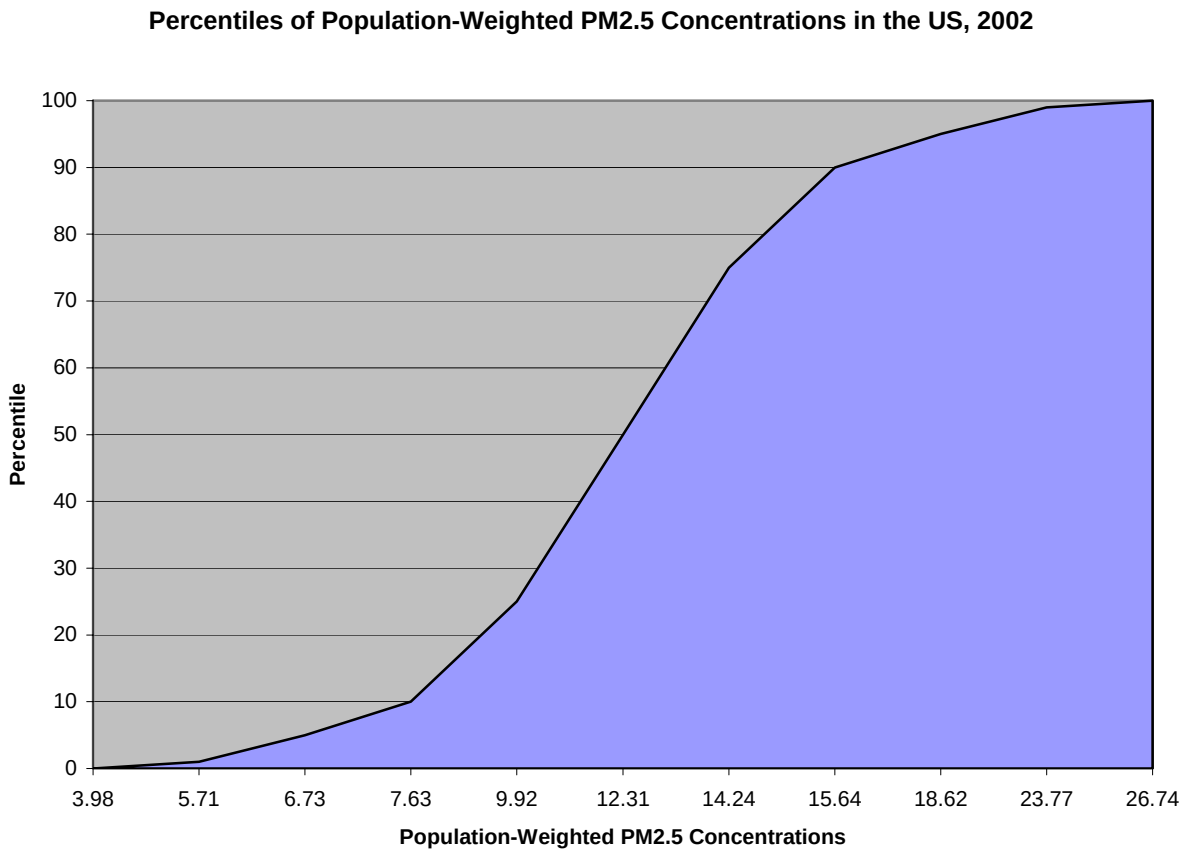
BETA COEFFICIENTS FROM LONG-TERM COHORT STUDIES DESCRIBING THE CONCENTRATION-RESPONSE RELATIONSHIP BETWEEN MORTALITY AND A 1 μg/m^3 INCREASE IN PARTICULATE MATTER

Study	PM[1]	Total Mortality		Cardiopulmonary Mortality		Lung Cancer Mortality	
		Beta	SE	Beta	SE	Beta	SE
Six City[2]	$PM_{2.5}$	0.0122	0.0044	0.0167	0.0058	0.0166	0.0150
Six City New[3]	$PM_{2.5}$	0.0131	0.0039	0.0174	0.0058	0.0191	0.0147
ACS[4]	$PM_{2.5}$	0.0064	0.0015	0.0113	0.0023	0.0012	0.0052
ACS New[5]	$PM_{2.5}$	0.0068	0.0014	0.0113	0.0023	0.0008	0.0049
ACS Extend.[6]	$PM_{2.5}$ 1979-1983	0.0040	0.0016	0.0057	0.0020	0.0079	0.0036
ACS Extend.	$PM_{2.5}$ 1999-2000	0.0057	0.0019	0.0076	0.0028	0.0120	0.0041
ACS Extend.	$PM_{2.5}$ Avg.	0.0060	0.0023	0.0089	0.0031	0.0127	0.0042
AHSMOG[7]	$PM_{2.5}$	0.0082	0.0056	0.0207	0.0122	0.0329	0.0319
VA[8]	$PM_{2.5}$	0.0003					
	$PM_{2.5}$	-0.0105					

[1] Increments are 10 μg/m^3 for $PM_{2.5}$
[2] From Dockery et al. (1993); Krewski et al. (2000), Part II, Table 21a, original model.
[3] From Krewski et al. (2000), Part I, Table 21c.
[4] From Krewski et al. (2000), Part I, Table 25a.
[5] From Krewski et al. (2000), Part I, Table 25c.
[6] From Pope et al. (2002).
[7] From McDonnell et al. (2000), two-pollutant (fine and coarse) models; males only
[8] From Lipfert et al. (2000), Males only, exposure period 1979-1981 from Table 7. Standard errors not provided

BACKGROUND INFORMATION

POPULATION-WEIGHTED $PM_{2.5}$ CONCENTRATIONS IN THE UNITED STATES

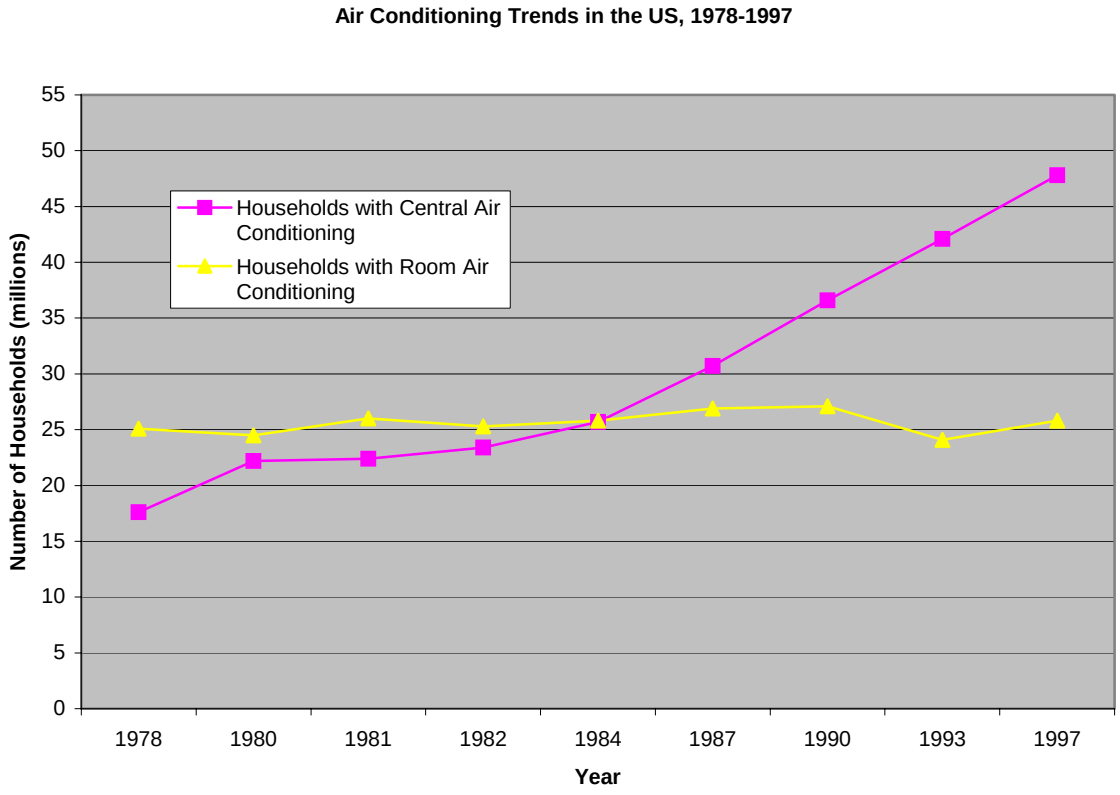


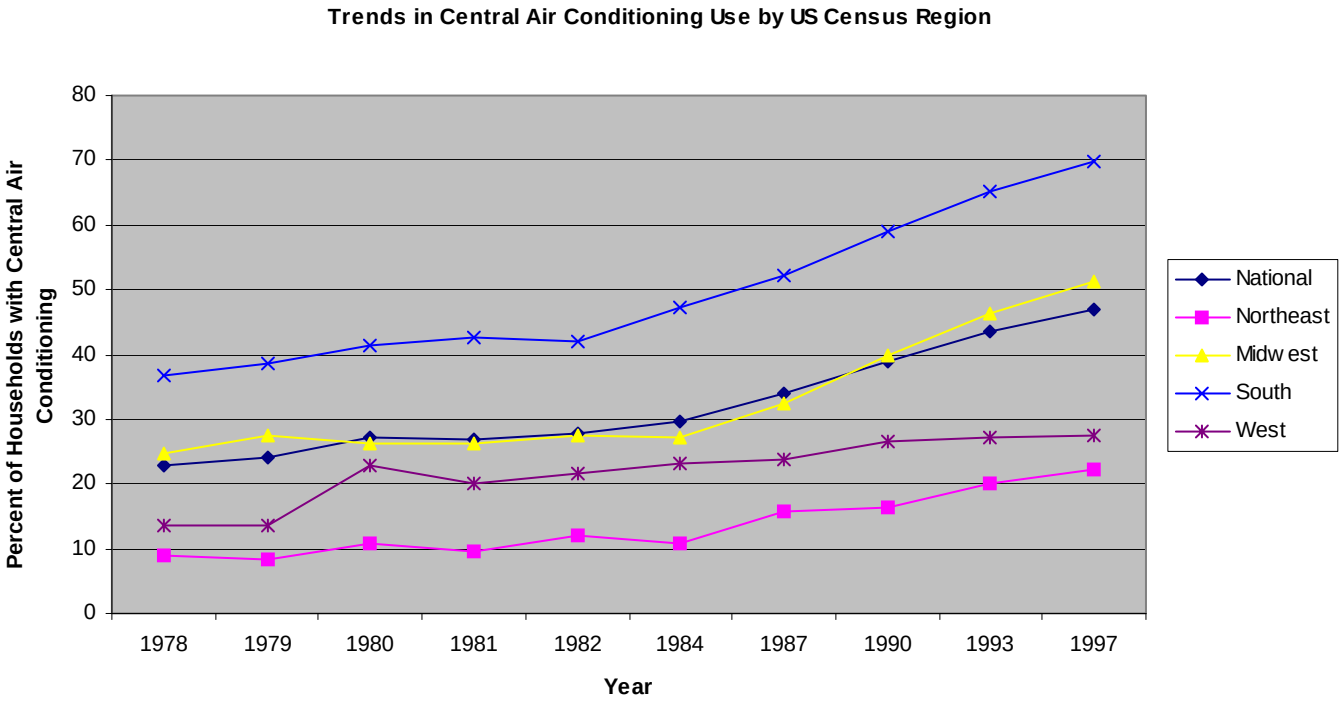
*Source: BenMAP (USEPA, 2003)

*Note: $PM_{2.5}$ monitor data from 2002, Population data from the 2000 US Census

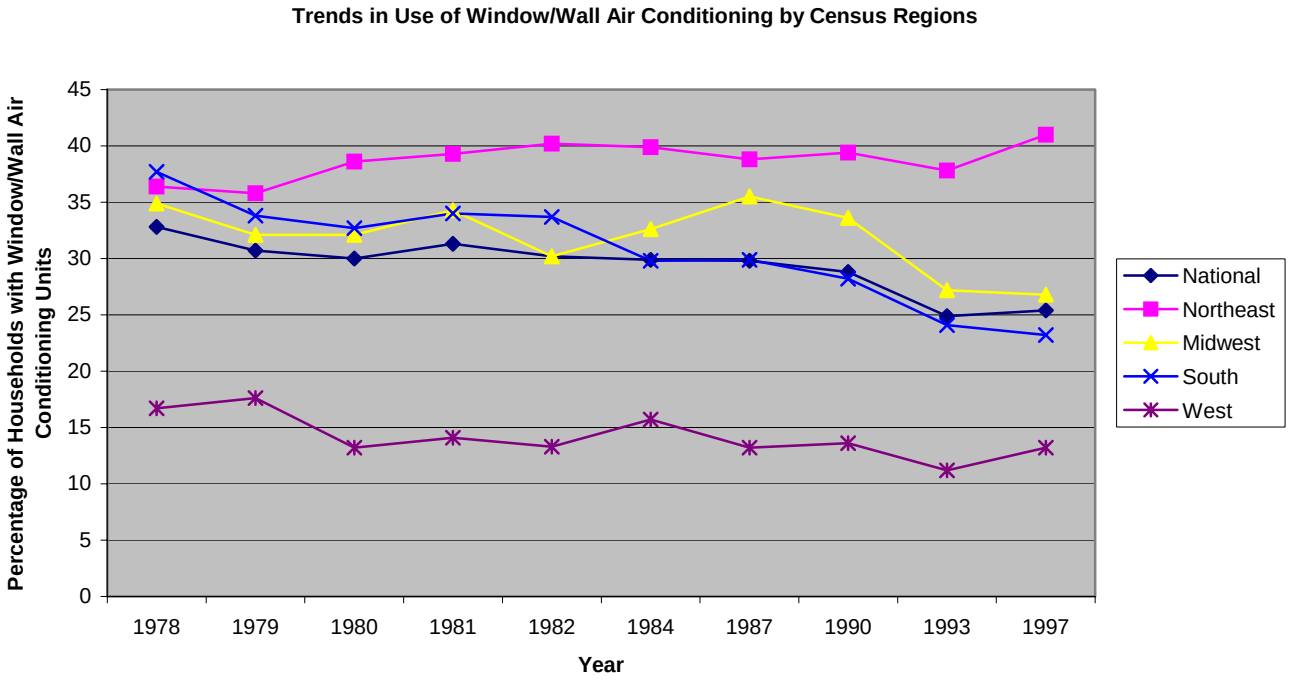
BACKGROUND INFORMATION

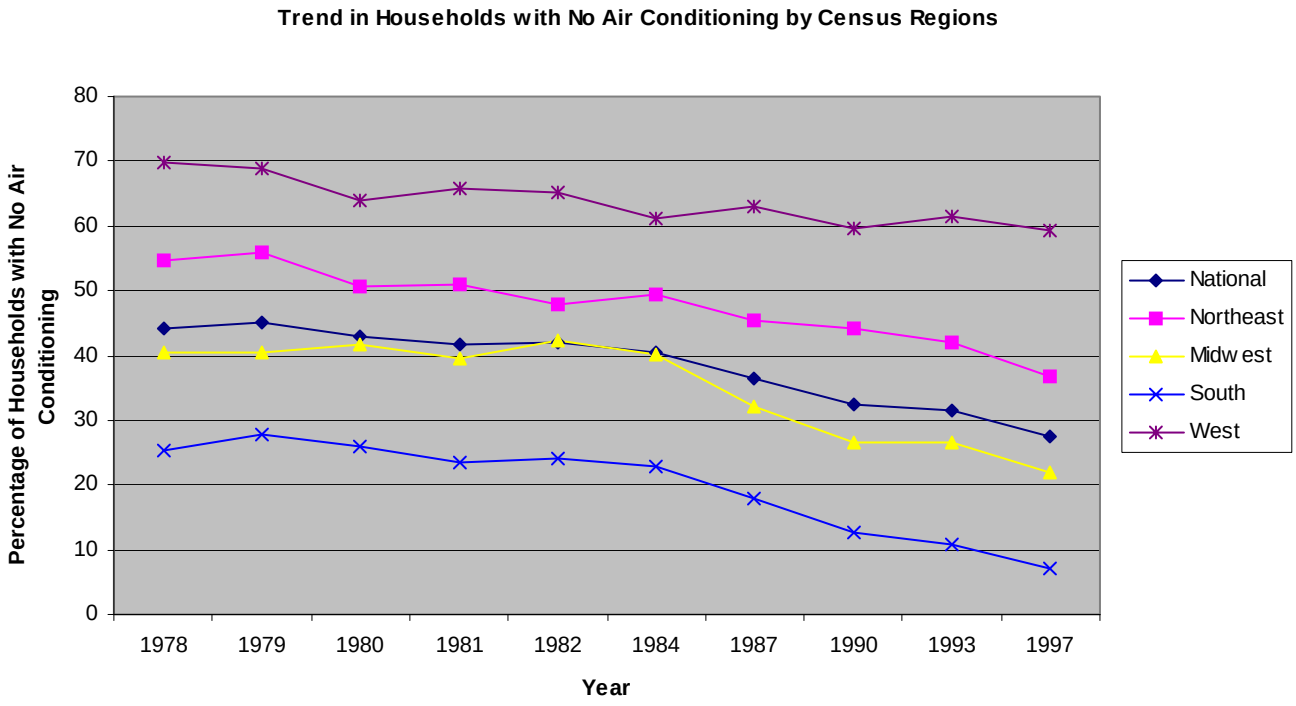
TRENDS IN AIR CONDITIONING USE IN THE UNITED STATES, 1978-1997





TRENDS IN AIR CONDITIONING USE IN THE UNITED STATES, 1978-1997 (CONTINUED)

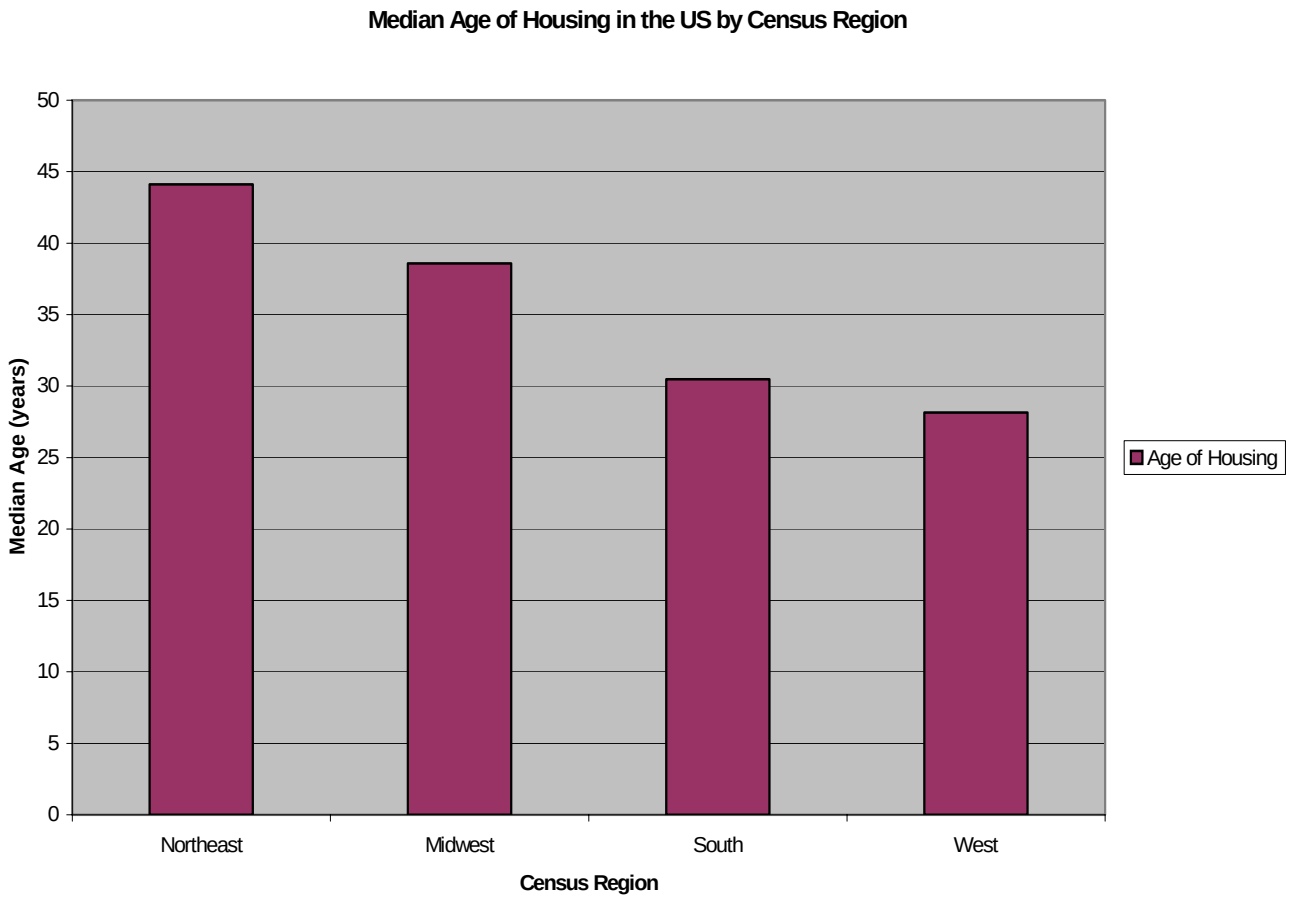




*Source: US Department of Energy (2000). *Trends in Residential Air-Conditioning Usage from 1978 to 1997*. (http://www.eia.doe.gov/emeu/consumptionbriefs/recs/actrends/recs_ac_trends.html)

BACKGROUND INFORMATION

MEDIAN AGE OF HOUSING IN THE U.S. BY CENSUS REGION, 2003



*Source: U.S. Census American Community Survey, 2003 (http://www.census.gov/acs/www/)

Census Regions:

Northeast = Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont.

Midwest = Indiana, Illinois, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin.

South = Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia.

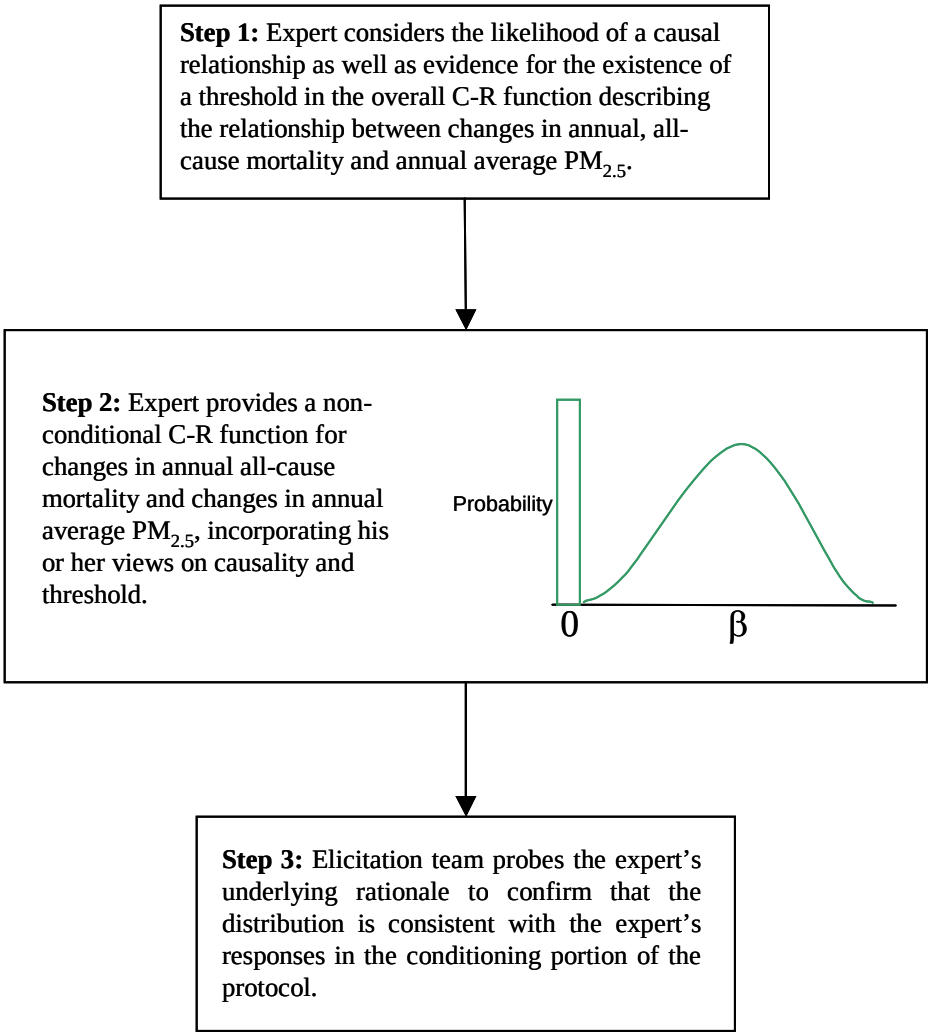
West = Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

BACKGROUND INFORMATION

TWO APPROACHES FOR INCORPORATING THRESHOLD AND CAUSALITY INTO AN OVERALL CONCENTRATION-RESPONSE (C-R) FUNCTION

Experts may choose how they would like factor their views on causality and threshold into their quantitative characterization of the C-R function between annual average exposures to $PM_{2.5}$ and changes in annual mortality. Below are two examples of ways that experts may choose to do this. In the first, called the "Aggregated Approach," the expert reflects their views on causality and/or threshold directly in the percentiles of the uncertainty distribution. In the second, called the "Disaggregated Approach" the expert provides a C-R distribution that is conditional on the existence of a causal relationship and/or a threshold. This distribution is then combined with the experts' probabilities for causality and threshold to create a final C-R function.

AGGREGATED APPROACH



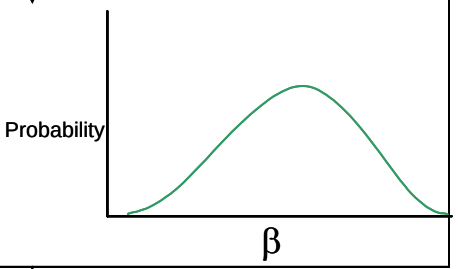
DISAGGREGATED

Step 1: Expert provides a most likely value for the probability of a causal relationship.

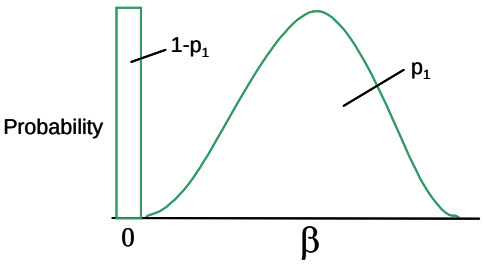
p_1 = probability that there is a causal relationship

1 - p_1 = probability that there is not a causal relationship

Step 2: Expert provides a C-R function for changes in annual mortality due to a 1 μg/m^3 reduction in $PM_{2.5}$, conditional on causality.



Step 3: Conditional C-R function from Step 1 is combined using Monte Carlo simulation with the probability of a causal relationship provided in Step 2.

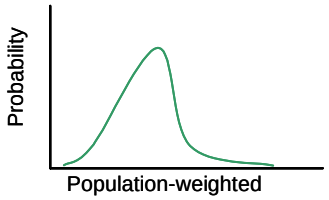


Step 4: Expert provides their view on the likelihood of a threshold.

Option 1: Expert believes that there is no threshold. The combined distribution from Step 3 remains the same and serves as the final C-R function.

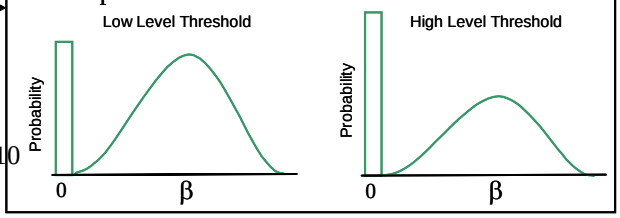
Option 2: Expert believes there is a threshold. Expert then specifies the likelihood of the level of the threshold, either with a discrete or continuous probability distribution.

Distribution of population-weighted $PM_{2.5}$ concentrations in the U.S., from BenMAP.



Step 5: Distribution from Step 3 is combined using Monte Carlo simulation with the probability or distribution provided in Step 4, Option 2 and the distribution of population-weighted $PM_{2.5}$ levels in the U.S.

Example:



BACKGROUND INFORMATION

PERCENT EXCESS MORTALITY IN KEY U.S./CANADIAN SHORT-TERM STUDIES ASSOCIATED WITH A 10 μg/m^3 INCREASE IN 24-HOUR AVERAGE PM

Study	Number of Cities	Lag	Model	Percent Excess Mortality[1]	95% CI
PM_{10} Studies					
HEI (2003)[2]	90 U.S. Cities	1 day	GLM with natural cubic splines	0.21%	(0.09, 0.33%)
Schwartz (2003a)	10 U.S. Cities	Mean of 0 and 1 day	GAM with stringent convergence criteria	0.66%	(0.52, 0.80%)
			Natural splines	0.55%	(0.39, 0.70%)
$PM_{2.5}$ Studies					
Schwartz (2003b)	6 U.S. Cities	Mean of 0 and 1 day	GAM with stringent convergence criteria	1.37%	(0.98, 1.76%)
			GLM with natural splines	1.29%	(0.88, 1.70%)
			B-Splines	1.17%	(0.78, 1.56%)
			P-Splines	1.13%	(0.70, 1.56%)
			Thin plate splines	1.04%	(0.61, 1.47%)
Burnett (2003)	8 Canadian Cities	1 day	GAM with stringent convergence criteria, temporal adjustment using LOESS smoothing with 90-day span	1.44%	(0.54, 2.34%)

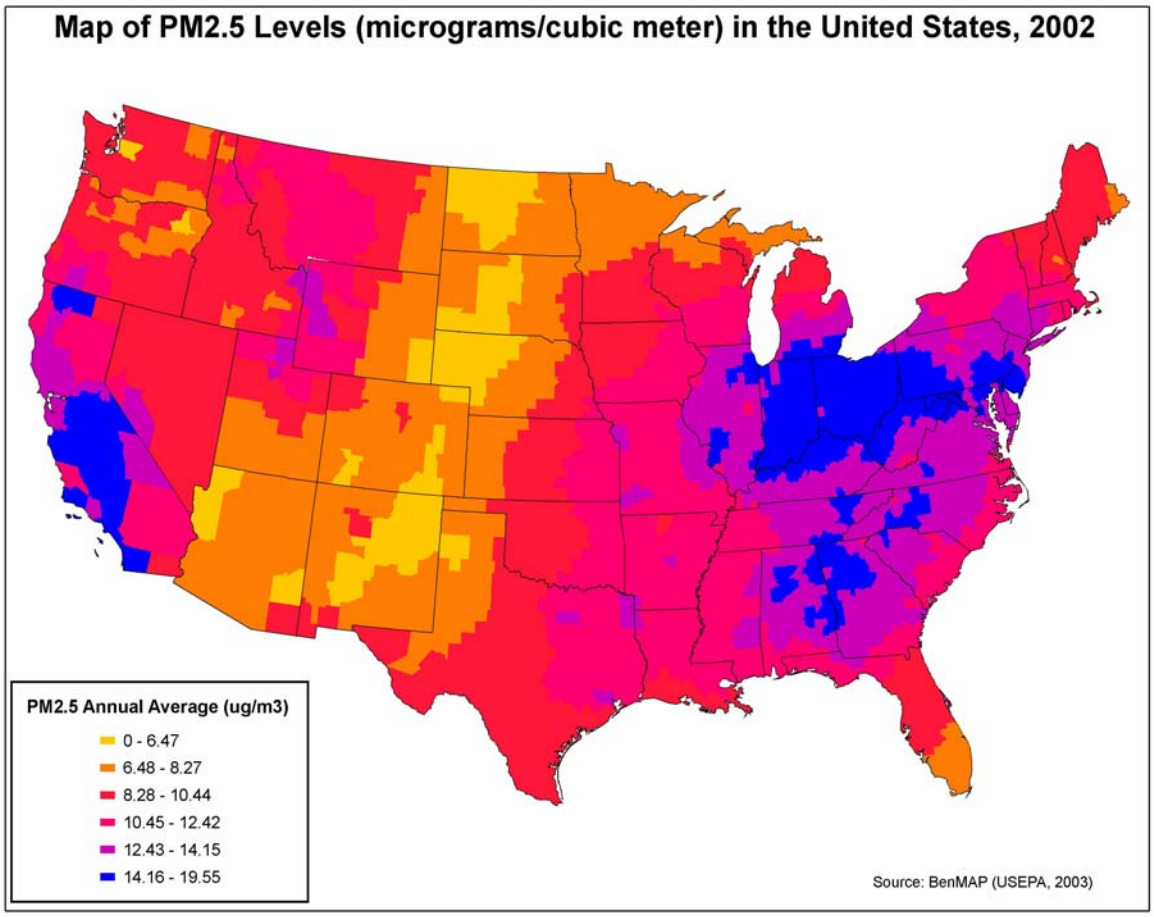
[1] Estimates are for a 10 μg/m3 increase in daily 24-hour average PM
[2] Revised analysis of the National Morbidity, Mortality, and Air Pollution Study
GLM = Generalized Linear Model
GAM = Generalized Additive Model

BACKGROUND TECHNICAL INFORMATION PAGES

EMISSIONS AND AIR QUALITY DATA

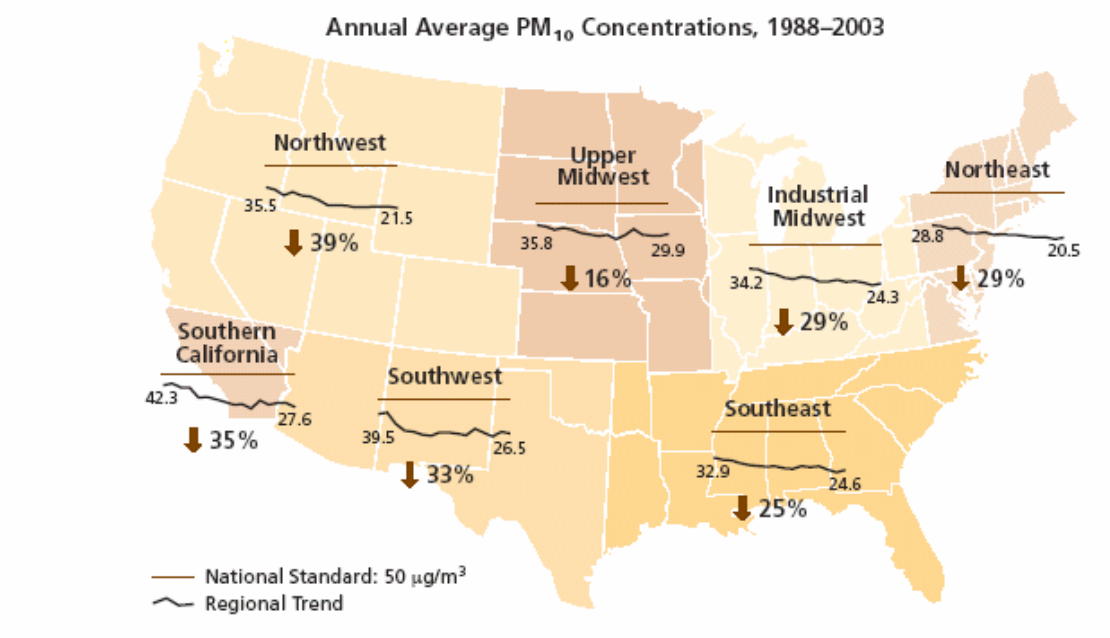
BACKGROUND INFORMATION

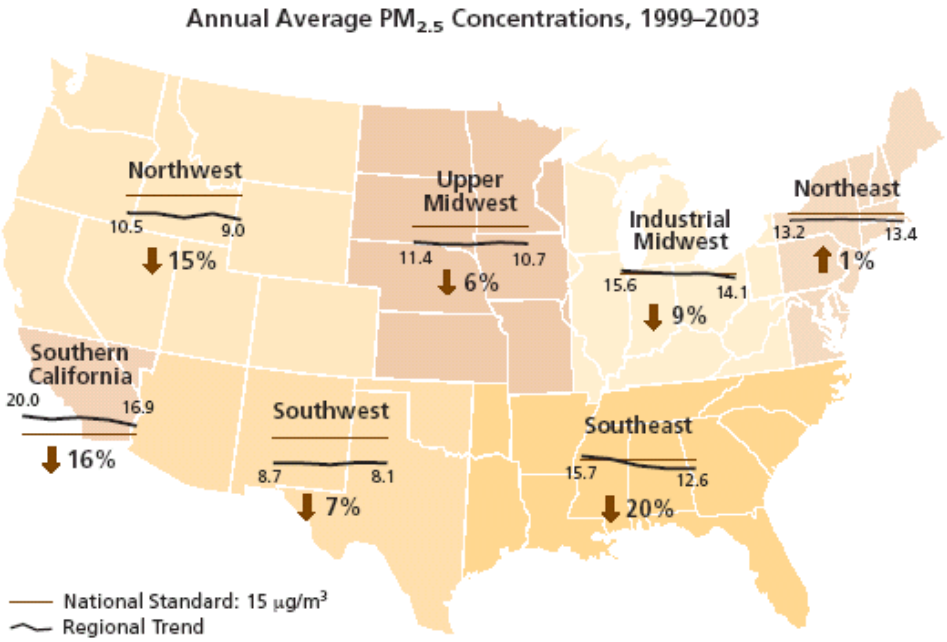
MAP OF ANNUAL AVERAGE $PM_{2.5}$ CONCENTRATIONS IN THE UNITED STATES



BACKGROUND INFORMATION

NATIONAL TRENDS OF PM_{10} AND $PM_{2.5}$ IN THE UNITED STATES





*Source: USEPA (2004) *The Particle Pollution Report: Current Understanding of Air Quality and Emissions through 2003*. EPA/454-R-04-002.

BACKGROUND INFORMATION

CRITERIA POLLUTANT CONCENTRATIONS IN THE UNITED STATES 2000-2004[1]

$PM_{2.5}$ CONCENTRATIONS ($\mu g/m^3$) (STANDARD: 15.0 $\mu g/m^3$ ANNUAL MEAN)			
Year	**Annual Mean[1]**	**Annual Maximum[2]**	**Annual Minimum[3]**
2000	12.92	28.3	1.8
2001	12.41	31.0	1.6
2002	11.94	27.4	2.2
2003	11.70	24.8	2.0
2004	11.53	43.0	3.3

[1] Mean of individual annual means from each monitoring station
[2] Maximum annual mean of monitoring stations
[3] Minimum annual mean of monitoring stations

PM_{10} CONCENTRATIONS ($\mu g/m^3$) (STANDARD: 50 $\mu g/m^3$ ANNUAL MEAN)			
Year	**Annual Mean**	**Annual Maximum**	**Annual Minimum**
2000	24.97	153.0	4.0
2001	24.87	289.0	3.0
2002	24.47	152.0	4.0
2003	24.72	140.0	4.0
2004	23.18	117.0	3.0

$S0_2$ CONCENTRATIONS (ppm) (STANDARD: 0.030 ppm ANNUAL MEAN)			
Year	**Annual Mean**	**Annual Maximum**	**Annual Minimum**
2000	0.005	0.017	0.001
2001	0.005	0.023	0.001
2002	0.004	0.017	0.001
2003	0.004	0.021	0.001
2004	0.004	0.015	0.001

$N0_2$ CONCENTRATIONS (ppm) (STANDARD: 0.053 ppm ANNUAL MEAN)			
Year	**Annual Mean**	**Annual Maximum**	**Annual Minimum**
2000	0.015	0.044	0.001
2001	0.015	0.041	0.003
2002	0.014	0.040	0.002
2003	0.013	0.038	0.001
2004	0.012	0.035	0.001

CO CONCENTRATIONS (ppm) (STANDARDS: 35 ppm (1-HOUR AVERAGE); 9 ppm (8-HOUR AVERAGE))				
Year	**1st Max (1-hr)[1]**	**2nd Max (1-hr)[2]**	**1st Max (8-hr)[3]**	**2nd Max (8-hr)[4]**
2000	6.15	5.48	3.77	3.33
2001	5.85	5.15	3.52	3.12
2002	5.20	4.63	3.17	2.82
2003	4.80	4.27	2.94	2.60
2004	4.43	3.90	2.75	2.40

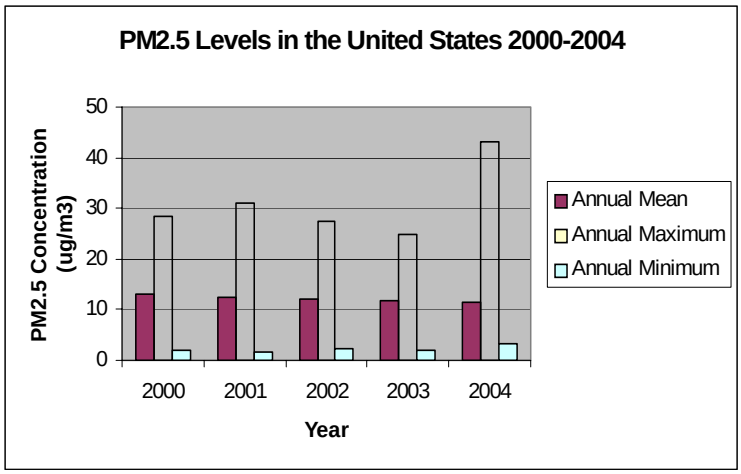
[1] Mean of highest 1-hour maximum from monitoring stations
[2] Mean of second highest 1-hour maximum from monitoring stations
[3] Mean of highest 8-hour maximum from monitoring stations
[4] Mean of second highest 8-hour maximum from monitoring stations

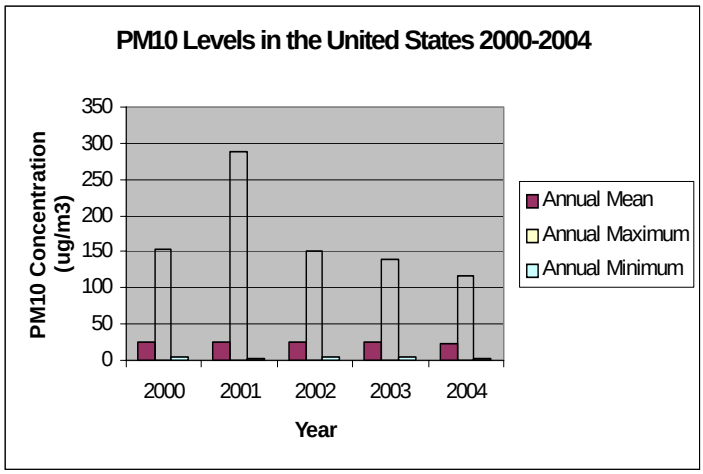
O_3 CONCENTRATIONS (ppm) (STANDARD: 0.12 ppm (1-HOUR AVERAGE); 0.08 ppm (8-HOUR AVERAGE))				
Year	**1st Max (1-hr)**	**2nd Max (1-hr)**	**1st Max (8-hr)**	**2nd Max (8-hr)**
2000	0.107	0.099	0.091	0.085
2001	0.106	0.099	0.090	0.085
2002	0.110	0.104	0.094	0.089
2003	0.107	0.099	0.092	0.086
2004	0.095	0.089	0.081	0.077

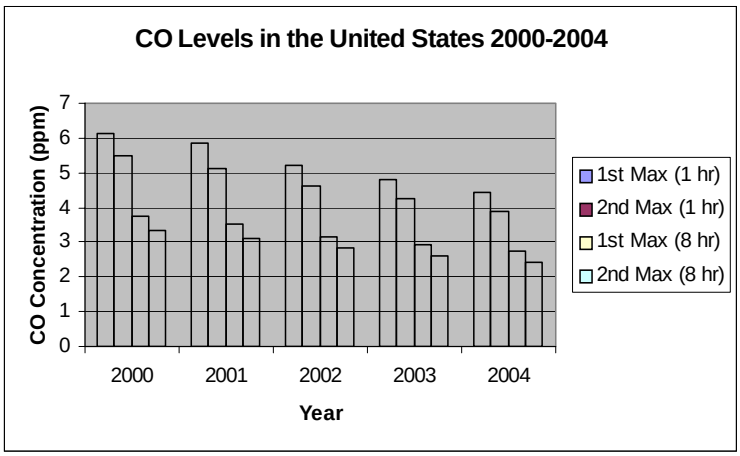
[1] Source: EPA's AirData monitoring data: www.epa.gov/air/data

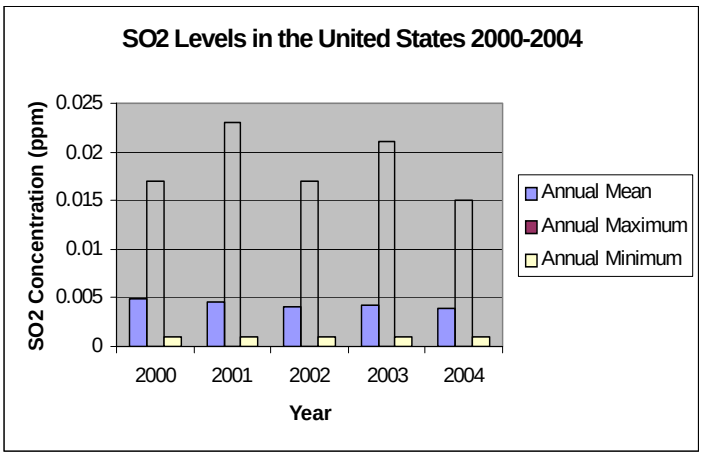
BACKGROUND INFORMATION

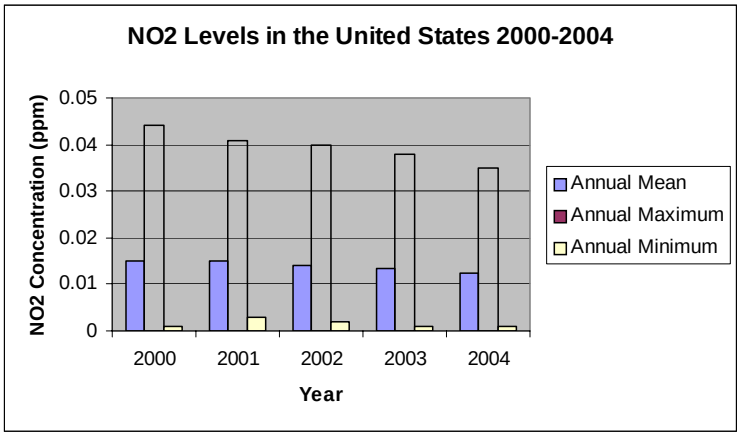
CRITERIA POLLUTANT CONCENTRATIONS IN THE UNITED STATES 2000-2004[2]

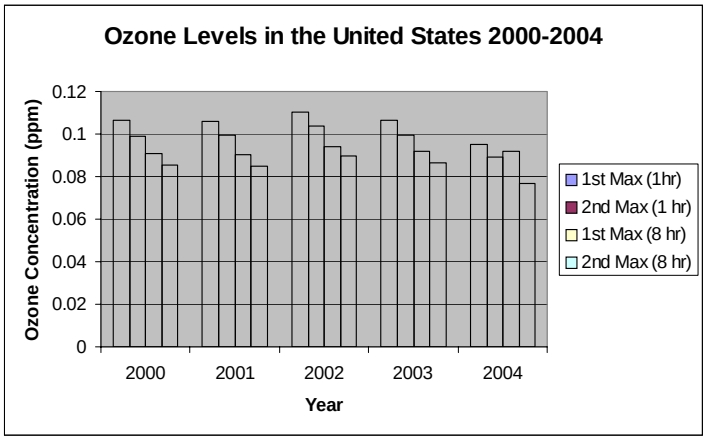








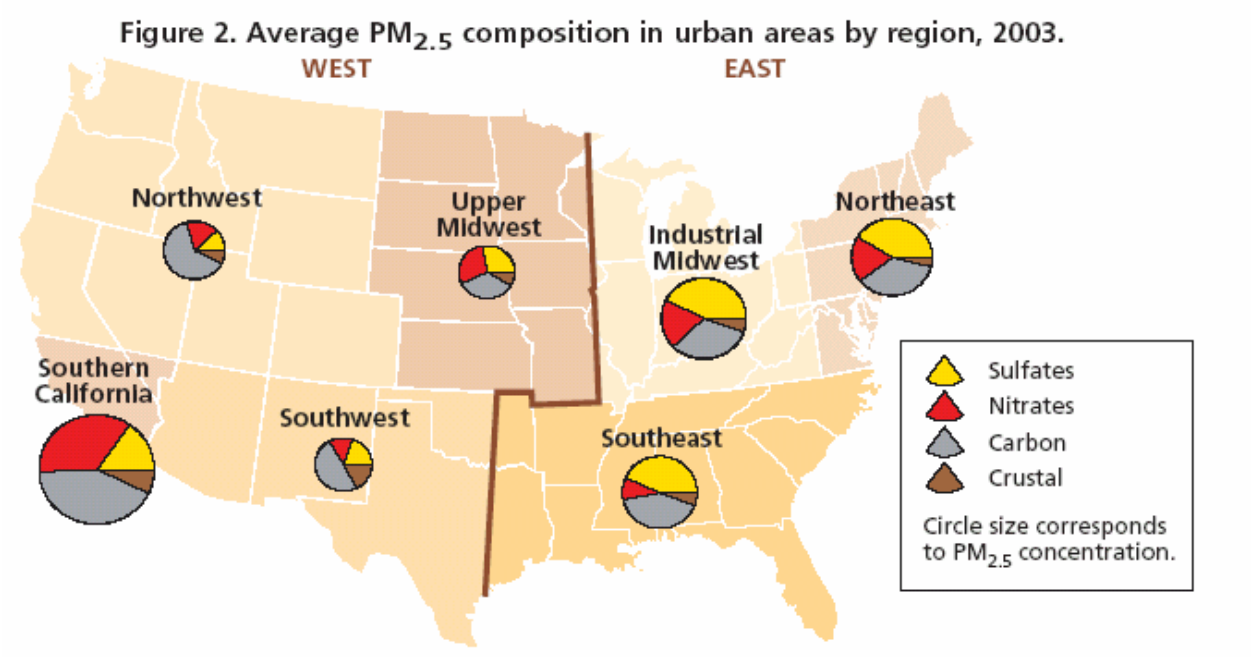




[2] Source: EPA's AirData monitoring data: www.epa.gov/air/data

BACKGROUND INFORMATION

CURRENT COMPOSITION OF $PM_{2.5}$ IN URBAN AREAS BY REGION



*Source: USEPA, December 2004, The Particle Pollution Report: Current Understanding of Air Quality and Emissions through 2003. EPA/454-R-04-002.

Note: "Sulfates" refers to ammonium sulfate and "nitrates" refers to ammonium nitrate. "Carbon" refers to total carbonaceous mass, which is the sum of estimated organic carbon mass and elemental carbon. "Crustal" is estimated using the IMPROVE equation for fine soil at vist.cira.colostate.edu/improve.

BACKGROUND INFORMATION

$PM_{2.5}$ COMPOSITION BY REGION, 2003

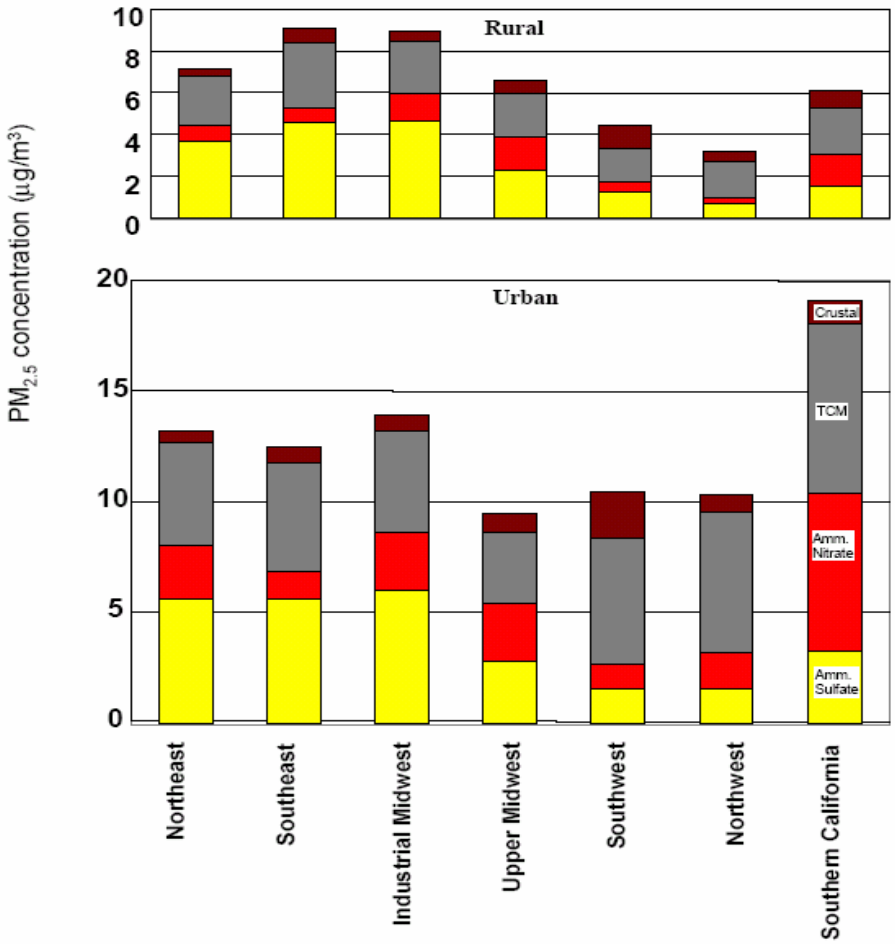
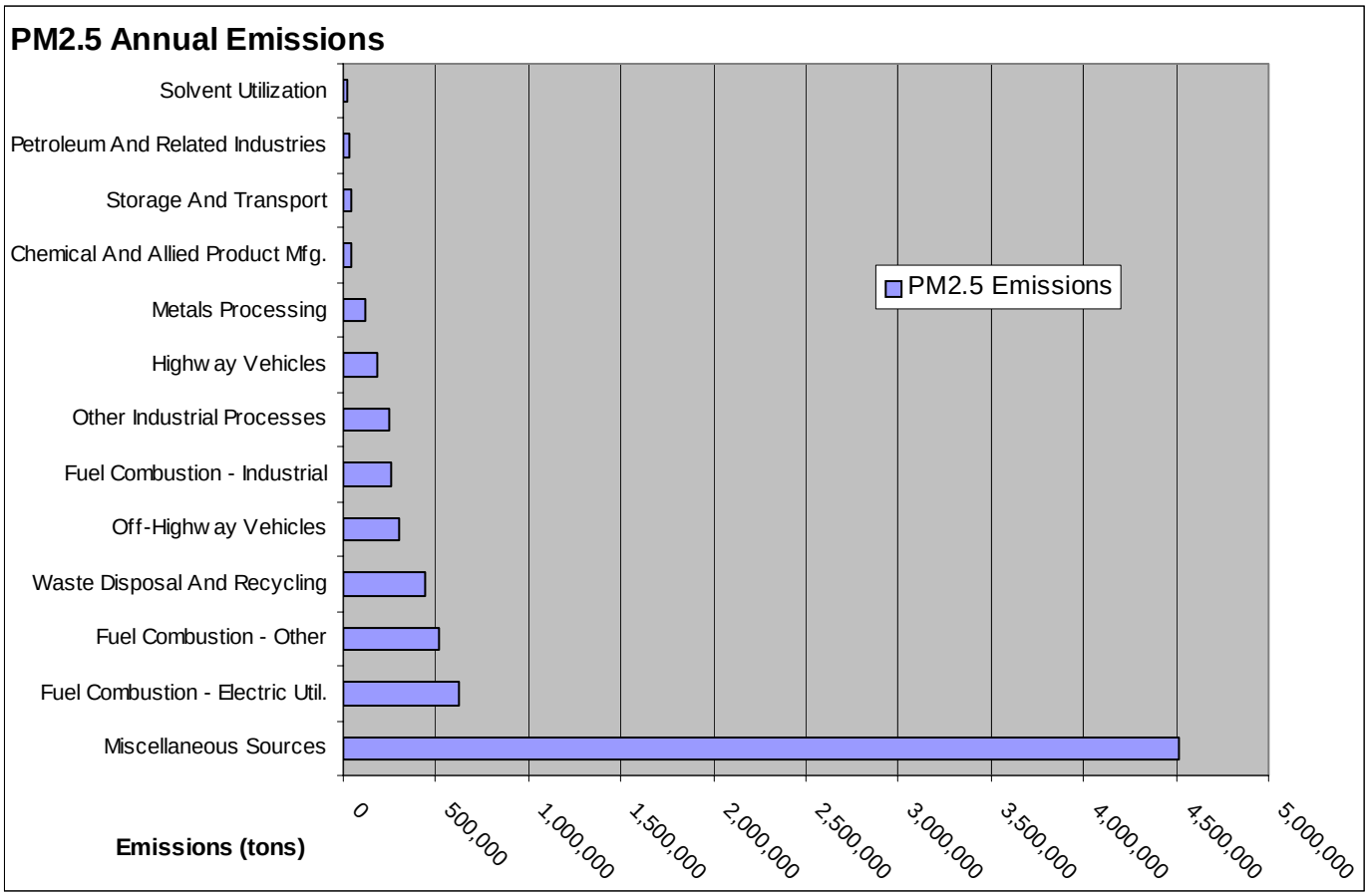


Figure 2-15. Annual average composition of $PM_{2.5}$ by region, 2003. Rural data (top panel) from IMPROVE network, urban data (bottom panel) from EPA Speciation Network. Components (from top to bottom) are crustal material, total carbonaceous mass (TCM), ammonium nitrate, and ammonium sulfate.

*Source: USEPA, January 2005, Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information - OAQPS Staff Paper - Second Draft. EPA-452/D-05-001.

BACKGROUND INFORMATION

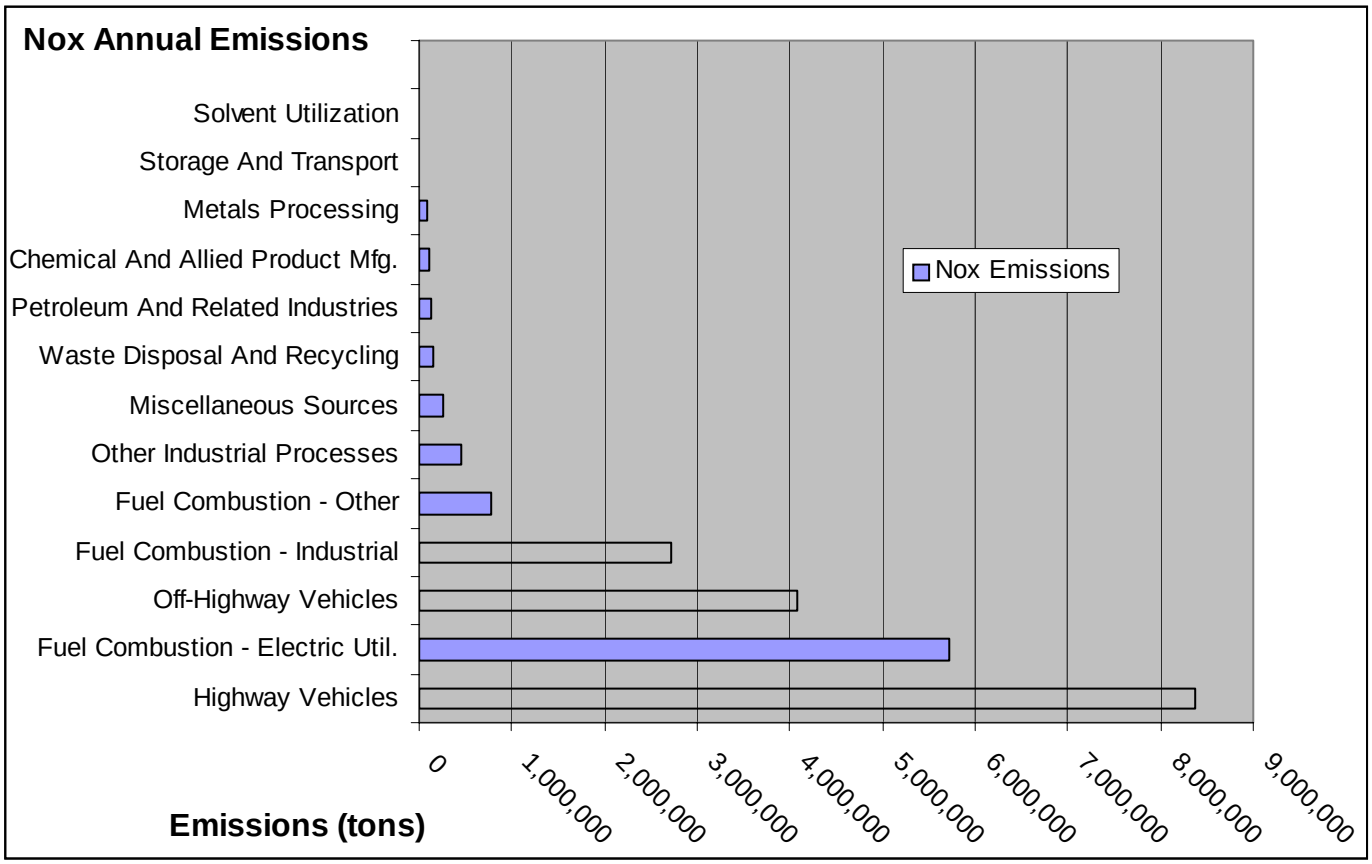
ANNUAL EMISSIONS OF $PM_{2.5}$ IN THE UNITED STATES BY CATEGORY, 1999



* Source: Source: EPA's AirData monitoring data: www.epa.gov/air/data

BACKGROUND INFORMATION

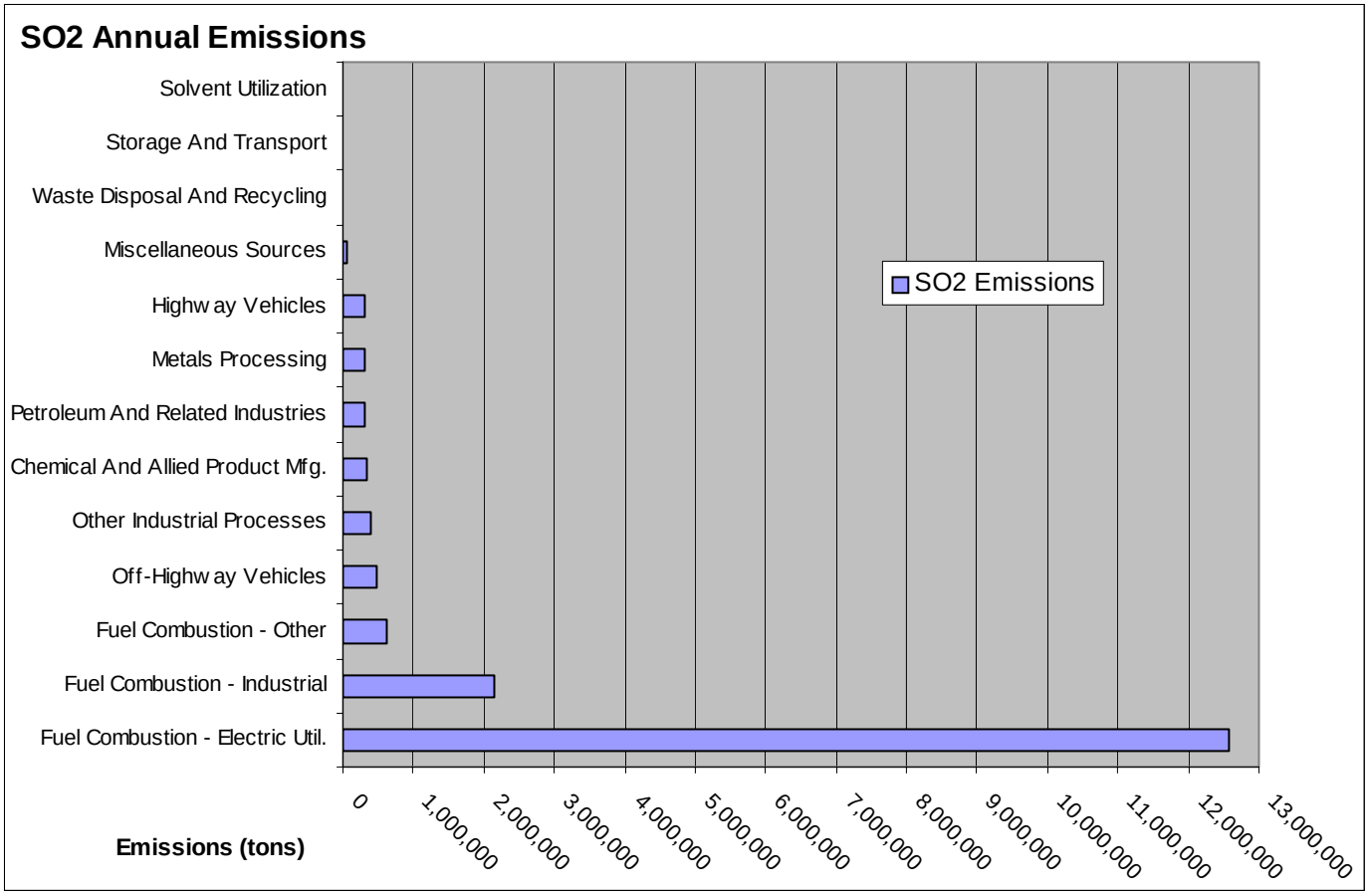
ANNUAL EMISSIONS OF NO_x IN THE UNITED STATES BY CATEGORY, 1999



* Source: Source: EPA's AirData monitoring data: www.epa.gov/air/data

BACKGROUND INFORMATION

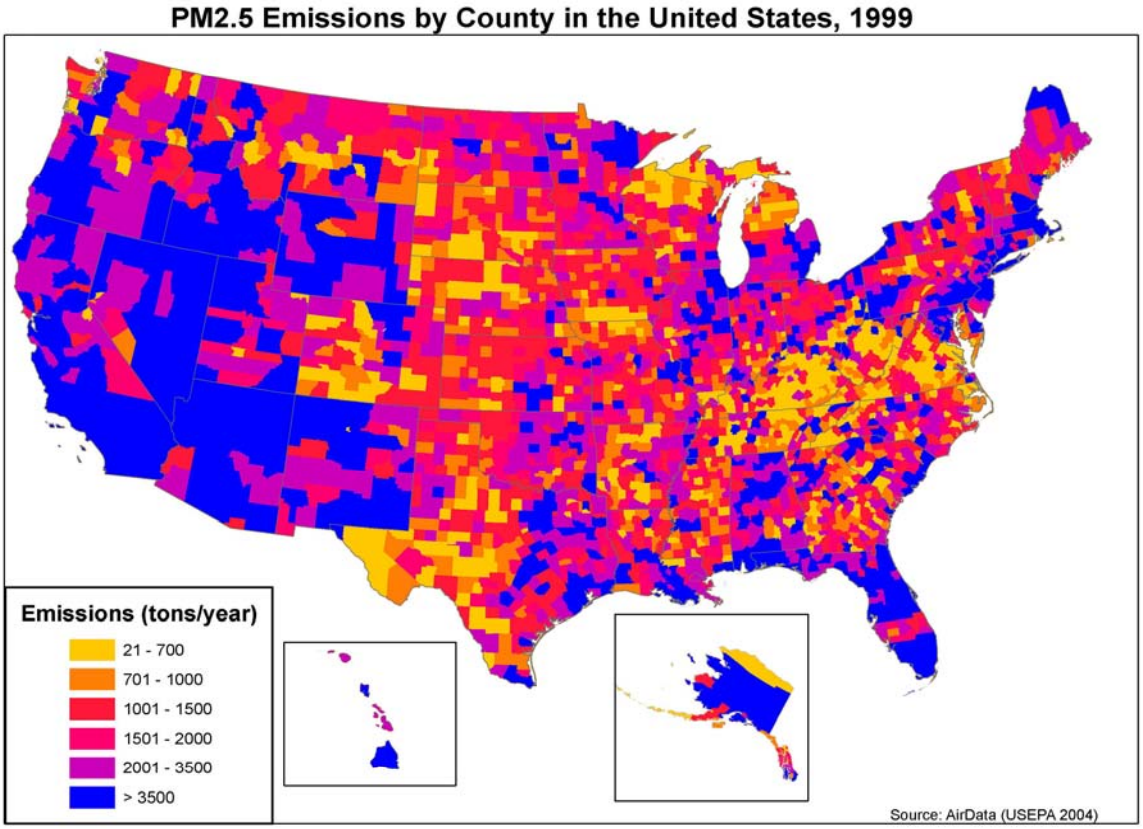
ANNUAL EMISSIONS OF SO_2 IN THE UNITED STATES BY CATEGORY, 1999



* Source: Source: EPA's AirData monitoring data: www.epa.gov/air/data

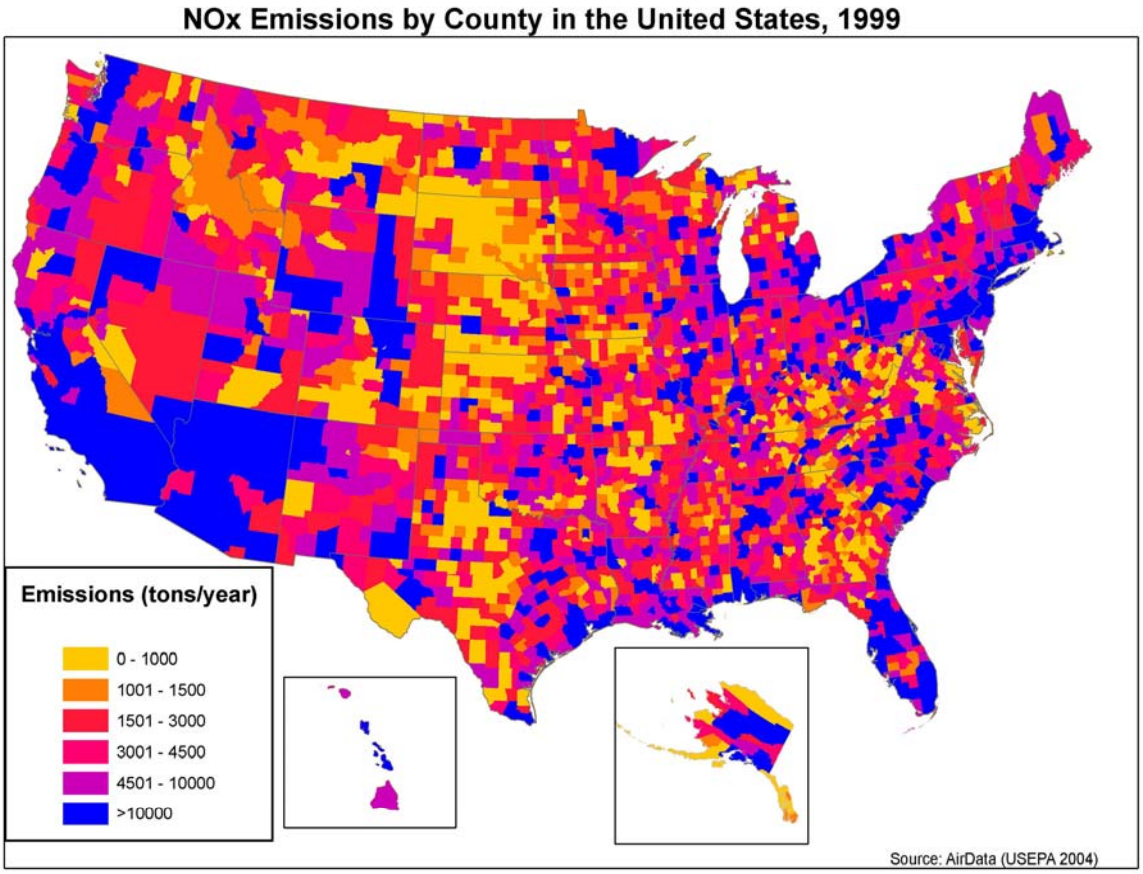
BACKGROUND INFORMATION

MAP OF $PM_{2.5}$ EMISSIONS IN THE UNITED STATES



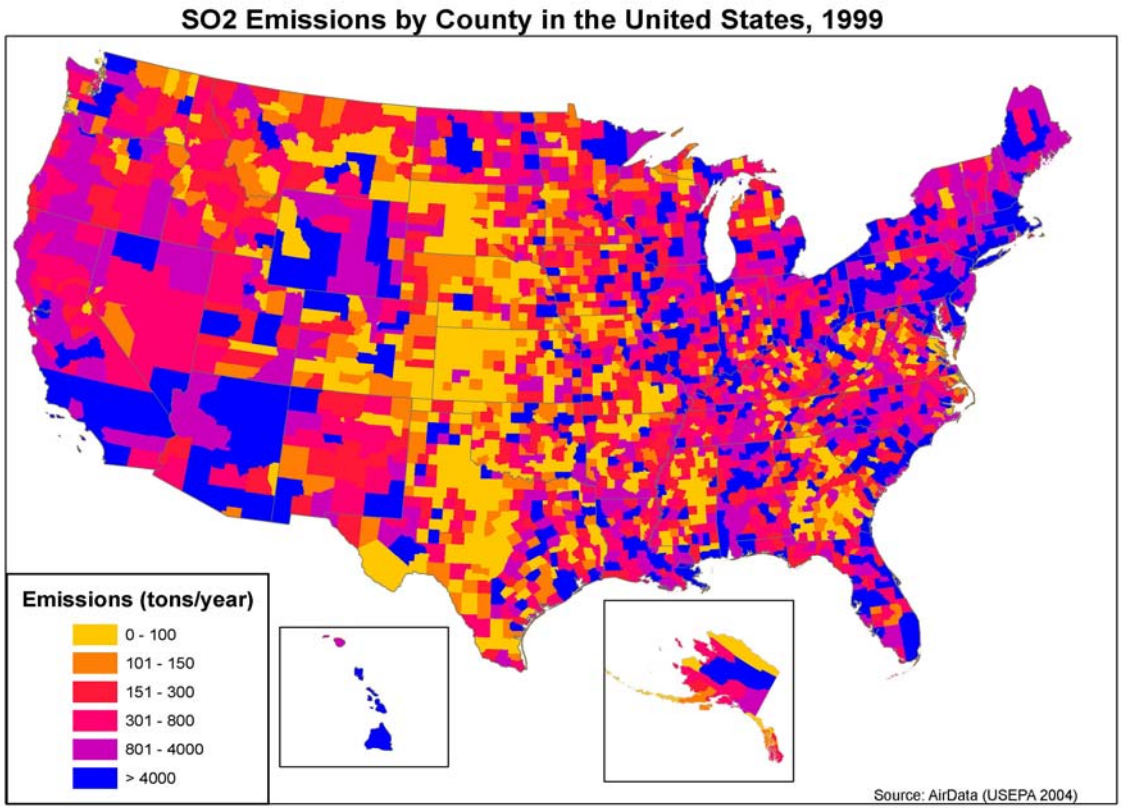
BACKGROUND INFORMATION

MAP OF NO_x EMISSIONS IN THE UNITED STATES



BACKGROUND INFORMATION

MAP OF SO_2 EMISSIONS IN THE UNITED STATES



BACKGROUND INFORMATION

24-HOUR AVERAGE $PM_{2.5}$ CONCENTRATIONS IN THE U.S., 2004

	1st Max	2nd Max	3rd Max	4th Max	98th %ile
2000					
Min	8	7	6	5	7
Max	200	116	110	107	121
Mean	38	34	31	29	32
Median	41	34	32	30	32
Mode	38	32	34	32	34
2001					
Min	8	8	7	7	8
Max	641	96	94	91	96
Mean	40	34	31	29	32
Median	41	34	31	29	32
Mode	41	39	28	29	33
2002					
Min	5	4	4	4	4
Max	138	111	77	76	77
Mean	38	33	30	28	31
Median	41	34	31	29	31
Mode	34	33	34	28	31
2003					
Min	5	4	4	4	5
Max	239	105	94	79	77
Mean	37	32	29	27	30
Median	40	32	29	27	30
Mode	31	39	31	31	32
2004					
Min	6	6	6	6	6
Max	997	128	120	117	101
Mean	37	32	29	27	30
Median	36	31	29	27	29
Mode	38	30	28	30	28

Key: 1st Max, 2nd Max, 3rd Max, 4th Max = The four highest 24-hour values of the year in micrograms per cubic meter for each monitor; 98th %ile = The 98th %ile 24-hour value in micrograms per cubic meter for each monitor.
Note: EPA Standard for 24-hour average $PM_{2.5}$ is 65 $\mu g/m^3$.

*Source: EPA AirData (http://www.epa.gov/air/data/info.html)

BACKGROUND TECHNICAL INFORMATION PAGES

POPULATION HEALTH DATA

BACKGROUND INFORMATION

TOTAL ANNUAL DEATHS IN THE UNITED STATES (2002) AND LEADING CAUSES OF DEATH

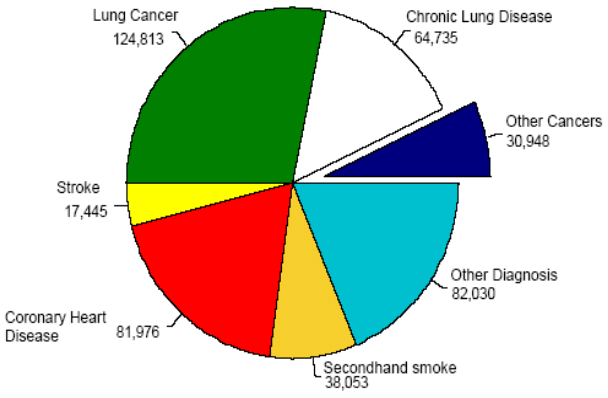
Table C. Deaths and percentage of total deaths for the 10 leading causes of death: United States, 2001–02

Cause of death and year	Rank[1]	2002		2001	
		Deaths	Percentage of total deaths	Deaths	Percentage of total deaths
All causes	...	2,443,387	100.0	2,416,425	100.0
Diseases of heart (I00–I09,I11,I13,I20–I51)	1	696,947	28.5	700,142	29.0
Malignant neoplasms (C00–C97)	2	557,271	22.8	553,768	22.9
Cerebrovascular diseases (I60–I69)	3	162,672	6.7	163,538	6.8
Chronic lower respiratory diseases (J40–J47)	4	124,816	5.1	123,013	5.1
Accidents (unintentional injuries) (V01–X59,Y85–Y86)	5	106,742	4.4	101,537	4.2
Diabetes mellitus (E10–E14)	6	73,249	3.0	71,372	3.0
Influenza and pneumonia (J10–J18)	7	65,681	2.7	62,034	2.6
Alzheimer's disease (G30)	8	58,866	2.4	53,852	2.2
Nephritis, nephrotic syndrome and nephrosis (N00–N07,N17–N19,N25–N27)	9	40,974	1.7	39,480	1.6
Septicemia (A40–A41)	10	33,865	1.4	32,238	1.3

... Category not applicable.
[1]Rank based on number of deaths.

* Source: National Vital Statistics Reports, Vol. 53, No. 17, March 7, 2005. (http://www.cdc.gov/nchs/data/nvsr/nvsr53/nvsr53_17.pdf)

Figure 1: 440,000 U.S. DEATHS ATTRIBUTABLE EACH YEAR TO CIGARETTE SMOKING *



* AVERAGE ANNUAL NUMBER OF DEATHS, 1995-1999
SOURCE: CDC, MMWR April 12, 2002; 51 (14); 300-303

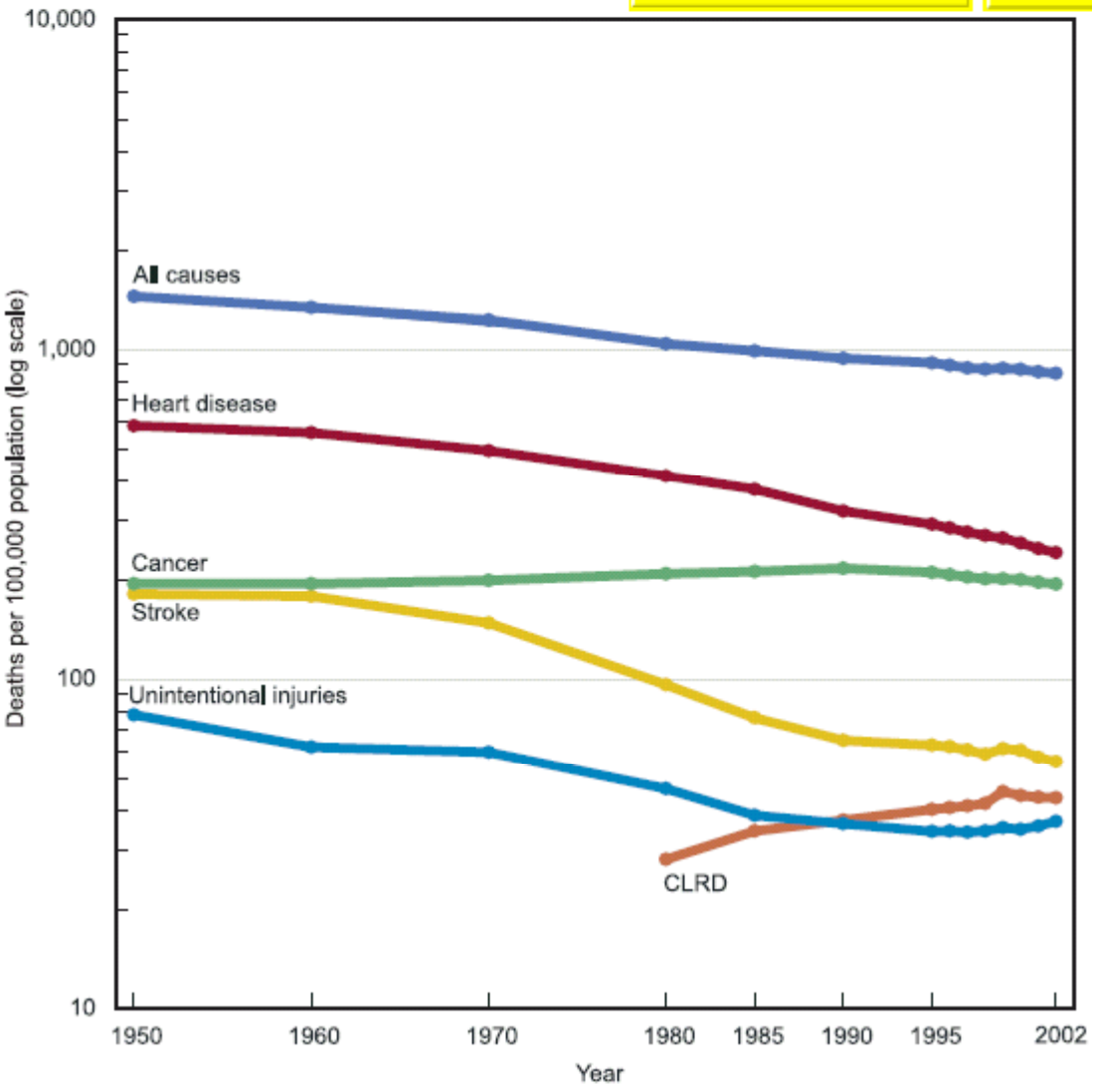
* Source: Trends in Tobacco Use 2004, American Lung Association. (http://www.lungusa.org/atf/cf/{7A8D42C2-FCCA-4604-8ADE-7F5D5E762256}/SMK2.PDF)

BACKGROUND INFORMATION

DEATH RATES FOR LEADING CAUSES OF DEATH IN THE UNITED STATES

Figure 25. Death rates for leading causes of death for all ages: United States, 1950-2002

Click here for spreadsheet version | Click here



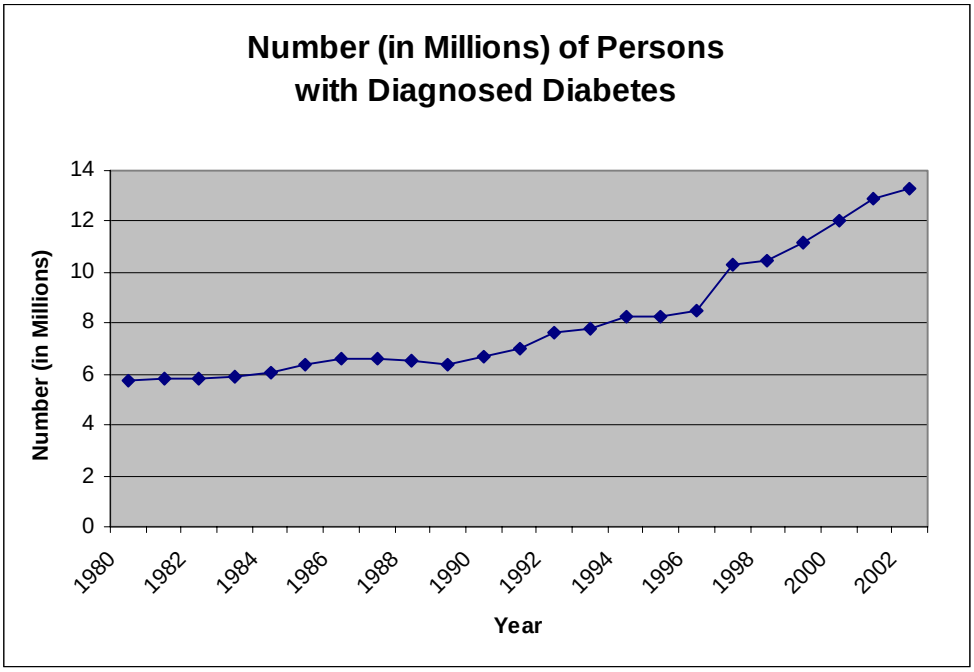
NOTES: Rates are age adjusted. Causes of death shown are the five leading causes of death for all ages in 2002. CLRD is chronic lower respiratory diseases. Starting in 1999 data were coded according to ICD-10. See Data Table for data points graphed and additional notes.

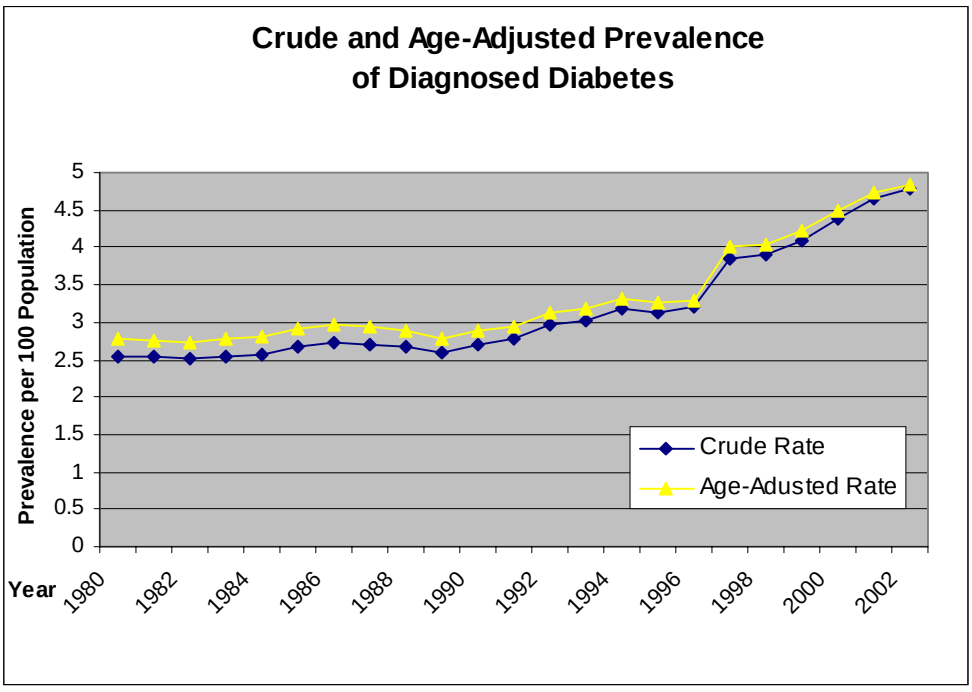
SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System.

*Source: National Center for Health Statistics. (2004). *Health, United States, 2004 - With chartbook on trends in the health of Americans.* http://www.cdc.gov/nchs/data/hus/hus04.pdf.

BACKGROUND INFORMATION

DIABETES PREVALENCE IN THE UNITED STATES, 1980-2002



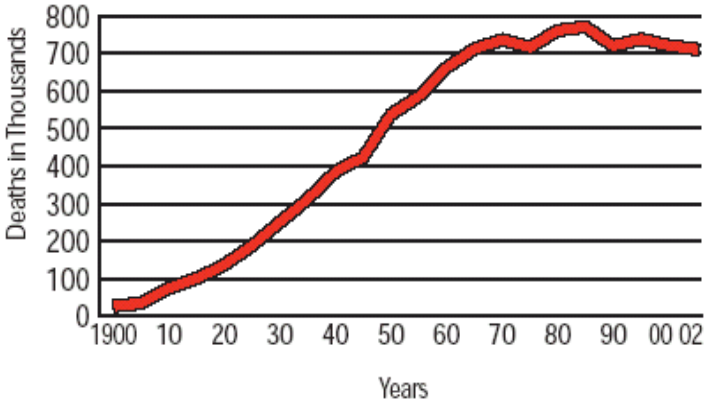


*Source: Center for Disease Control Diabetes Program - Data & Trends.
http://www.cdc.gov/diabetes/statistics/index.htm.

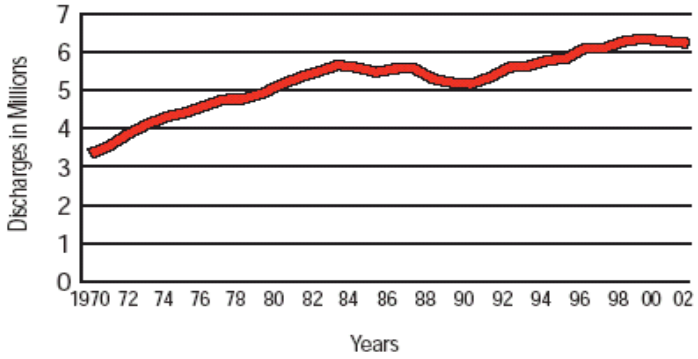
BACKGROUND INFORMATION

HEART DISEASE IN THE UNITED STATES

Deaths From Diseases of the Heart
United States: 1900–2002

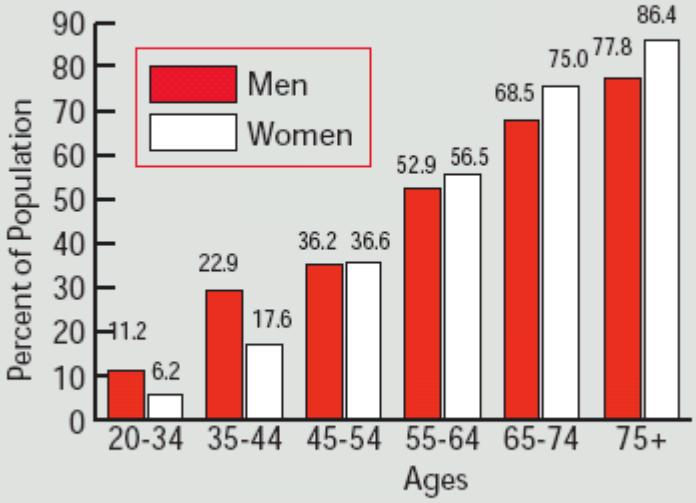


Hospital Discharges for Cardiovascular Diseases
United States: 1970–2002



Note: Hospital discharges include people both living and dead.

Prevalence of Cardiovascular Diseases in Americans Age 20 and Older by Age and Sex
NHANES: 1999–2002



Source: CDC/NCHS and NHLBI. These data include CHD, CHF, stroke and hypertension.

*Source: American Heart Association. *Heart Disease and Stroke Statistics - 2005 Update*. http://www.americanheart.org/downloadable/heart/1105390918119HDSStats2005Update.pdf

BACKGROUND TECHNICAL INFORMATION PAGES

POLICY/REGULATORY INFORMATION

BACKGROUND INFORMATION

SAMPLE LIST OF INDUSTRIES LIKELY TO BE AFFECTED BY PARTICULATE MATTER (PM) REGULATIONS

Fossil Fuel-Fired Steam Electric Plants (>250 MMBTU heat input per hour)
Fossil Fuel-Fired Industrial Boilers (>250 MMBTU heat input per hour)
Petroleum Refineries
Kraft Pulp Mills
Portland Cement Plants
Iron and Steel Mill Plants
Hydrofluoric, Sulfuric, and Nitric Acid Plants
Coke Oven Batteries
Sulfur Recovery Plants
Primary Lead Smelters
Primary Copper Smelters
Primary Zinc Smelters
Primary Aluminum Ore Reduction Plants
Municipal Incinerators (> 250 tons refuse per day)
Lime Plants
Phosphate Rock Processing Plants
Carbon Black Plants (furnace process)
Fuel Conversion Plants
Sintering Plants
Secondary Metal Production Facilities
Chemical Process Plants
Petroleum Storage and Transfer Facilities (capacity > 300,000 barrels)
Taconite Ore Processing Plants
Glass Fiber Processing Plants
Charcoal Production Facilities
Coal Cleaning Plants (thermal dryers)

* This table represents industries affected by Best Available Retrofit Technology (BART) requirements of the EPA Regional Haze Rulings. The BART requirements of the regional haze rule apply to facilities built between 1962 and 1977 that have the potential to emit more than 250 tons a year of visibility-impairing pollution. Those facilities fall into 26 categories, including utility and industrial boilers, and large industrial plants such as pulp mills, refineries and smelters. (Source: http://www.epa.gov/air/visibility/factsheet.html)

BACKGROUND INFORMATION

EXAMPLES OF RECENT EPA RULES AND REPORTS INVOLVING MORTALITY EFFECTS OF $PM_{2.5}$

Title	Purpose	Date of Final Rule
Clean Air Fine Particles Rules	Designate those areas whose air does not meet the health-based standards for fine-particle pollution. Requires states to submit plans for reducing the levels of particulate pollution in areas where the fine-particle standards are not met.	Early 2006
Clean Air Interstate Rule[1]	Reduce the emissions of sulfur dioxide and nitrogen oxides from power plants through caps.	March 2005
Clean Air Nonroad Diesel Rule[2]	Reduce emissions from non-road diesel engines by integrating engine and fuel requirements.	May 2004
Guidelines for Best Available Retrofit Technology (BART) Determinations Rule	Amendments to the Regional Haze Rule that require emissions controls (BART) for industrial facilities emitting air pollutants that reduce visibility.	May 2004
Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements	Require advanced exhaust emission control devices for heavy-duty engines and vehicles. Will also reduce the level of sulfur in highway diesel fuel.	December 2000
Tier 2 Gasoline/Sulfur Rulemaking	Control air pollution from passenger cars and light trucks	February 2000
Regional Haze Rule	Prevention of any future, and the remedying of existing, impairment of visibility in Class I areas (e.g., national parks).	August 1999
Section 812 of the Clean Air Act Amendments of 1990	Provide a series of cost-benefits studies of the Clean Air Act.	1st report published October 1997, 2nd report published November 1999, 3rd report analytic blueprint released May 2003

1 Expert Elicitation Pilot Study results were used in the final Regulatory Impact Analysis conducted by EPA. See USEPA (2005) Regulatory Impact Analysis for the Clean Air Interstate Rule, Section 4.3. http://www.epa.gov/cair/pdfs/finaltech08.pdf

[2] Expert Elicitation Pilot Study results were used in the final Regulatory Impact Analysis conducted by EPA. See USEPA (2004) Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines, Appendix 9B. EPA420-R-04-007 http://www.epa.gov/nonroad-diesel/2004fr.htm#ria.

BACKGROUND INFORMATION

USE OF THE PILOT STUDY RESULTS IN THE UNCERTAINTY SECTION OF THE COST-BENEFITS ANALYSIS OF THE CLEAN AIR NON-ROAD DIESEL RULE[1]

- BenMAP model (USEPA 2003) used for benefits assessment.

- "Pooled" approach used to combine expert responses. Each expert's elicited C-R function was run through a benefits model to derive a total mortality incidence. Combined mortality estimates into an aggregate value and calculated an average mortality incidence.

- Converted each expert's percentile responses about mortality associated with long-term exposure into a custom distribution such that each percentile is correctly represented and percentiles in between are represented as continuous functions.

    - For experts specifying a log-linear C-R function (A, D and E), the following formula was used: $\Delta y = y_0 (e^{\beta \Delta x} - 1)$, where $\beta = \ln(1+B/100)$, where B is the percent change in all cause mortality associated with a 1 $\mu g/m^3$ reduction in $PM_{2.5}$. BenMAP then represented the distribution of Δy based on the custom distribution of β.

    - One expert (C) provided a set of conditional C-R functions for different baseline levels of $PM_{2.5}$ (e.g., one at 8, 10, 15 and 20 $\mu g/m^3$). Linear interpolations between the responses for each pair of points was performed (e.g., 10 to 15 or 15 to 20). Interpolated values for 13 points, ranging from 8 to 20 μg were calculated. For each conditional function, a log-linear specification was used.

    - One expert (B) specified a log-linear C-R function, conditional on an unknown threshold characterized by a triangular distribution bounded by 4 and 15 μg. The triangular distribution was discretized into 12 ranges of unit length (i.e., 4 to 5, 5 to 6, etc.) and calculated the expected value of the response at each population gridcell based on the observed baseline $PM_{2.5}$ and the probability of that baseline value exceeding the potential threshold.

- Based on air quality modeling conducted for the Nonroad Diesel preliminary control option, calculated the reduction in incidence of premature mortality associated with $PM_{2.5}$ and the value of that reduction.

- Used Monte Carlo simulations (sampling from a distribution of the reduction in mortality incidence and the distribution of Value of a Statistical Life (VSL)) to derive the distributions of the dollar values of estimated reductions in premature mortality.

- Monte Carlo process conducted using estimated distribution for each expert individually and the combined (pooled) distribution as well as for the distribution derived from the Pope et al. (2002) study.

[1] Source for text and Figures: USEPA (2004) "Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines" EPA420-R-04-007, Office of Transportation and Air Quality.

Figure 9B-4 Results of Illustrative Application of Pilot Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Modeled Preliminary Control Option for the Nonroad Diesel Rule

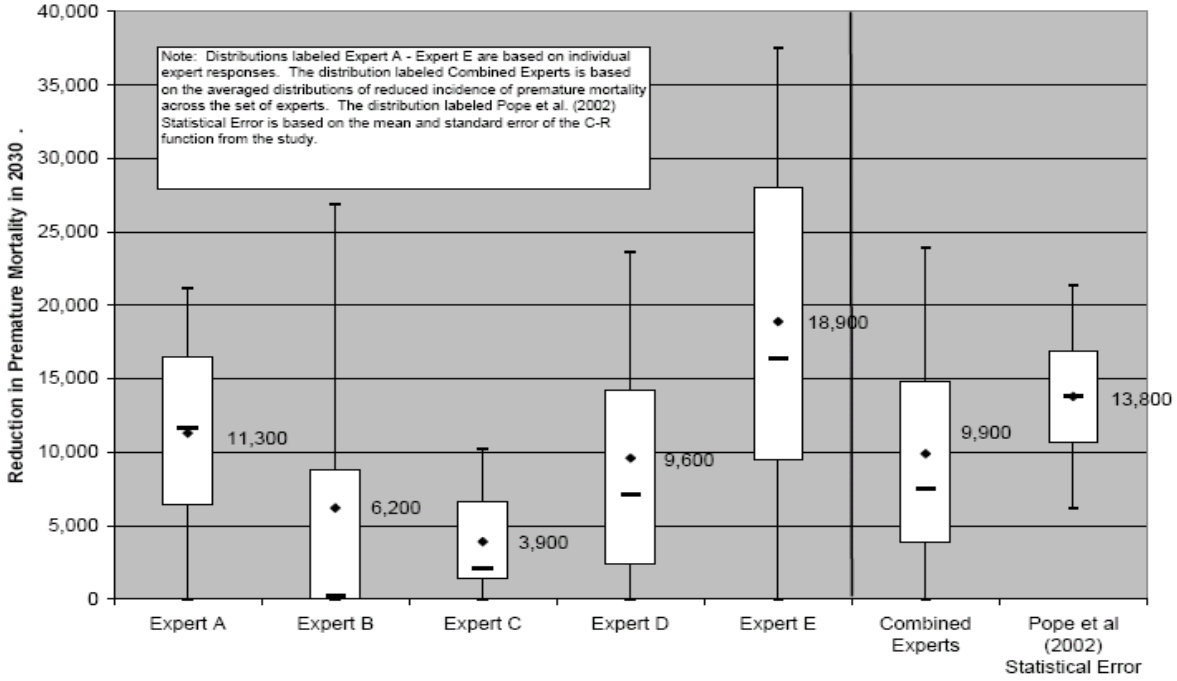
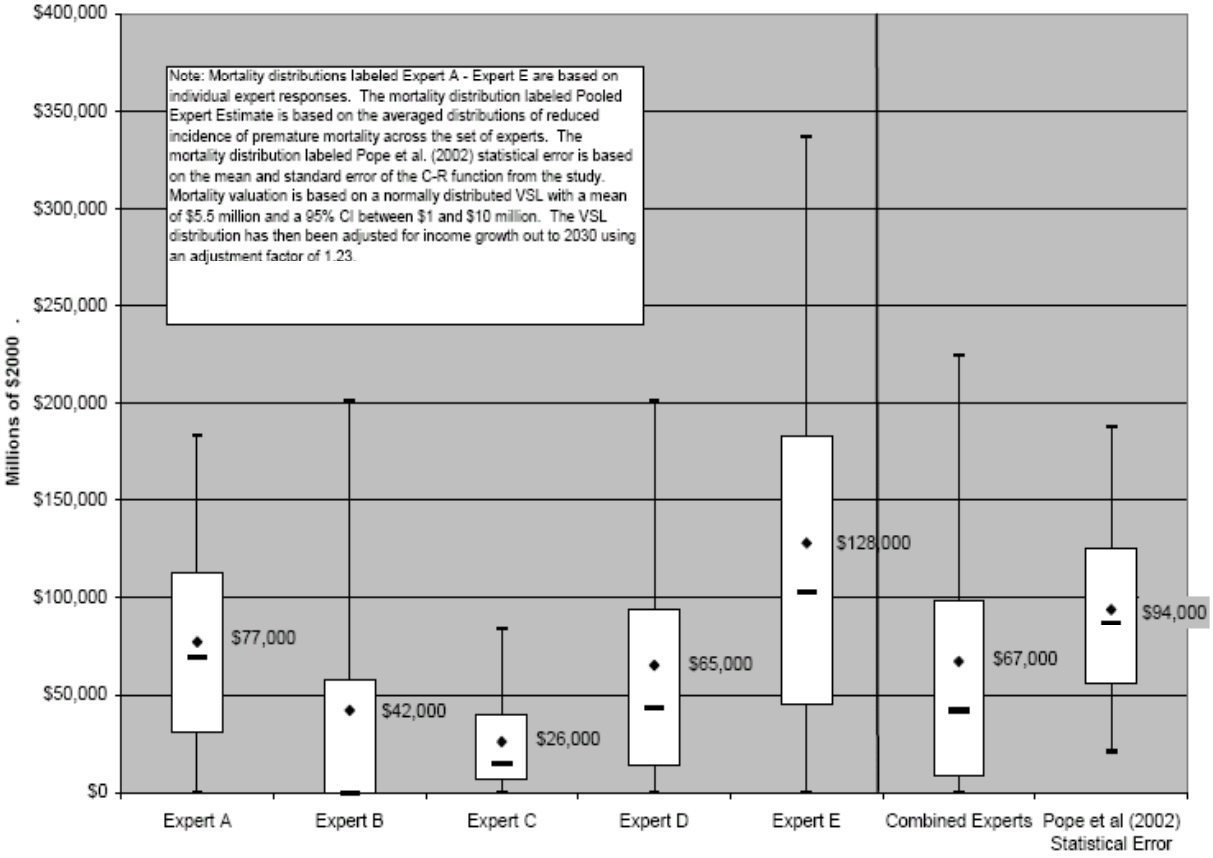


Figure 9B-6. Results of Illustrative Application of Pilot Expert Elicitation: Dollar Value of Annual Reductions in Premature Mortality in 2030 Associated with the Modeled Preliminary Control Option for the Nonroad Diesel Rule



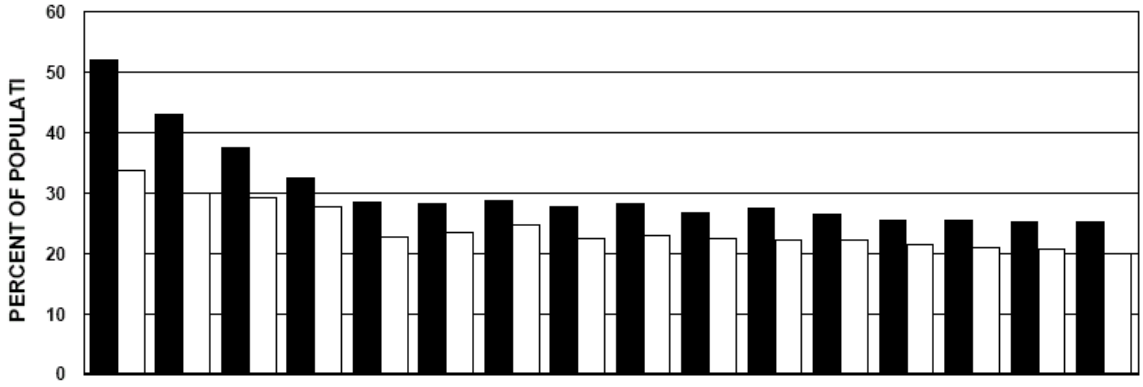
BACKGROUND TECHNICAL INFORMATION PAGES

POPULATION DEMOGRAPHICS

BACKGROUND INFORMATION

CIGARETTE SMOKING IN THE UNITED STATES POPULATION, 1965-2002

FIGURE 2: CURRENT CIGARETTE SMOKING IN PERSONS AGE 18 YEARS AND OLDER BY SEX, 1965-2002 (1,2)

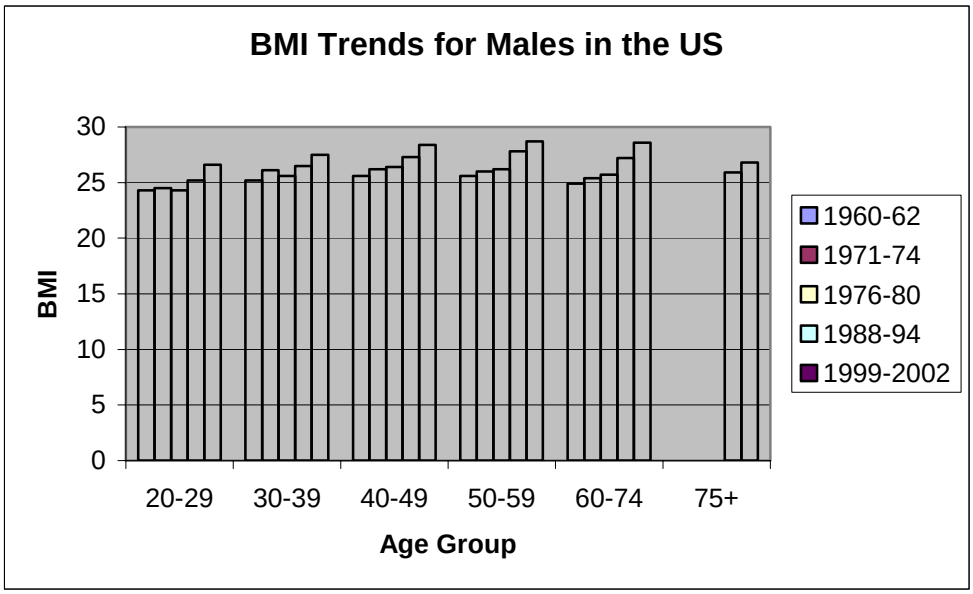


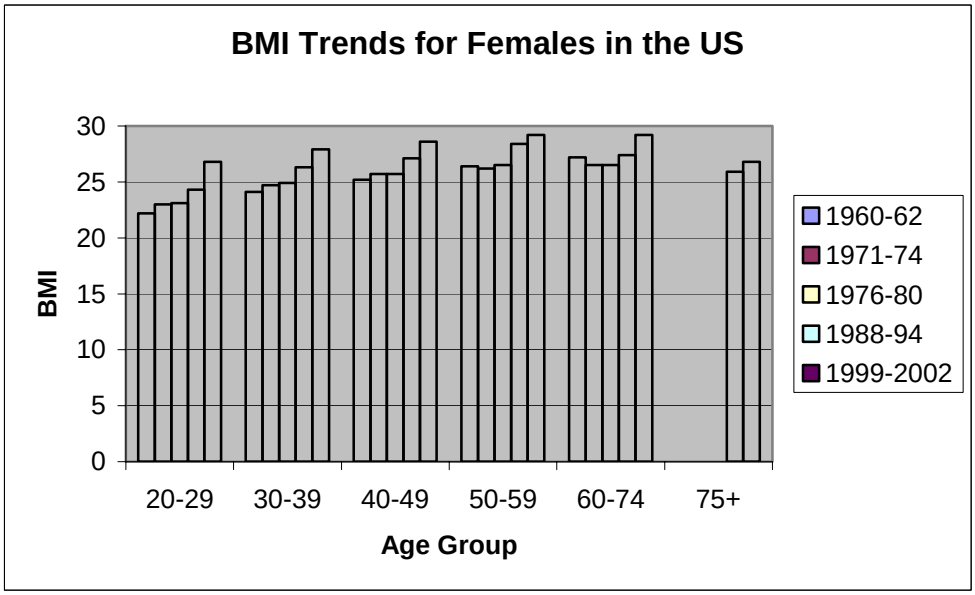
	1965	1974	1980	1985	1990	1991	1992	1993	1994	1995	1997	1998	1999	2000	2001	2002
■ MALE	51.9	43.1	37.6	32.6	28.4	28.1	28.6	27.7	28.2	27.0	27.6	26.4	25.7	25.7	25.2	25.2
□ FEMALE	33.9	29.9	29.3	27.9	22.8	23.5	24.6	22.5	23.1	22.6	22.1	22.0	21.5	21.0	20.7	20.0

SOURCE: NATIONAL HEALTH INTERVIEW SURVEY, SELECTED YEARS AND MMWR REPORTS

BACKGROUND INFORMATION

BODY MASS INDEX IN THE UNITED STATES, 1960-2002



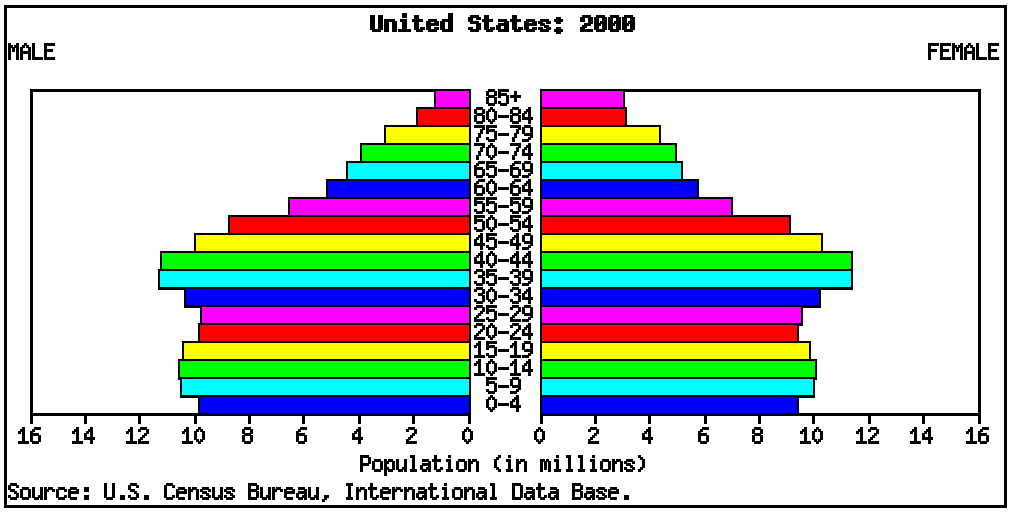


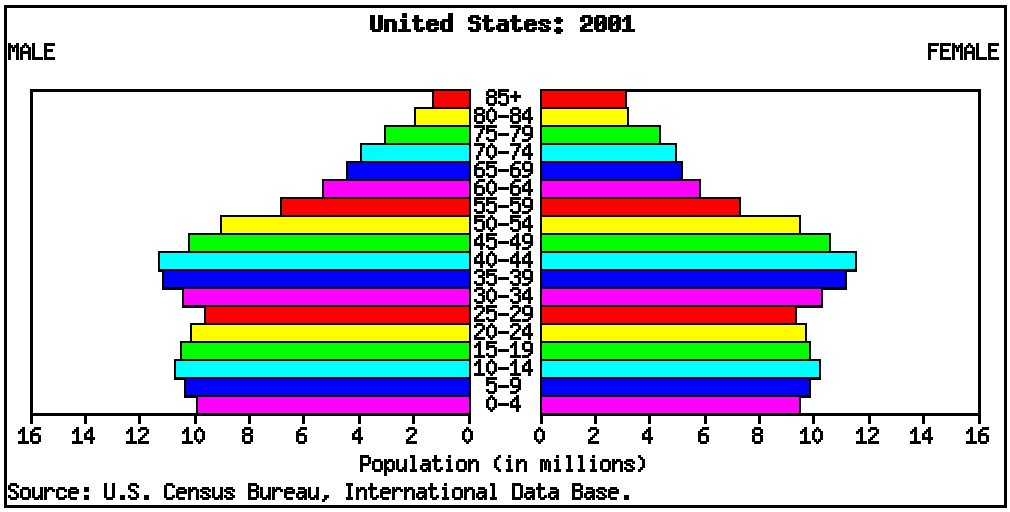
Note: There is no data for the Age Group of 75+ for the years 1960-1980.

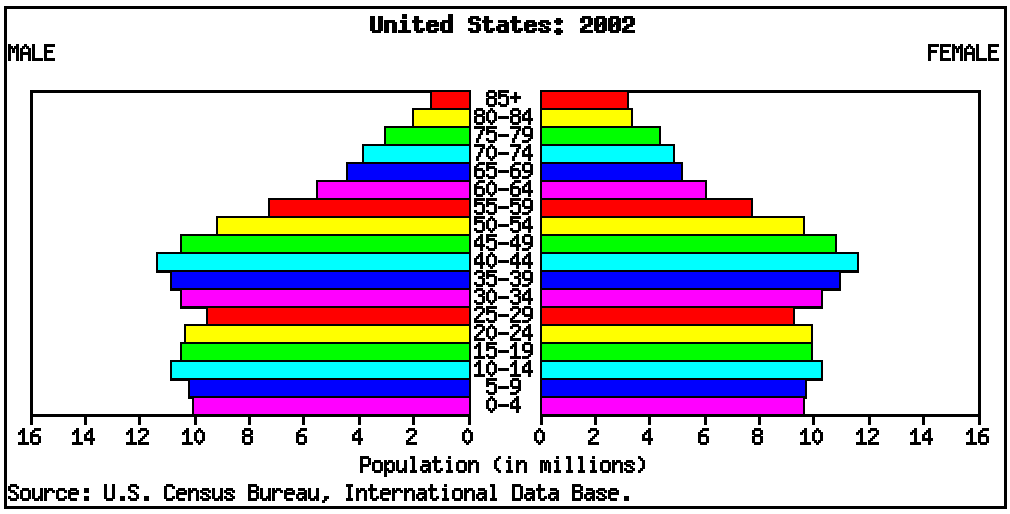
*Source: Ogden, C.L., et al. (2004). *Mean Body Weight, Height, and Body Mass Index, United States 1960-2002*. Advance Data From Vital and Health Statistics, U.S. Department of Health and Human Services, No. 347. (Data comes from the National Health Examination and the National Health and Nutrition Examination Surveys (NHANES).)

BACKGROUND INFORMATION

UNITED STATES POPULATION BY AGE GROUP AND GENDER, 2000-2004

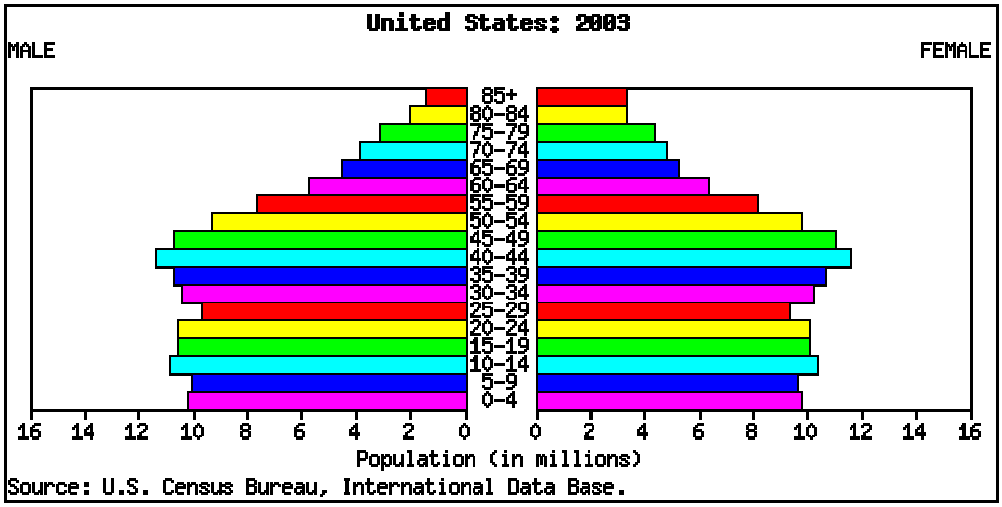


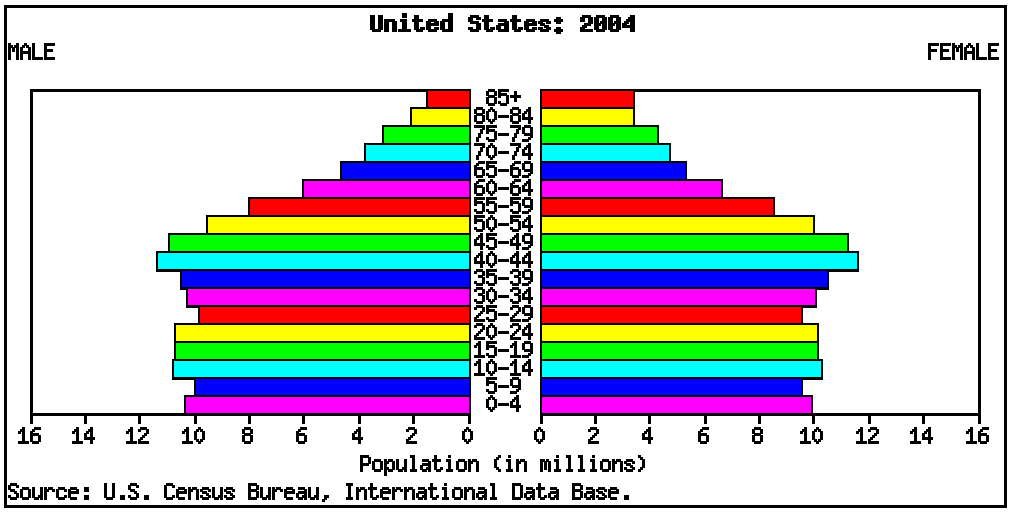




BACKGROUND INFORMATION

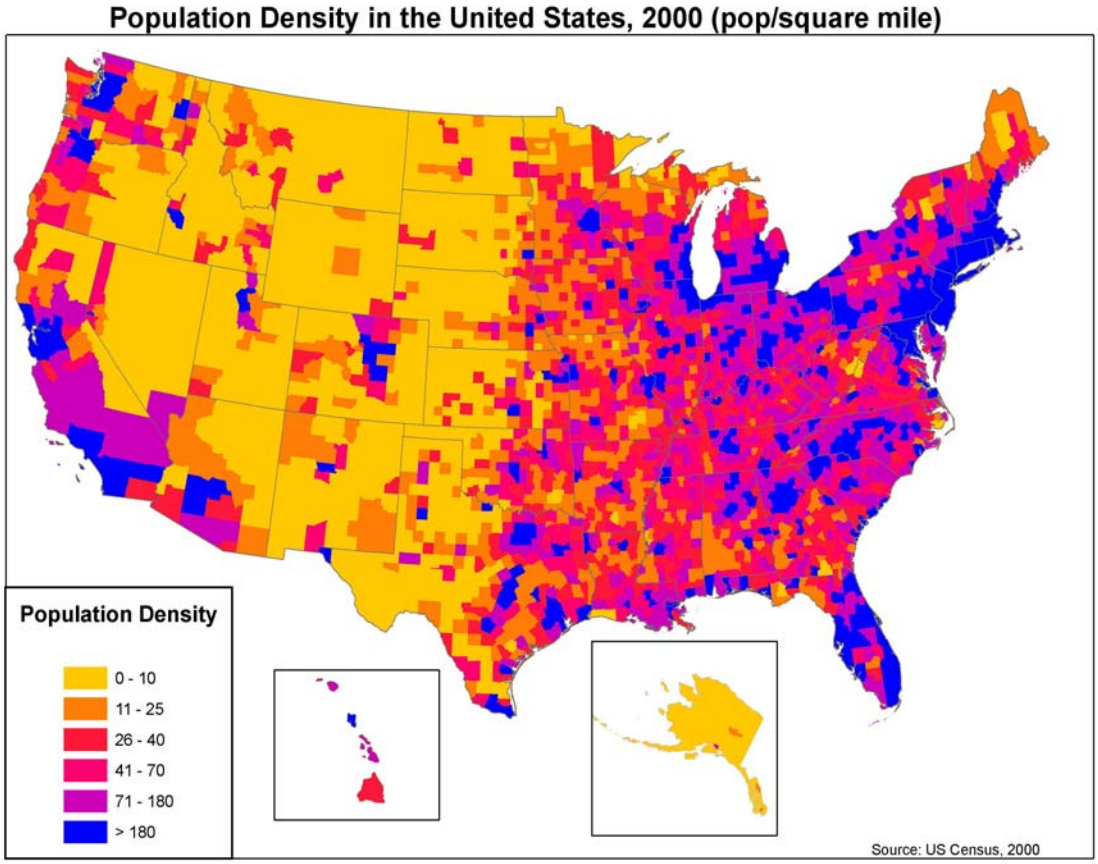
UNITED STATES POPULATION BY AGE GROUP AND GENDER, 2000-2004 (CONTINUED)





BACKGROUND INFORMATION

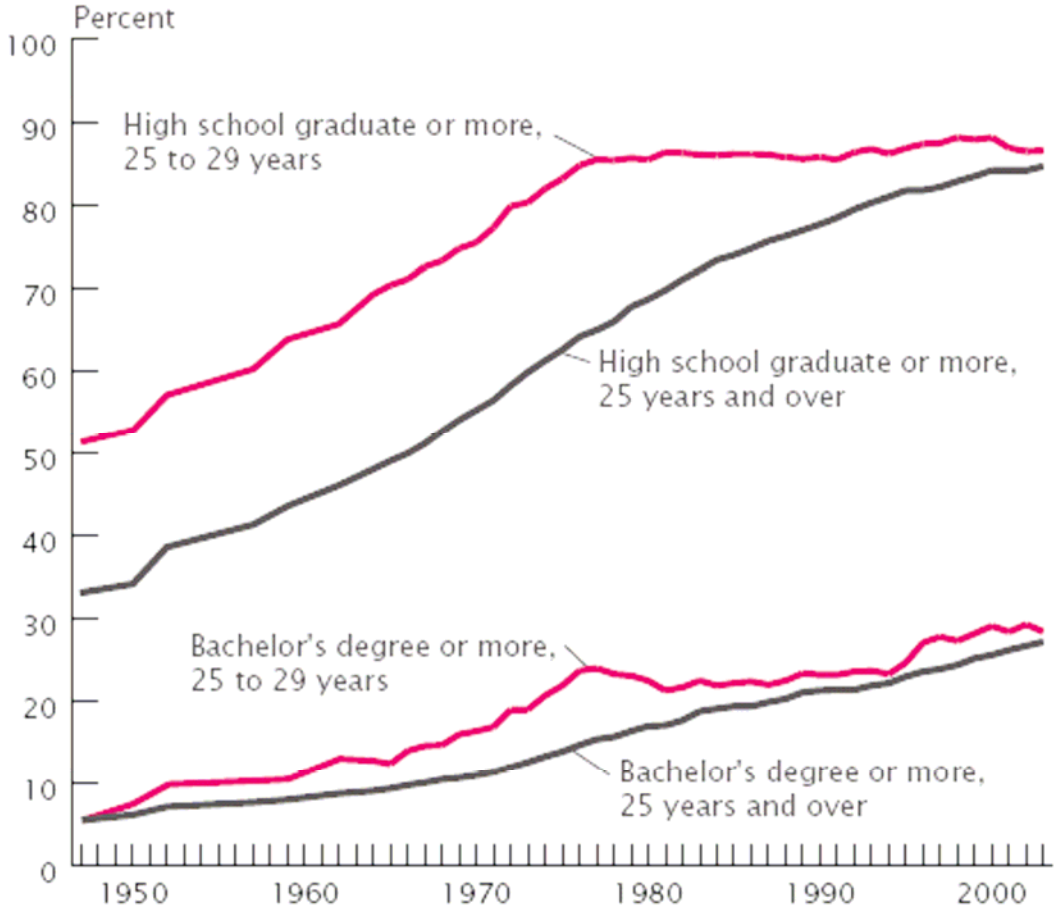
POPULATION DENSITY IN THE UNITED STATES



BACKGROUND INFORMATION

EDUCATIONAL ATTAINMENT OF THE U.S. POPULATION, 1947-2003

Figure 1.
Educational Attainment of the Population 25 Years and Over by Age: 1947 to 2003



Note: Prior to 1964, data are shown for 1947, 1950, 1952, 1957, 1959, and 1962.
Source: U.S. Census Bureau, Current Population Survey and the 1950 Census of of Population.

*Source: U.S. Census Bureau, Current Population Survey, Annual Social and Economic Supplement, 2003. http://www.census.gov/prod/2004pubs/p20-550.pdf

BACKGROUND INFORMATION

EDUCATIONAL ATTAINMENT OF THE U.S. POPULATION, 2003

Table A.
Summary Measures of the Educational Attainment of the Population 25 Years and Over: 2003

Characteristic	Number of people (in thousands)	High school graduate or more		Some college or more		Bachelor's degree or more	
		Percent	90-percent confidence interval	Percent	90-percent confidence interval	Percent	90-percent confidence interval
Population 25 years and over	185,183	84.6	84.4 - 84.8	52.5	52.3 - 52.7	27.2	27.0 - 27.4
Age group:							
25 to 29 years	18,721	86.5	86.0 - 87.0	57.4	56.7 - 58.1	28.4	27.8 - 29.0
30 to 34 years	20,521	87.6	87.2 - 88.0	58.6	58.0 - 59.2	31.5	30.9 - 32.1
35 to 39 years	21,284	87.6	87.2 - 88.0	56.5	55.9 - 57.1	29.8	29.2 - 30.4
40 to 44 years	22,790	88.4	88.0 - 88.8	56.5	55.9 - 57.1	29.1	28.6 - 29.6
45 to 49 years	21,420	89.3	88.9 - 89.7	57.4	56.8 - 58.0	29.9	29.3 - 30.5
50 to 54 years	18,814	88.7	88.3 - 89.1	58.9	58.3 - 59.5	31.1	30.5 - 31.7
55 to 59 years	15,470	86.9	86.4 - 87.4	55.1	54.4 - 55.8	29.0	28.3 - 29.7
60 to 64 years	11,930	83.0	82.4 - 83.6	47.3	46.5 - 48.1	24.5	23.8 - 25.2
65 to 69 years	9,438	76.9	76.1 - 77.7	39.1	38.2 - 40.0	19.6	18.9 - 20.3
70 to 74 years	8,673	72.8	71.9 - 73.7	36.4	35.5 - 37.3	18.5	17.7 - 19.3
75 years and over	16,123	67.5	66.8 - 68.2	32.4	31.7 - 33.1	15.4	14.9 - 15.9
Sex:							
Men	88,597	84.1	83.9 - 84.3	53.2	52.9 - 53.5	28.9	28.6 - 29.2
Women	96,586	85.0	84.8 - 85.2	51.9	51.6 - 52.2	25.7	25.4 - 26.0
Race and origin:							
White alone	153,188	85.1	84.9 - 85.3	52.9	52.7 - 53.1	27.6	27.4 - 27.8
Non-Hispanic White alone	133,488	89.4	89.2 - 89.6	56.4	56.2 - 56.6	30.0	29.8 - 30.2
Black alone	20,527	80.0	79.5 - 80.5	44.7	44.0 - 45.4	17.3	16.8 - 17.8
Asian alone	7,691	87.6	87.0 - 88.2	67.4	66.5 - 68.3	49.8	48.8 - 50.8
Hispanic (of any race)	21,189	57.0	56.5 - 57.5	29.6	29.1 - 30.1	11.4	11.1 - 11.7
Nativity:							
Native	158,128	87.5	87.3 - 87.7	54.2	54.0 - 54.4	27.2	27.0 - 27.4
Foreign born	27,055	67.2	66.6 - 67.8	42.7	42.1 - 43.3	27.2	26.6 - 27.8
Marital status:							
Never married	28,694	84.9	84.5 - 85.3	54.8	54.3 - 55.3	29.0	28.5 - 29.5
Married spouse present	113,748	87.0	86.8 - 87.2	55.9	55.6 - 56.2	30.5	30.3 - 30.7
Married spouse absent	7,389	72.5	71.6 - 73.4	38.2	37.2 - 39.2	16.1	15.3 - 16.9
Separated	4,447	74.5	73.3 - 75.7	38.6	37.3 - 39.9	13.8	12.9 - 14.7
Widowed	13,970	67.2	66.5 - 67.9	30.3	29.6 - 31.0	12.5	12.0 - 13.0
Divorced	21,382	86.5	86.1 - 86.9	50.9	50.3 - 51.5	21.0	20.5 - 21.5
Region:							
Northeast	36,182	85.7	85.4 - 86.0	50.7	50.3 - 51.1	30.3	29.9 - 30.7
Midwest	41,728	87.8	87.5 - 88.1	52.5	52.1 - 52.9	26.0	25.6 - 26.4
South	66,071	82.2	81.9 - 82.5	50.1	49.7 - 50.5	25.3	25.0 - 25.6
West	41,202	84.0	83.6 - 84.4	58.1	57.6 - 58.6	28.7	28.3 - 29.1

Source: U.S. Census Bureau, Current Population Survey, Annual Social and Economic Supplement, 2003.

*Source: http://www.census.gov/prod/2004pubs/p20-550.pdf

IEc

APPENDIX C: SENSITIVITY ANALYSIS

APPENDIX C | SENSITIVITY ANALYSIS

Due to differences in underlying assumptions (e.g., whether an expert's concentration response (C-R) coefficient distribution directly incorporates his likelihood of causality or whether it represents mortality impacts conditional on the existence of a causal mechanism), it is not possible to directly compare the 12 experts' C-R coefficient uncertainty distributions. Therefore, in order to assess the sensitivity of the study results to individual expert responses or to elements of the study design, IEc performed a simplified example benefits analysis and pooled the results across experts. This approach accomplished two things. First, it enabled us to transform each expert's distribution into a common metric (deaths avoided) and to incorporate judgments about both causality and about thresholds. Second, it produced a single distribution that could be used as the baseline for a sensitivity analysis.

The simplified benefits analysis estimated the annual avoided deaths associated with a 1 $\mu g/m^3$ reduction in annual average $PM_{2.5}$, from 12 $\mu g/m^3$ to 11 $\mu g/m^3$.[1] This calculation was performed using the entire U.S. as the exposed population. The first step in the sensitivity analysis was to estimate 12 distributions of avoided deaths using each expert's C-R coefficient distribution and the following damage function:[2]

$$D = P \times M \times (\exp(\beta \times \Delta PM) - 1)$$

Where:

D = Number of Annual Deaths Avoided

P = U.S. Population[3]

M = Background mortality rate in the U.S. (deaths/100,000 population)[4]

β = expert's C-R coefficient (percent change in mortality per 1 $\mu g/m^3$ change in $PM_{2.5}$ divided by 100)

ΔPM = change in annual average $PM_{2.5}$ ($\mu g/m^3$)

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[1] If an expert provided more than one C-R coefficient uncertainty distribution that was concentration dependent, we calculated mortality benefits using the expert's distribution at the concentration of interest.

[2] In order to create a distribution of avoided deaths, we took each of the percentiles directly specified by the expert and converted it into avoided deaths using the damage function. We then used Crystal Ball to re-create the distribution in the new metric. For example, if an expert had specified a 5th and 50th percentile to be used to create a normal distribution, we first converted his 5th and 95th percentile values into avoided deaths, and then input these two values into a normal distribution using Crystal Ball. We then ran a simulation in Crystal Ball and extracted a set of 10,000 data points from the distribution to use in the sensitivity analyses.

[3] The U.S. population data was taken from the U.S. Census Bureau website (www.census.gov). The data was from the year 2004 and only included individuals aged 25 and above.

[4] The background mortality rate for individuals in the U.S. aged 25 and over was taken from the Centers for Disease Control and Prevention website (www.cdc.gov).

Next, if an expert's distribution did not include his judgments concerning the likelihood of a causal relationship or concerning a threshold in the C-R function, we used Crystal Ball™ to integrate these judgments via Monte Carlo statistical sampling into the distribution of avoided deaths. We first multiplied the original distribution by a Yes/No distribution representing the expert's likelihood of a causal relationship.[5] To incorporate an expert's views on threshold, we multiplied the distribution incorporating causality by zero if the threshold was above the $PM_{2.5}$ concentration that was the focus of that analysis, and a one otherwise. We then generated a pooled distribution of avoided deaths by sampling with equal weights from all 12 distributions. The mean and standard deviation of this pooled distribution served as the baseline for four sensitivity analyses: one that removed each expert individually from the pooled estimate; one that assessed the influence of participation in the Pre-elicitation Workshop; one that compared experts who specified parametric distributions with those who specified "custom" distributions; and one that examined the influence of changes made by expert's to their judgments following the Post-elicitation Workshop.

To assess the potential influence of individual expert responses, we removed each expert's estimated mortality benefits data sequentially and calculated the percent change in the mean and standard deviation of the pooled dataset. Exhibit C-1 presents the results of this analysis, indicating the percent changes in the mean and standard deviation of the pooled dataset after removing each expert. The largest changes in the mean occurred as the result of removing Expert E (8 percent decrease in the mean) and Expert K (8 percent increase in the mean). The largest changes in the standard deviation occurred as the result of removing Expert E (8 percent decrease in the standard deviation) and Expert K (5 percent decrease in the standard deviation). No expert changed the pooled estimate's mean or standard deviation by more than 10 percent. We performed the same analysis at baseline $PM_{2.5}$ concentrations of 6 and 18 $\mu g/m^3$ and found results that were generally similar.

We also assessed potential effects of participation in the Pre-elicitation Workshop on the experts' judgments. The mean and standard deviation for a pooled estimate of avoided deaths for those experts who did not attend the Pre-elicitation Workshop were within 10 percent of the mean and standard deviation of the pooled estimate for Pre-elicitation Workshop attendees. Therefore, participation did not appear to have a significant effect on the results.

In addition, we examined the differences between distributions from experts specifying a custom, non-parametric distribution and those specifying a parametric distribution. The standard deviation of the pooled estimate for the parametric group was 23 percent greater than that for the non-parametric group. (The means of the two distributions remained close to that of the overall mean for all experts). This suggests that the use of parametric distributions led to distributions with increased uncertainty compared to experts who provided percentiles of a non-parametric distribution.

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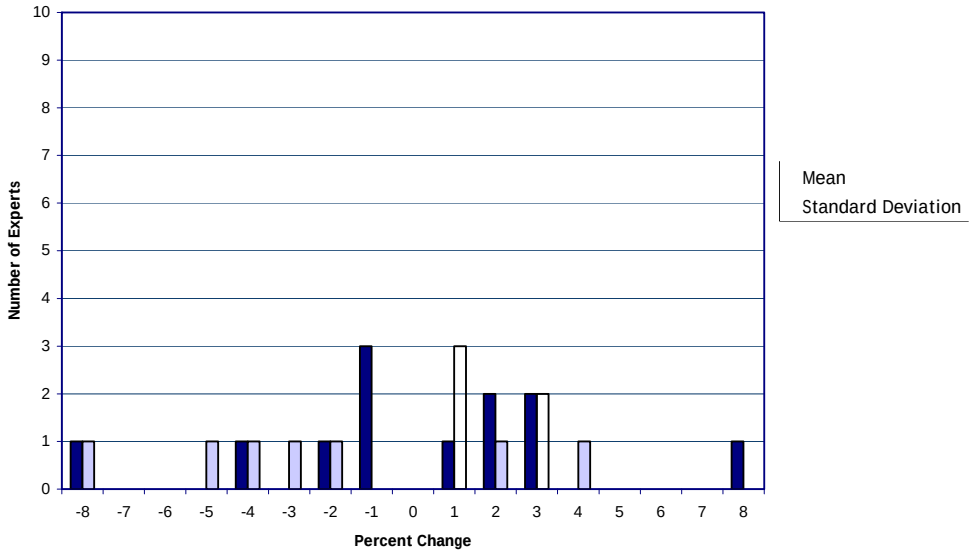
[5] For example, if an expert specified a causal likelihood of 95 percent, his distribution would be multiplied by one for 95 percent of the time and zero for five percent of the time. This approach assumes that the expert's causality distribution and conditional mortality effect distribution are independent.

Finally, we assessed the impact of the Post-elicitation Workshop on the pooled mean and standard deviation, since a few of the experts elected to make modifications to their judgments after the workshop. We found that the mean pooled estimate of mortality benefits increased by approximately 1 percent after the workshop and the standard deviation did not change.

EXHIBIT C-1: PERCENT CHANGE IN THE MEAN AND STANDARD DEVIATION OF AN EXAMPLE POOLED MORTALITY BENEFIT ESTIMATE BASED ON RESULTS OF THE EXPERT ELICITATIONS, AFTER REMOVING EACH EXPERT



Note: Mortality benefits were pooled of a 1 $\mu g/m^3$ decrease in $PM_{2.5}$, beginning at an annual average concentration of 12 $\mu g/m^3$. The pooled estimate weighted each expert equally.