Estimate of Premature Deaths Associated with Fine Particle Pollution (PM2.5) in California Using a U.S. Environmental Protection Agency Methodology

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California Air Resources Board

California Environmental Protection Agency

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Acronyms and Abbreviations

ACS	American Cancer Society
ARB	Air Resources Board
BenMAP	Benefits Mapping and Analysis Program software
CASAC	Clean Air Scientific Advisory Committee
CCACA	Council on Clean Air Compliance Analysis
C-R function	concentration-response function
CPS-II	Cancer Prevention Study II
HEI	Health Effects Institute
ISA	Integrated Science Assessment
NO_X	nitrogen oxides
PM	particulate matter
PM2.5	fine particulate matter; particulate matter 2.5 micrometers aerodynamic diameter and smaller
PM10	particulate matter 10 micrometers aerodynamic diameter and smaller
RIA	Regulatory Impact Analysis
RR	relative risk
SIP	State Implementation Plan
SO_X	sulfur oxides
U.S. EPA	United States Environmental Protection Agency
VOC	volatile organic compounds

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EXECUTIVE SUMMARY

The United States Environmental Protection Agency's (U.S. EPA) recently released "Quantitative Health Risk Assessment for Particulate Matter" provides national estimates of premature mortality associated with fine particulate matter pollution (PM2.5), supported by its finding that the scientific evidence shows a causal connection between mortality and exposure to PM2.5. This report describes the U.S. EPA's risk assessment methodology for calculating premature mortality, and its 2009 Integrated Science Assessment for particulate matter that provides the underlying scientific basis for the calculations. These U.S. EPA reports were prepared as part of U.S. EPA's periodic review of the National Ambient Air Quality Standards (NAAQS) for particulate matter. The U.S. EPA risk assessment estimated premature deaths associated with PM2.5 nationwide, and in 15 urban areas including Los Angeles and Fresno. This report applies the U.S. EPA methodology to California on a statewide basis.

The U.S. EPA's reports were peer reviewed in a public process by the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter Review Panel, an independent peer review body of national scientists. The methodology described in this report is used to quantify the premature deaths associated with current levels of PM2.5 in California, and to estimate the premature deaths avoided by achieving compliance with the current annual air quality standard for PM2.5. This report also describes the method used by U.S. EPA to calculate the health benefits of PM2.5 emission reductions from specific source categories.

The foundation of the methodology is the association between long-term PM2.5 concentrations and premature death, which is provided by peer reviewed health studies. There are a large number of published health studies that estimate the additional risk of mortality due to long-term exposure to PM2.5. U.S. EPA's new quantitative health risk assessment for particulate matter uses a 2009 study (Krewski et al., 2009) for the core analysis. This study is an extension of a 2002 study (Pope et al., 2002) used in the previous PM2.5 NAAQS risk assessment. This report estimates premature death from PM2.5 in California based on the 2009 Krewski study.

Using U.S. EPA's methodology, the estimated number of annual PM2.5-related premature deaths in California is 9,200 with an uncertainty range of 7,300 – 11,000. This estimate of premature deaths is based on the latest exposure period in the 2009 Krewski study with data from 116 U.S. cities and about 500,000 people.

I. NATIONAL PM2.5 STANDARDS

Clean Air Act Requirements

The federal Clean Air Act (Section 109) directs the U.S. Environmental Protection Agency (U.S. EPA) to promulgate national ambient air quality standards (NAAQS or standards) through a process which includes review by an independent scientific review committee. For over twenty years independent review of the science supporting national air quality standards has been provided by the Clean Air Scientific Advisory Committee (CASAC or scientific advisory committee). The Clean Air Act requires U.S. EPA to review the standards every five years, and the agency is considering whether to strengthen the standard for fine particles (PM2.5) based on the latest scientific peer reviewed studies. This latest review has resulted in the preparation of a series of documents on the adverse health effects of PM2.5 which have all undergone review by the scientific advisory committee.

U.S EPA first established air quality standards for particulate matter in 1971, which were expressed as "total suspended particulates." In 1987, new standards were added to focus on the inhalable size fraction defined as PM10 (particles less than 10 microns in diameter). As monitoring techniques further improved and more health studies were completed, new standards were adopted in 1997 to focus on one of the smallest components of PM10, the fine particles defined as PM2.5 (particles less than 2.5 microns in diameter). The PM2.5 standards were reviewed and updated in 2006. The current review process builds on previous peer reviewed studies, with emphasis on newly available studies published through May 2009. As of June 2010, U.S. EPA's proposed and final rulemaking notices for the current review of the standards are scheduled for November 2010 and July 2011, respectively (Table 1).

Table 1: History of PM2.5 Standards (μg/m^3)

Year	24-hour Standard	Annual Standard
1997	65	15
2006	35	15
2010*	35-30	13-11

*Ranges considered in June 2010 Second Draft Policy Assessment.

After U.S. EPA promulgates a new or revised standard, a series of mandatory Clean Air Act requirements are triggered beginning with the identification of areas of the country which do not comply with the standard. Once identified, states with such areas must prepare a plan to demonstrate how the standard will be met by the mandatory deadlines in the Clean Air Act. The plans are called State Implementation Plans (SIPs or plans), and they provide the enforceable mechanism to implement air quality standards. The air quality plans developed by California to meet Clean Air Act requirements must be approved by the U.S. EPA, and implemented by California according to the mandatory deadlines.

The Clean Air Act includes sanctions if states do not comply with the requirements for implementing the health-based air quality standards.

In addition to adopting national air quality standards, U.S. EPA also adopts national regulations to reduce air pollution from cars, trucks, industrial facilities and other sources of air pollution. In states with lower pollution levels, federal regulations are often enough to show compliance with air quality standards. However, achieving federal air quality standards in California is more challenging than anywhere else in the nation. The Clean Air Act recognizes California's challenges by providing the unique authority to regulate air pollution beyond what other states can do. As U.S. EPA and ARB adopt regulations necessary to meet federal air quality standards, both agencies calculate the health benefits of these actions.

U.S. EPA quantifies the nationwide benefits of achieving the PM2.5 standards, including reduced premature mortality, as part of the economic analysis of its regulations. The health benefits of reducing PM2.5 in California are high from a national perspective because Californians are exposed to some of the highest levels of PM2.5 air pollution in the country.

Nature of PM2.5 Air Pollution

PM2.5 is a mixture of multiple constituents, including both directly emitted particles ("primary particles") and particles that form in the atmosphere ("secondary particles") through chemical reactions and physical transformations. The PM2.5 air quality standard is defined on a mass basis as measured by air monitors. Both primary and secondary particles are captured by the monitors, and both types of particles are regulated in order to comply with the standard. The key sources of PM2.5 are combustion processes, although other pollution sources also contribute. In California, air quality monitoring indicates that both primary and secondary PM2.5 significantly contributes to non-compliance with the PM2.5 standards.

In California, local air districts regulate PM2.5 pollution from industrial sources. U.S EPA and ARB regulate PM2.5 emissions from mobile sources, including both gasoline and diesel engines. Uncontrolled diesel engines emit much larger quantities of primary PM2.5 particles than gasoline engines, as well as significant amounts of nitrogen oxides (NO_X) that form secondary PM2.5. The relative contribution of PM2.5 from transportation compared to sources such as power plants is much greater in California than nationwide. However, in order to meet air quality standards, PM2.5 emissions from virtually all combustion sources are regulated in California, from small engines and boilers to residential wood burning.

California's PM2.5 Plans

U.S. EPA determined that the South Coast Air Basin and San Joaquin Valley were out of compliance with the federal PM2.5 standard which triggered mandatory development of SIPs with new regulatory strategies. In 2007-2008, California submitted the required PM2.5 SIPs to the U.S. EPA. These region-specific plans outline how California will attain the current annual PM2.5 standard by 2014, and include descriptions of the types of regulations planned for adoption. The plans for the South Coast and San Joaquin Valley Air Basins rely on reductions in directly emitted PM2.5 as well as in pollutants that form PM2.5 in the atmosphere.

State law gives the ARB the responsibility to ensure that SIPs meet federal requirements, and to adopt regulations necessary to meet federal air quality standards. This regulatory responsibility and authority applies to a number of different types of air pollution sources including cars, trucks, construction equipment, portable engines, recreational boats, fuels, lawn and garden equipment, and consumer products.

California's local air districts are responsible for development of SIPs for their regions, and for regulating industrial and commercial sources of air pollution located within their jurisdictions. Compared to other parts of the nation, the mix of air pollution sources in California is less industrial so relatively more emission reductions must be achieved through ARB regulations of mobile sources.

U.S. EPA Scientific Review Process

U.S. EPA begins the process to adopt or revise an air quality standard by developing an Integrated Science Assessment (ISA or science assessment), which is a comprehensive review of published scientific information. The science assessment describes the state of the science on topics including the chemistry and physics of the pollutant, causality determinations for health effects, monitored air pollution data, background concentrations of pollutants, population exposure, and other effects. The science assessment forms the scientific basis for the review of an air quality standard. The next step is development of a quantitative risk assessment, which builds upon the health effects evidence presented and evaluated in the science assessment, as well as the advice of the science advisory committee.

Over the past two years, U.S. EPA has been in the process of evaluating the latest science on the health effects of inhalable particulate matter, including fine particulate matter (PM2.5), as part of a review of its current air quality standards. In December 2009, U.S. EPA released its "Integrated Science Assessment for Particulate Matter."[1] In June 2010, U.S. EPA released its "Quantitative Health

[1] Available at: http://www.epa.gov/ncea/pdfs/partmatt/Dec2009/PM_ISA_full.pdf

Risk Assessment for Particulate Matter"[2] which includes nationwide estimates of premature mortality. This new U.S. EPA risk assessment forms the basis for the calculation of premature deaths associated with long-term exposure to PM2.5 in California presented in this report.

II. U.S. EPA ASSESSMENT OF PREMATURE MORTALITY FROM PM2.5

U.S. EPA Integrated Science Assessment

In December 2009, U.S. EPA released its most recent health effects review for particulate matter, including PM2.5, which evaluated hundreds of epidemiology, toxicology, and human exposure studies. The U.S. EPA produced two drafts of the science assessment, which were released for public comment, and were peer reviewed in a public process by the Clean Air Scientific Advisory Committee. The final science assessment reflected two sets of CASAC peer review comments.[3] The 2009 report builds on the two past assessments, in 1996 and 2004, which were also reviewed by CASAC.

In U.S. EPA's 2004 report of health effects of particulate matter pollution, the discussion of mortality and long-term exposure placed the greatest weight on the findings of the American Cancer Society Study and Harvard Six Cities studies which were found to be broadly representative of the U.S. population. Collectively, these and other studies were found to provide strong evidence that long-term exposure to PM2.5 was associated with increased risk of mortality. Effect estimates for mortality (all cause) ranged from 6 to 13% increased risk per 10 $\mu g/m^3$ of PM2.5. The U.S. EPA 2009 report finds that recent evidence is largely consistent with past studies, further supporting the evidence of associations between long-term PM2.5 exposure and increased risk of mortality.

The 2009 science assessment discusses scientific studies linking PM2.5 to a variety of health effects, including cardiovascular and respiratory effects. These effects are evaluated for both short-term and long-term exposures. The assessment also discusses the relative susceptibility of various populations to the effects of particulate matter exposures, including the young, elderly, and individuals with pre-existing disease. Based on recent inhalation studies,

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[2] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf

[3] Available at: http://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/73ACCA834AB44A10852575BD0064346B/$File/EPA-CASAC-09-008-unsigned.pdf (first draft)

http://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/151B1F83B0231455852576780068 36B9/$File/EPA-CASAC-10-001-unsigned.pdf (second draft)

potential biological mechanisms underlying the health effects of particulate matter pollution are identified, including systemic inflammation, changes in heart autonomic response, and changes in lung function.

A significant addition to the science assessment is a U.S. EPA framework for evaluating the causal nature of air pollution-induced health or environmental effects. The framework recognizes that causality determinations are based on evaluation and synthesis of evidence from across scientific disciplines. The science assessment discusses the types of scientific evidence used in establishing causality, including epidemiological studies of humans, controlled human exposure studies, and animal exposure studies.

Epidemiological studies provide information on observed associations between health effects and human exposures to air pollution. Such studies need to consider potential "confounding" or confusion of effects by extraneous factors such as smoking. Controlled human exposure studies and experimental animal data can help in understanding the biological plausibility of effects observed in human epidemiological studies. The U.S. EPA framework identifies various aspects to consider in a weight of evidence approach to determining causality, including the following:

- Consistency of the observed effect in multiple independent studies
- Multiple types of evidence supporting epidemiological observations
- Biological plausibility of the observed associations
- Evidence of increasing health effects with greater exposure
- Strength and specificity of the observed association
- Evidence linking changes in exposures to changes in health effects

U.S. EPA points out that the above list of aspects to be considered is not designed to be applied as a checklist, but rather to determine the weight of evidence for causality determinations. In the science assessment, U.S. EPA evaluates the results of recent relevant publications, building on the evidence available during the previous NAAQS review, to draw conclusions on the causal relationships between pollutant exposures and health effects. Annexes to the integrated science assessment provide details of the literature published since the last NAAQS review.

U.S. EPA's framework uses the following five categories for causal determinations:

- Causal relationship
- Likely to be a causal relationship
- Suggestive of a causal relationship
- Inadequate to infer a causal relationship
- Not likely to be a causal relationship

After making a determination on causality, the next step is to quantify health risk based on an understanding of the quantitative relationship between pollutant exposures and health effects.

U.S. EPA's 2009 science assessment states "Collectively, the evidence is sufficient to conclude that the relationship between long-term PM2.5 exposures and mortality is causal".[4] The science assessment finds when looking at cause of death, the strongest evidence is for mortality due to cardiovascular disease. The science assessment discusses in detail how the most recent health studies build on the core studies evaluated in the 2004 assessment which U.S. EPA prepared in its previous review of particulate matter standards. The 2009 science assessment discusses the findings related to premature mortality in the most recent analyses of the American Cancer Society Study, the Harvard Six-Cities Study, and eleven recent studies, including California-specific studies. The new assessment discusses the nature and findings of each study, and provides the scientific basis for selecting the best studies to use in quantifying health effects of PM2.5 exposure, including premature mortality.

U.S. EPA PM2.5 Risk Assessment

Overview

As part of U.S. EPA's last NAAQS review completed in 2006, the agency conducted a risk assessment to quantify various health effects associated with particulate matter, including premature mortality. That assessment focused on nine urban areas and included estimates of risk of total mortality (non-accidental), cardiovascular-related, and respiratory-related mortality. U.S. EPA's June 2010 Quantitative Health Risk Assessment for Particulate Matter also includes estimates of premature mortality associated with long-term exposure to PM2.5. The risk assessment relies on the December 2009 Integrated Science Assessment which was peer reviewed by CASAC. The risk assessment includes a national scale analysis as well as a case study analysis of 15 urban areas, including Los Angeles and Fresno.

In selecting epidemiological studies for quantifying risk, U.S. EPA focused on the two large multi-city studies used in previous assessments – the American Cancer Society and Harvard Six Cities studies. In modeling premature mortality for long-term PM2.5 exposure in the 15 urban areas, U.S. EPA used the latest reanalysis of the American Cancer Society dataset (Krewski et al., 2009) for the core analysis. The Harvard Six Cities study, which shows higher risk than other studies, was used in sensitivity analyses designed to explore the potential range of risk. For the national scale analysis U.S. EPA also relied on Krewski et al.

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[4] Available at: http://www.epa.gov/ncea/pdfs/partmatt/Dec2009/PM_ISA_full.pdf, page 7-96.

(2009), with additional estimates based on the extended analysis of the Six Cities study (Laden et al., 2006).

In addition to quantifying premature mortality from current levels of PM2.5 air pollution, the risk assessment looks at the benefit of attaining air quality standards. As part of the NAAQS review process, U.S. EPA also quantifies the potential benefits of alternative standards which are under consideration. This ARB report focuses on one aspect of the risk assessment – the estimates of premature mortality associated with long-term exposure to PM2.5. While U.S. EPA's integrated science assessment made causal determinations for both short-term and long-term exposures to PM2.5, the risk assessment points out that mortality associated with long-term exposure is likely to include mortality related to short-term exposures. This ARB report therefore focuses on U.S. EPA's quantification of premature mortality associated with long-term exposures.

In the risk assessment process, U.S. EPA used the following criteria for selecting health effects to include:

- Weight of evidence regarding causality
- Significance of the health effect
- Availability of well-conducted epidemiological studies that provide concentration-response functions
- Availability of sufficient air quality monitoring data in the areas included in the epidemiological studies
- Availability of baseline incidence rates for the selected health effects for NAAQS decision-making

Based on these criteria, U.S. EPA selected four categories of premature mortality for quantification for long-term PM2.5 exposure: all-cause mortality, ischemic heart disease-related, cardiopulmonary-related, and lung cancer-related. CASAC peer reviewed two drafts of the particulate matter NAAQS risk assessment. The panel supported the methodology adopted and the categories selected. U.S. EPA also applied the above criteria to health effects other than premature mortality, some of which were quantified, but these endpoints are not discussed in this report.

The key elements of the PM2.5 risk assessment are an exposure assessment based on air quality data, the PM2.5 concentration response function from epidemiological studies, baseline health incidence information, and population in the study area. A concentration-response function (C-R function) is a mathematical equation that describes the relationship between exposure, in this case long-term exposure to PM2.5, and a health outcome, in this case, mortality. The C-R function expresses the relative risk of mortality associated with an incremental change in PM2.5 concentration.

U.S. EPA Selection of Core Studies

U.S. EPA has quantified the health impacts of particulate matter exposure using epidemiological studies since 1997, using the results from two U.S. studies, the American Cancer Society study and the Harvard Six Cities study, as the basis for the first annual-average NAAQS for PM2.5. Updated and extended follow-ups of these two studies have produced results that are consistent with the original papers, but with improved statistical methods.

The ACS prospective cohort study (Pope et al., 2002) was used in the particulate matter risk assessment that was part of the 2006 review of the PM standards. In its latest risk assessment, U.S. EPA selected an extension of this study, Krewski et al. (2009), as the primary basis for estimating premature mortality associated with long-term exposure to PM2.5. The ACS data set has consistently been selected for estimating premature mortality related to long-term exposure to PM2.5 because of the large study population and the study's national coverage.

U.S. EPA cited a number of advantages of this study as the basis for the concentration-response functions used in the core analysis, including:

- Additional air quality analysis extending the study period to eighteen years
- Rigorous examination of a range of model forms and effect estimates
- Coverage for social, economic, and demographic variables to allow for consideration of potential confounding
- A related analysis considering spatial gradients in PM2.5 concentration and whether they effect response models
- A large data set with up to about 500,000 individuals and 116 metropolitan statistical areas.

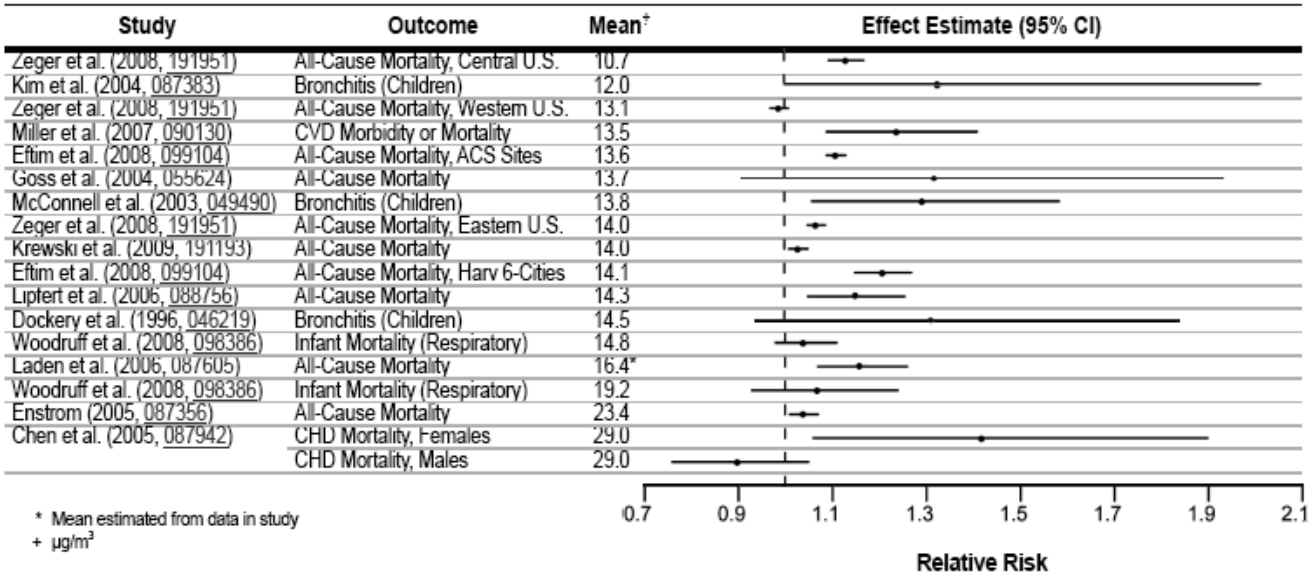
In addition to the ACS and Harvard Six Cities studies, U.S. EPA considered a number of other studies as candidates for the core PM2.5 premature mortality analysis, and the risk assessment provides a brief summary of the rationale for not selecting other studies.[5] The types of limitations discussed include use of visibility data to estimate PM2.5 levels, lack of baseline incidence data necessary for quantitative analysis, and lack of confounder control for smoking.

Figure 1 (U.S. EPA, 2009) shows the relative risks (RR), shown as solid circles, and the 95% confidence intervals, shown as the horizontal lines through the circles, from U.S. multi-city studies examining the association between long-term exposure to PM2.5 and cardiovascular and respiratory effects, and mortality. The RR represents the percentage change in risk of the stated health endpoint with a 10 $\mu g/m^3$ change in the annual average PM2.5 concentration. The 95%

[5] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, page 3-38.

confidence interval provides a range within which the true value is expected to be found. The vertical dashed line represents a no effect level (RR=1). Relative risks greater than 1 suggest an effect, although for a relative risk to be statistically significant, the 95% confidence interval must not include 1.

Figure 1: Summary of effect estimates (per 10 µg/m^3) by increasing concentration from U.S. studies examining the association between long-term exposure to PM2.5 and cardiovascular and respiratory effects, and mortality*



*From Figure 2-2 of U.S. EPA Integrated Science Assessment (2009), page 2-15

As can be seen in Figure 1, virtually all of the relative risks are greater than 1, pointing to an effect of long-term exposure to PM2.5 on mortality. In some cases the 95% confidence intervals surrounding these relative risks include 1, and so not all of these associations are statistically significant. There are several reasons that could explain why not all of the relative risks shown are statistically significant. In some cases, the study may not have sufficient statistical power due to the number of participants, and/or to the number of cities included. In other cases there may be less than optimal consideration of and adjustment of the statistical models used to account for factors that are related to the endpoint under study yet have no relationship with PM2.5 exposure. These factors are called confounders, and a few examples include age, income, and educational attainment. However, overall, the summary of available U.S. studies presented in this figure points to the conclusion that long-term exposure to PM2.5 is associated with mortality.

History of Key Health Studies

The American Cancer Society initiated the Cancer Prevention Study II (CPS-II) cohort of subjects in 1982 to study the influence of environmental tobacco smoke

exposure on lung cancer. There are about 1.2 million people enrolled in this cohort, although air quality data are available for only about 300,000 or 500,000, depending on the published study.

This well-characterized cohort of subjects provided the opportunity for the relationship between long-term PM2.5 exposure and mortality to be explored (Pope et al., 1995). The investigators estimated PM2.5 exposure for the portion of the cohort that lived in 50 metropolitan areas included in the U.S. EPA Inhalable Particulate Network of air quality monitors. Participants were at least 30 years of age and lived in households where at least one individual was 45 years of age or older. Mean age at enrollment was 56.6 years and participants lived in all 50 states, the District of Columbia, and Puerto Rico. Each participant completed an extensive questionnaire at enrollment that included age, gender, weight, height, demographic characteristics, medical history, medication use, occupational exposures, dietary habits, alcohol and tobacco use, and exercise and health-related behaviors. There have been no updates to the questionnaires during the follow-up period, although information on deaths of participants was periodically updated for follow-up analyses. The study compared the risk of death between metropolitan areas, but did not examine risk over time. Information from approximately 300,000 individuals was included in the first publication describing this cohort (Pope et al., 1995).

The original ACS study (Pope et al., 1995) was extensively reanalyzed by Krewski et al. (2000), which was coordinated by the Health Effects Institute (HEI). This reanalysis validated and replicated the original findings reported by Pope et al. (1995). In addition, the Krewski et al. (2000) reanalysis included a number of additional exploratory analyses that identified and addressed issues related to spatial autocorrelation in the study data set. Spatial autocorrelation refers to the statistical issue that data points near to each other are more similar than those that are farther apart. An update to the ACS study with longer follow-up was published by Pope et al. (2002). Epidemiologic analyses, such as those performed by Krewski et al. (2009) must take this into account in the statistical model used so that the effect of PM2.5 can be isolated from influence by other factors that may affect mortality but are not related to PM exposure. For example, it would be expected that people who live near to each other would have responses to PM2.5 that are more alike than those who live far apart. This is due to the influence of factors that make neighbors more alike, such as similar exposures, and similar socioeconomic characteristics. Subsequent follow-up analyses explicitly investigated spatial autocorrelation in the extended data sets (Pope et al., 2002; Krewski et al., 2009). Improved methods of adjusting for occupational exposures have also been instituted since the Krewski et al. (2000) reanalysis study, as well as for climate differences among the study areas.

The most recent follow-up of the ACS study cohort, with a total follow-up period of 18 years, is Krewski et al. (2009). This extension includes exposure assessments based on the original time period of 1979 to 1983, and a new

exposure period of 1999 to 2000. The latter exposure period allowed the cohort size to be increased to approximately 500,000 subjects in 116 metropolitan areas due to the increased availability of PM2.5 data beginning in 1999. Analyses were performed using the two exposure periods separately and averaged. Similar results were reported for all three analyses.

A major focus of the 2009 Krewski analysis was to better account for covariates, those risk factors for death not related to PM2.5 exposure, such as age, gender and gaseous air pollutants that vary spatially. These characteristics vary across and between regions and neighborhoods, so analytical models need to be adjusted for covariates and factors that vary spatially, to remove their influence so that the true effect of PM2.5 exposure on mortality can be measured.

The Krewski et al. (2009) study also explored several methods to control for ecologic covariates. Ecological covariates are neighborhood-level factors, such as education level, income and other socioeconomic factors, related to the environment in which a person lives that influence the outcome under study. These are factors that affect mortality but are not related to air pollution exposure. The seven selected for inclusion had a significant influence on the statistical model output and were previously identified by Krewski et al. (2000). Control for ecological variables tends to slightly increase the magnitude and uncertainty of the effect estimates compared to models that did not include ecological covariates. Including these factors in the model removes their influence on model output, and helps to better isolate the effect of PM2.5 exposure. The 2009 analysis estimated hazard ratios (HR) for all-cause, cardiopulmonary, lung cancer, and ischemic heart disease mortality.

A concentration-response function from the Harvard Six Cities study was used for sensitivity analyses in U.S. EPA NAAQS reviews to explore the influence of different assumptions on the size of the population impact. The risk estimates (hazard ratios) from the Harvard Six Cities study are among the highest in the literature. The results of this analysis were used to estimate a plausible range of population risk for mortality associated with long-term PM2.5 exposure.

The Harvard Six Cities study followed a randomly selected cohort of 8,111 well-characterized white adults 25 to 74 years of age at enrollment (mean age about 50 years). Participants lived in one of six cities in the eastern and mid-western United States (Portage, WI; Watertown, MA; Kingston-Harriman, TN; St. Louis, MO; Steubenville, OH; Topeka, KS), and were relatively evenly divided among the six study sites. The analysis used the Cox proportional hazards approach; the models were adjusted for smoking, education, and body mass index, and stratified by gender and five-year age groups. The original study followed participants from 1974 to 1989 and found a statistically significant association between PM2.5 and all-cause mortality as well as mortality due to cardiopulmonary causes (Dockery et al., 1993). This study was also extensively

reanalyzed by Krewski et al. (2000) as part of the HEI-coordinated reanalysis effort.

Although the reanalysis (Krewski et al., 2000) confirmed the original findings, the exploratory and sensitivity analyses of the Six Cities Study were less extensive than for the ACS study because the study was considerably smaller and not suitable for some of the analyses undertaken as part of the ACS reanalysis. A subsequent publication from the Six Cities Study extended follow-up through 1998 and reported similar results using exposure assessments for 1974-1989, 1990-1998, and the average of both exposure periods (Laden et al., 2006).

Epidemiological studies exploring the influence of PM2.5 exposure on premature mortality, such as the two described above, use the Cox proportional hazards model, a statistical model widely used in survival analysis (Cox and Oakes, 1984). This model enables researchers to estimate the relationship between an explanatory variable and the risk of an adverse health outcome, such as the influence of the annual-average ambient PM2.5 concentration on mortality. Results are reported in the form of a relative risk, which is the percent change in risk associated with a 10 $\mu g/m^3$ change in annual-average PM2.5 concentration. The relative risk can be used to predict the effect of changes in PM2.5 concentrations on mortality. The prediction equation derived from the relative risk is called the concentration-response (C-R) function. U.S. EPA discusses uncertainties in the epidemiological studies in its Quantitative Health Risk Assessment (U.S. EPA, 2010).[6]

The key studies used by U.S. EPA to assess PM2.5-related mortality are multi-city, which has several advantages over single-city studies. First, multi-city studies use the same study design in each of the cities included in the study, so that city-specific results are readily comparable. Second, when they are estimating a single C-R function based on several cities, multi-city studies also tend to have more statistical power and provide effect estimates with relatively greater precision than single city studies due to larger sample sizes, reducing the uncertainty around the estimated C-R function.

U.S. EPA Quantification of Premature Mortality

The risk assessments U.S. EPA prepares in the air quality standard setting process include estimates of the impacts of current air pollution levels, as well as analyses which explore the health impacts of alternative standards. The U.S. EPA's June 2010 risk assessment includes an analysis of PM2.5 mortality on a national scale, and a case study of 15 urban areas.

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[6] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, section 3.5, page 3-63.

To perform the national scale analysis, U.S. EPA used a standard air quality model (Community Model for Air Quality), monitoring data, and the environmental Benefits Mapping and Analysis Program (BenMAP).[7] Excess mortality was quantified by applying two estimates of all-cause mortality risk. The first is found in the Krewski et al. (2009) PM2.5 mortality extended analysis of the American Cancer Society study. The second, is an estimate of all-cause mortality risk found in the Laden et al. (2006) PM2.5 mortality extended analysis of the Six Cities study. The deaths were estimated down to the lowest measured PM2.5 level in each epidemiological study.

The mortality assessment combines information about PM2.5 air quality levels, population, baseline mortality rates, and PM2.5 related mortality risk factors (coefficients) from health studies. This information is used in the BenMAP program to estimate premature mortality associated with PM2.5. BenMAP aggregates population data to the same resolution (grid cell) used in the air quality model. A single year is selected to match the population data, air quality monitoring data, and emissions inventory data used in the air quality model.

The air quality model was used to estimate the annual mean concentrations at each grid cell, and these data were input to BenMAP which matches PM2.5 levels with population. The modeled PM2.5 levels were derived with a "fusion" technique that merges monitoring data into the modeling system. The details of the national scale assessment are found in Appendix G of the U.S. EPA risk assessment.

Key elements in the U.S. EPA quantification of risk of premature mortality using the BenMAP program are:

- Estimates of PM2.5 air quality
- Concentration-response relationship
- Population data
- Baseline mortality rates

The results of the national scale assessment are shown in Table 2. In U.S. EPA's table the bolded figures indicate the estimate that corresponds with the lowest measured level in the epidemiological study. The bolded estimates in the Krewski et al. (2009) column were calculated using the same risk coefficients as the urban case study. U.S. EPA indicates a greater emphasis is placed on the results calculated using the lowest measured level reported in the epidemiological studies.[8] The estimated total PM2.5-related premature mortality

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[7] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, Appendix G.

[8] "In studies estimating a relationship between mortality and long-term exposure to PM2.5 the lowest measured levels (LMLs) reported in the epidemiological studies were substantially above PRB (policy relevant background). Thus, estimating risk down to PRB would have required

ranges from 63,000 – 80,000 for the two time periods in the Krewski et al. (2009) study to the lowest measured level of 5.8 µg/m^3. For the Laden et al. (2006) study the estimate is 88,000 with a lowest measured level of 10 µg/m^3. The 90% percent confidence intervals are shown in each case. The U.S. EPA national assessment is based on 2005 PM2.5 levels. This report provides a California estimate based on air quality data from the years 2006-2008.

Table 2: U.S. Environmental Protection Agency National Assessment: Estimated PM2.5-related premature mortality associated with incremental air quality differences between 2005 ambient mean PM2.5 levels and lowest measured level from epidemiology studies or policy relevant background (90th percentile confidence interval)[9]

Air Quality Level	Estimates Based on Krewski et al. (2009)		Estimates Based on Laden et al. (2006)
	'79-'83 estimate (90th percentile confidence interval)	*'99-'00 estimate (90th percentile confidence interval)*	*(90th percentile confidence interval)*
10 µg/m^3 (LML for Laden et al., 2006)	26,000 (16,000—36,000)	33,000 (22,000—44,000)	**88,000 (49,000—130,000)**
5.8 µg/m^3 (LML for Krewski et al., 2009)	**63,000 (39,000—87,000)**	**80,000 (54,000—110,000)**	210,000 (120,000—300,000)
Policy-Relevant Background	110,000 (68,000—150,000)	140,000 (94,000—180,000)	360,000 (200,000—500,000)
Bold indicates that the minimum air quality level used to calculate this estimate corresponds to the lowest measured level identified in the epidemiological study			

The U.S. EPA's urban case study focused on 15 urban areas. Two of the selected cities, Los Angeles and Fresno, are in California. These areas were selected according to the following criteria:

- Inclusion in U.S. EPA's previous PM risk assessment
- Availability of mortality rates, air quality data, and epidemiological models in the selected urban areas
- Preference for locations with high PM2.5 concentrations

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substantial extrapolation of the estimated C-R functions below the range of the data on which they were estimated. Therefore, we estimated risk only down to the LML, to avoid introducing additional uncertainty related to this extrapolation into this analysis. To provide consistency for the different C-R functions selected from the long-term exposure studies, and, in particular, to avoid the choice of LML unduly influencing the results of the risk assessment, we selected a single LML - 5.8 µg/m^3 from the later exposure period evaluated in Krewski et al. (2009) – to be used in estimating risks associated with long-term PM2.5 exposures." Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, page 3-3.

[9] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, pg. G6-G7.

- Adequate representation of the spectrum of conditions across the U.S. potentially affecting PM-related risk, including the mix of sources, particle composition and other factors

U.S. EPA quantified premature mortality for the 15 urban areas under several scenarios in order to inform the air quality standard setting process. Appendix E[10] of U.S. EPA risk assessment shows the mortality risk quantified as annual incidence, as well as percent or total incidence, and percent reduction in incidence from current PM2.5 levels. More populated urban areas with high levels of PM2.5 show the largest incidence of premature mortality. Of the 15 urban areas, Los Angeles and New York showed the highest incidence.

The selection of the study used for the concentration-response relationship significantly affects the results of the quantification of premature mortality, as shown by the sensitivity analysis using the Harvard Six City dataset (Laden et al., 2006). The U.S. EPA evaluated all the relevant health studies and concluded, as described in the risk assessment, that the highest quality study from which to obtain a concentration response relationship is the one done by Krewski et al. (2009).[11] The scientific advisory committee peer reviewed the risk assessment, and agreed that U.S. EPA's choice of core study was appropriate and reasonable.[12]

U.S. EPA chose Krewski et al. (2009) for quantifying PM2.5-related mortality from long-term PM2.5 exposure for several reasons. First, the cohort includes both men and women where enrollment was not dependent on underlying health status. It also includes data from cities from across the U.S. PM2.5 exposure was based on monitored data collected over two time periods (1979-1983 and 1999-2000); the effect estimates were presented both for each time period and as an average. The study was validated through extensive reanalysis that demonstrated the results to be robust. Extensive exploratory analysis of potential individual and ecologic covariates was conducted, and the results were adjusted for all covariates that influenced the model fit. Finally, spatial autocorrelation was evaluated and adjusted for in the ecologic covariates.

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[10] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, Appendix E.

[11] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, pg. 3-37 to 3-38.

[12] http://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/BC1ECC5D539EF72385257678006D5754/$File/EPA-CASAC-10-003-unsigned.pdf (first draft)

http://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/BC4F6E77B6385155852577070002F09F/$File/EPA-CASAC-10-008-unsigned.pdf (second draft)

Krewski et al. (2009) published several risk estimates that reflect different degrees of adjustment for confounders. U.S. EPA selected the concentration-response functions that are most thoroughly adjusted for individual and ecologic covariates. The effect estimates from the two exposure periods differ slightly, but the difference is not statistically significant. Because there is no compelling reason to select one exposure period over the other, both were used in making the range of estimates presented in the U.S. EPA PM NAAQS risk assessment.

III. APPLICATION OF U.S. EPA METHODS IN CALIFORNIA

U.S. EPA's quantification methods can be applied at different scales provided the input data are available. The risk assessment included a national scale analysis and individual analyses of 15 urban areas. The method can be applied on a statewide basis to quantify the premature mortality associated with PM2.5 in California, as well as to estimate the number of premature deaths that would be avoided by attaining the PM2.5 NAAQS. Table 3 compares the elements of the mortality calculation used by U.S. EPA and ARB.

Calculation of the current statewide mortality estimates involves several steps:

- Estimate exposure at the census tract level using measured air quality data and population
- Estimate incidence of premature death by applying concentration functions to estimated exposures and baseline mortality rates
- Aggregate results to air basin and statewide totals

Table 3: Comparison of U.S. EPA and ARB Mortality Calculation Method

Elements	U.S. EPA	ARB
Source of Concentration-Response functions	Krewski et al., 2009	Krewski et al., 2009
Threshold	5.8 μg/m^3	5.8 μg/m^3
Model	BenMAP	BenMAP
PM2.5 exposure	Air quality modeling and measured data	All measured data

Premature mortality associated with long-term exposure to PM2.5 was estimated using the same concentration response functions from Krewski et al. (2009) used by U.S. EPA in the risk assessment. Relative risk is expressed as the percent change in the baseline mortality rate associated with a 10 μg/m^3 change in ambient PM2.5 concentration.

Calculation of the number of deaths associated with PM2.5 exposure also requires estimation of population exposure to PM2.5, which is estimated from monitored or modeled concentrations of PM2.5. ARB and U.S. EPA use the software program BenMAP, a GIS-based program developed by U.S. EPA, which uses input exposure data and concentration-response functions to calculate estimated mortality.

Exposure Assessment

For its national scale analysis U.S. EPA used an exposure assessment approach that combined ambient data with modeled PM2.5 concentrations, which is a so-called "fusion" approach. To some extent this is necessitated by the large areas of the country where PM2.5 monitoring is sparse, which introduces uncertainties in the exposure assessment.

In contrast, California has the most extensive PM2.5 monitoring network in the nation, comprising approximately 100 monitors that collect PM2.5 mass data using federally approved methods. For the present analysis, air quality data from California's PM2.5 monitoring network for the years 2006, 2007, and 2008 were used to estimate population exposure using spatial interpolation, which is a method of estimating concentrations based on nearby monitors. PM2.5 monitors are not evenly distributed throughout the state, but are mainly located in heavily populated areas that have the highest PM2.5 levels. Approximately half the population of California lives in a zip code that is within 6 miles of a PM2.5 monitor. For example, during the 2006-2008 period there were 19 monitoring sites operating in the South Coast Air Basin.

Even with an extensive air quality monitoring network, the quantification method requires use of a technique for applying the monitoring results across a geographic area. Using a method called spatial interpolation, population exposure in areas between monitors can be estimated. ARB uses a standard spatial interpolation method known as inverse distance-squared weighting **(**Shepard, 1968; Goodin and McRae, 1979). This method yields reasonable accuracy in estimating pollutant concentrations near monitoring stations, although when distance from the monitoring station increases the uncertainty in the interpolated concentration also increases. This method gives more accurate estimates of concentration in areas with a large number of monitors with good spatial coverage as is the case in populated areas in California.

Use of Concentration-Response Function

To calculate PM2.5-related deaths, the ARB employs the same method used by the U.S. EPA. The method links changes in PM2.5 concentration with predicted changes in the number of premature deaths. The method has 4 elements: 1) a concentration-response (C-R) function (explained below), 2) a predicted change

in PM2.5 concentration, 3) death rates for people older than 30 years of age, and 4) number of people in affected counties from the U.S. Census Bureau.

Health studies show that when the PM2.5 concentration decreases so does the death rate. The C-R function describes how much the death rate changes when the PM2.5 concentration changes. The concentration-response functions used by U.S. EPA are listed in Table 4. They relate the change in the baseline mortality rate for every decrease of 1 $\mu g/m^3$ of PM2.5. Using the C-R function and knowing the death rate, the change in PM2.5 concentration, and the number of people over 30, the U.S. EPA is able to make predictions about health outcomes when PM2.5 improves.

Table 4: Concentration-response functions per $\mu g/m^3$ used in U.S. EPA Risk Assessment (Krewski et al., 2009)

Endpoint	Lower Bound	Coefficient	Upper Bound
First exposure period			
Mortality, all-cause	0.00276	0.00431	0.00583
Mortality, cardiopulmonary	0.00677	0.00898	0.01115
Mortality, ischemic heart disease	0.01363	0.01689	0.02005
Mortality, lung cancer	0.00325	0.00880	0.01432
Second exposure period			
Mortality, all-cause	0.00354	0.00554	0.00760
Mortality, cardiopulmonary	0.01007	0.01293	0.01587
Mortality, ischemic heart disease	0.01748	0.02167	0.02585
Mortality, lung cancer	0.00554	0.01293	0.02029

Premature Deaths in California Associated with Current PM2.5 Levels

Mortality estimates are calculated in three ways which reflect the nature and scope of epidemiological studies: cardiopulmonary, ischemic heart disease, and all-cause mortality. PM2.5 exposure has been most closely associated with cardiopulmonary deaths, which are also the most frequent causes of death in the U.S. In addition, the cardiopulmonary deaths represent an endpoint judged to be causally related to PM2.5 exposure[13]. The greater scientific certainty for this effect, along with the greater specificity of the endpoint, leads to an effect estimate for cardiopulmonary deaths that is both higher and more precise than that for all-cause mortality. Cardiopulmonary mortality and all-cause mortality are estimated separately, and the estimates represent independent measures of the effect of PM2.5 exposure.

[13] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, pages 3-20 to 3-22.

The estimates for cardiopulmonary mortality are generally larger, although not distinguishable considering the overlapping confidence intervals, than for all-cause mortality, particularly in analyses based on the second exposure period in Krewski et al. (2009), for several reasons. For example, the incidence data for all-cause mortality includes categories which would not plausibly be linked to PM2.5 exposure. Deaths due to such causes as complications of surgery, gastrointestinal diseases, homicides, and accidents are included in all-cause mortality, although it is unlikely that PM2.5 exposure has any influence on these deaths.

Including these unrelated causes of death has the effect of "diluting" the effect estimate for all-cause mortality related to PM2.5 exposure, as can be seen in the results in Appendix B of this report. This effect is particularly evident in the results using the second period exposure data, possibly related to the influence of the larger number of people in the second time period analyses (about 500,000 people in the second time period versus about 300,000 in the first time period), which would tend to increase the precision and robustness of the estimates from the second exposure period compared to the first.

Another factor that could influence these results is changes in the criteria for coding cause of death. The standards for coding specific causes of death have changed and become better defined over the period of the study. Because of this, the more precise categories into which more recent deaths are attributed would tend to increase the robustness and precision of estimates of the effect of PM2.5 exposure on these specific causes of death.

The third type of mortality found by U.S. EPA to be causally linked to long-term PM2.5 exposures is ischemic heart disease, which can lead to a heart attack due to inadequate blood flow to the heart. It is a subset of cardiopulmonary deaths, and represents a large fraction of cardiopulmonary deaths. Cardiopulmonary disease and ischemic heart disease are subsets of all-cause mortality, and ischemic heart disease is a subset of cardiopulmonary disease. Consequently these numbers should not be added together, and the results are each shown in separate tables. The three estimates presented are those associated with exposure down to 5.8 $\mu g/m^3$, which is the threshold for quantification used in U.S. EPA's risk assessment.

Estimates using a calculation threshold of 5.8 $\mu g/m^3$ assume that there is an effect down to that level of exposure. The U.S. EPA risk assessment discusses the issue of threshold of effect.[14] This level was chosen as the calculation threshold because it is the lowest annual-average PM2.5 concentration reported by Krewski et al. (2009). The tables show a mean estimate and a low and a high

[14] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, page 3-1 to 3-3. See also footnote 8.

estimate that represent the upper and lower 95% confidence intervals. The mortality estimates in Tables 5 through 10 are based on monitored PM2.5 data from years 2006 through 2008. The estimates presented reflect use of the C-R functions derived from the second exposure period (1999-2000) of Krewski et al. (2009). Estimates based the first exposure period are in Appendix B of this report. The baseline rates used for the analysis were supplied by the California Department of Public Health (CDPH, 2010).

The estimates of the number of premature deaths that would be avoided by reducing PM2.5 levels to the calculation threshold of 5.8 μg/m^3 (Tables 5-7) are larger than the estimated number of premature deaths avoided by reducing PM2.5 levels to the annual-average NAAQS of 15 μg/m^3 (Tables 8-10). This is because reduction to the calculation threshold represents a larger reduction in PM2.5 concentration than reduction to the level of the NAAQS. The larger the reduction in concentration, the greater the reduction in premature deaths predicted by the C-R function.

Table 5: Cardiopulmonary – Current Estimates of Annual Cardiopulmonary Deaths in California Associated with PM2.5 Exposure

Scenario	Low	Mean	High
Current Air Quality	7,300	9,200	11,000

*Presented here is the estimated mean (Mean) and the 95% confidence interval (Low, High). Air quality data from years 2006 to 2008. Health impacts were assessed only in areas with ambient PM2.5 levels greater than 5.8 μg/m^3. Population data from the 2000 U.S. Census were extrapolated to each corresponding year in BenMAP. The results are averages of annual impacts.

Table 6: Ischemic Heart Disease – Current Estimates of Annual Ischemic Heart Disease Deaths in California Associated with PM2.5 Exposure

Scenario	Low	Mean	High
Current Air Quality	5,500	6,800	7,900

*See footnote to Table 5

Table 7: All-Cause – Current Estimates of Annual All-Cause Deaths in California Associated with PM2.5 Exposure

Scenario	Low	Mean	High
Current Air Quality	5,400	8,400	11,000

*See footnote to Table 5

Most of the estimated premature deaths are in the South Coast Air Basin in southern California. This is because PM2.5 concentrations are high there, and a large portion of California's population lives there. The region with the next largest number of premature deaths is the San Joaquin Valley, with the remainder distributed around the state. No premature deaths were estimated in census tracts where the annual-average PM2.5 concentration was below the threshold of 5.8 μg/m^3. Premature mortality was estimated by census tract for all of California, and then aggregated into estimates at the county, air basin and

statewide levels. Estimates of the number of deaths by air basin are presented in Appendix B.

Deaths Avoided in California with PM2.5 NAAQS Compliance

To estimate the benefits of achieving the federal air quality standards requires calculating the difference between current PM2.5 levels and the level at which the standard is met, in this case an annual average of 15 μg/m^3. For its nationwide analysis, the U.S. EPA uses a calculation approach called "proportional rollback" to compute such estimates. The U.S. EPA risk assessment describes the proportional rollback calculation.[15] A rollback calculation was applied to California monitoring data to estimate the statewide benefits of achieving the federal PM2.5 annual air quality standard shown below.

The estimated number of premature deaths avoided by achieving the current PM2.5 NAAQS is shown in Tables 8-10. Table 8 shows the reduction in premature deaths due to cardiopulmonary disease. Table 9 shows the reduction in premature deaths due to ischemic heart disease, a subset of cardiopulmonary disease. Table 10 shows the reduction in premature deaths from all causes. Although cardiopulmonary mortality is a subset of all-cause mortality, the mean estimate for cardiopulmonary mortality is higher than all-cause deaths. While counterintuitive, this is not an error. The two numbers are independently estimated, with statistical uncertainty that overlap between the ranges of the two numbers.

Table 8: Cardiopulmonary – Annual Cardiopulmonary Deaths Avoided in California by Attainment of the Annual-Average Federal PM2.5 NAAQS

Scenario	Low	Mean	High
National standard (15 μg/m^3)	2,100	2,700	3,300

*See footnote to Table 5.

Table 9: Ischemic Heart Disease – Annual Ischemic Heart Disease Deaths Avoided in California by Attainment of the Annual-Average Federal PM2.5 NAAQS

Scenario	Low	Mean	High
National standard (15 μg/m^3)	1,700	2,100	2,500

*See footnote to Table 5.

[15] Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf, page 3-18.

Table 10: All-Cause – Annual All-Cause Deaths in California Avoided by Attainment of the Annual-Average Federal PM2.5 NAAQS

Scenario	Low	Mean	High
National standard (15 µg/m^3)	1,500	2,400	3,300

*See footnote to Table 5.

IV. CLEAN AIR ACT BENEFITS ANALYSIS

U.S. EPA Regulatory Impacts Analysis

In the 1997 report, "Benefits and Costs of the Clean Air Act, Retrospective Analysis 1970 – 1990," U.S. EPA used the first ACS study publication to estimate mortality related to long-term exposure to PM2.5 (Pope et al., 1995), as well as other health effects. This was done as part of a report required by the Clean Air Act (Section 812). The Clean Air Act requires the U.S. EPA Administrator, in consultation with the Secretaries of Commerce and Labor and the Council on Clean Air Compliance Analysis (CCACA), which operates through the U.S. EPA Science Advisory Board, to conduct a "comprehensive analysis of the impact of this Act on the public health, economy, and environment of the United States."

In 1999, U.S. EPA published the first prospective analysis of the benefits and costs of the Clean Air Act (U.S. EPA, 1999). This analysis continued to rely on the relative risk in Pope et al. (1995) to assess premature mortality associated with improvements in ambient PM2.5 concentrations, although the relative risk from Dockery et al. (1993) was included for sensitivity analyses. These regulatory analysis reports include estimates for a variety of other health effects based on single city studies that were conducted prior to 1997, and were reviewed during the 1997 PM NAAQS process.

In U.S. EPA's May 2004 regulatory impact analysis (RIA) for the Clean Air Non-Road Diesel Rule, the agency updated its methodology by using an update to the ACS study (Pope et al., 2002) to estimate premature mortality associated with long-term exposure to PM2.5, although U.S. EPA continued to use the same studies first applied in the retrospective analysis (U.S. EPA, 1997) for other health effects.

U.S. EPA is currently updating the Section 812 report, with a draft of the report reviewed by the CCACA in May 2010. The goal of this process is to bring the assessment, and the health effects included, into greater alignment with the NAAQS process. U.S. EPA staff indicated that some health effects currently used will be dropped, and others may be added. The RIA associated with the ongoing PM NAAQS review is scheduled for release in early 2011.

To obtain quantitative estimates of regulatory control benefits, the U.S. EPA developed a methodology which may be used instead of a full modeling analysis. The methodology is described in detail in Fann et al. (2009). Fann et al. (2009) estimate pollutant concentrations for nine urban areas (including one in

California, the San Joaquin Valley) using the Community Multiscale Air Quality Reactive Scavenging Model (CMAQ RSM) air quality model. Table 11 lists the emissions grouped by pollutant and source.

The model is run for a baseline scenario and for control scenarios. Health impacts are estimated and monetized with BenMAP, using mortality C-R functions from Pope et al. (2002). The monetized benefits are then divided by tons of emissions. The result is a matrix of dollars-per-ton benefit factors, one factor for each combination of urban area and source category. These benefit factors can then be multiplied by emission reductions achieved by a control measure to estimate health benefits.

Table 11: U.S. EPA Pollutant and Source Categories for Estimating Control Benefits (Fann et al., 2009)

Carbon	Area sources Mobile sources Electric utilities and industrial point sources
SO_X	Area sources Electric utilities Other facilities
VOC	Electric utilities and industrial point sources
Ammonium	Area sources Mobile sources
NO_X	Mobile sources Electric utilities Other facilities

Analysis of ARB Rules

Similar to U.S. EPA, ARB also estimates the health benefits of regulatory controls to reduce PM2.5. It is similar in concept to the simplified methodology developed by the U.S. EPA described above.

Across the range of ambient PM2.5 concentrations encountered in California, modeled changes in premature mortality are approximately proportional to changes in ambient pollutant concentrations. For primary pollutants such as directly emitted PM2.5, changes in ambient concentrations are approximately proportional to changes in emissions. Therefore, premature mortality is approximately proportional to emissions, and can be estimated by multiplying emissions by a scaling factor. This factor is derived by calculating the number of premature deaths associated with PM2.5 in an air basin using BenMAP, and dividing by the emissions of PM2.5.

V. CONCLUSION

Application of U.S. EPA's methodology for calculating premature mortality from long-term exposure to PM2.5 provides estimates of current PM2.5 impacts and estimates of the benefits of meeting the national ambient air quality standard. The calculations were done using the same concentration-response function and calculation threshold of 5.8 $\mu g/m^3$ that U.S. EPA used for estimating premature deaths associated with PM2.5 exposure, as described in U.S. EPA's PM NAAQS risk assessment (U.S. EPA, 2010), and part of the most recent NAAQS review.

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Appendix A: Map of California Air Basins and PM2.5 Monitoring Network

Appendix B: Annual Mortality by Air Basin

Federal NAAQS Standard, First Exposure Period of Krewski et al. (2009)

Annual premature deaths avoided by attainment of the federal PM2.5 standard of 15 μg/m^3. The numbers were calculated using rollback to 15 μg/m^3 and the first exposure period from Krewski et al. (2009). Air quality data is an average of the years 2006 to 2008. Health impacts were assessed only in areas with ambient PM2.5 levels greater than 5.8 μg/m^3. Only air basins with 1 or more estimated deaths are included.

All-Cause Mortality

Air Basin	Low	Mean	High
Mojave Desert	10	15	20
Mountain Counties	1	2	2
Sacramento Valley	1	1	1
San Diego County	0	1	1
San Francisco	1	2	3
San Joaquin Valley	320	500	670
South Central Coast	1	1	2
South Coast	860	1,300	1,800
Statewide Total	1,200	1,900	2,500

Ischemic Heart Disease Mortality

Air Basin	Low	Mean	High
Mojave Desert	11	13	15
Mountain Counties	1	1	1
Sacramento Valley	1	1	1
San Diego County	0	0	1
San Francisco	1	1	1
San Joaquin Valley	340	410	490
South Central Coast	1	1	1
South Coast	1,000	1,200	1,400
Statewide Total	1,300	1,700	2,000

Cardiopulmonary Mortality

Air Basin	Low	Mean	High
Mojave Desert	11	15	19
Mountain Counties	1	2	2
Sacramento Valley	1	1	1
San Diego County	0	1	1
San Francisco	1	2	2
San Joaquin Valley	380	500	620
South Central Coast	1	1	1
South Coast	1,000	1,400	1,700
Statewide Total	1,400	1,900	2,300

Federal NAAQS Standard, Second Exposure Period of Krewski et al. (2009)

Annual premature deaths avoided by attainment of the federal PM2.5 standard of 15 µg/m^3. The numbers were calculated using rollback to 15 µg/m^3 and the second exposure period from Krewski et al. (2009). Air quality data is an average of the years 2006 to 2008. Health impacts were assessed only in areas with ambient PM2.5 levels greater than 5.8 µg/m^3. Only air basins with 1 or more estimated deaths are included.

All-Cause Mortality

Air Basin	Low	Mean	High
Mojave Desert	12	19	26
Mountain Counties	1	2	3
Sacramento Valley	1	1	2
San Diego County	1	1	1
San Francisco	2	2	3
San Joaquin Valley	410	640	870
South Central Coast	1	2	2
South Coast	1,100	1,700	2,400
Statewide Total	1,500	2,400	3,300

Ischemic Heart Disease Mortality

Air Basin	Low	Mean	High
Mojave Desert	14	17	20
Mountain Counties	1	2	2
Sacramento Valley	1	1	1
San Diego County	0	1	1
San Francisco	1	1	2
San Joaquin Valley	430	520	620
South Central Coast	1	1	1
South Coast	1,300	1,600	1,800
Statewide Total	1,700	2,100	2,500

Cardiopulmonary Mortality

Air Basin	Low	Mean	High
Mojave Desert	17	22	26
Mountain Counties	2	3	3
Sacramento Valley	1	1	2
San Diego County	1	1	1
San Francisco	2	2	3
San Joaquin Valley	560	720	870
South Central Coast	1	2	2
South Coast	1,500	2,000	2,400
Statewide Total	2,100	2,700	3,300

Current Air Quality, First Exposure Period of Krewski et al. (2009)

Annual premature deaths associated with exposure to ambient PM2.5. The numbers were calculated using rollback to 5.8 μg/m^3 and the first exposure period from Krewski et al. (2009). Air quality data is an average of the years 2006 to 2008. Health impacts were assessed only in areas with ambient PM2.5 levels greater than 5.8 μg/m^3.

All-Cause Mortality

Air Basin	Low	Mean	High
Great Basin Valley	1	1	1
Lake County	1	2	3
Lake Tahoe	1	1	2
Mojave Desert	57	89	120
Mountain Counties	31	49	66
North Central Coast	13	20	26
North Coast	12	18	24
Northeast Plateau	9	13	18
Sacramento Valley	300	460	620
Salton Sea	37	58	78
San Diego County	340	530	710
San Francisco	410	630	850
San Joaquin Valley	710	1,100	1,500
South Central Coast	94	150	200
South Coast	2,200	3,500	4,700
Statewide Total	4,300	6,600	8,900

Ischemic Heart Disease Mortality

Air Basin	Low	Mean	High
Great Basin Valley	0	1	1
Lake County	1	1	2
Lake Tahoe	1	1	1
Mojave Desert	62	76	89
Mountain Counties	26	32	38
North Central Coast	10	13	15
North Coast	9	11	13
Northeast Plateau	7	8	10
Sacramento Valley	260	330	380
Salton Sea	40	49	58
San Diego County	300	370	440
San Francisco	340	420	500
San Joaquin Valley	720	880	1,000
South Central Coast	94	120	140
South Coast	2,500	3,100	3,600
Statewide Total	4,400	5,400	6,300

Cardiopulmonary Mortality

Air Basin	Low	Mean	High
Great Basin Valley	0	1	1
Lake County	2	2	3
Lake Tahoe	1	1	2
Mojave Desert	68	90	110
Mountain Counties	35	46	57
North Central Coast	14	18	22
North Coast	12	16	20
Northeast Plateau	9	12	15
Sacramento Valley	340	450	560
Salton Sea	44	58	72
San Diego County	370	490	610
San Francisco	440	580	720
San Joaquin Valley	840	1,100	1,300
South Central Coast	110	140	180
South Coast	2,700	3,500	4,300
Statewide Total	5,000	6,500	8,000

Current Air Quality, Second Exposure Period of Krewski et al. (2009)

Annual premature deaths associated with exposure to ambient PM2.5. The numbers were calculated using rollback to 5.8 µg/m^3 and the second exposure period from Krewski et al. (2009). Air quality data is an average of the years 2006 to 2008. Health impacts were assessed only in areas with ambient PM2.5 levels greater than 5.8 µg/m^3.

All-Cause Mortality

Air Basin	Low	Mean	High
Great Basin Valley	1	1	1
Lake County	2	3	4
Lake Tahoe	1	2	3
Mojave Desert	73	110	160
Mountain Counties	40	63	86
North Central Coast	16	25	34
North Coast	15	23	32
Northeast Plateau	11	17	23
Sacramento Valley	380	590	800
Salton Sea	47	74	100
San Diego County	430	680	920
San Francisco	520	810	1,100
San Joaquin Valley	910	1,400	1,900
South Central Coast	120	190	260
South Coast	2,900	4,400	6,000
Statewide Total	5,400	8,400	11,000

Ischemic Heart Disease Mortality

Air Basin	Low	Mean	High
Great Basin Valley	1	1	1
Lake County	2	2	2
Lake Tahoe	1	1	1
Mojave Desert	79	96	110
Mountain Counties	33	41	48
North Central Coast	13	16	20
North Coast	11	14	16
Northeast Plateau	9	11	12
Sacramento Valley	340	410	480
Salton Sea	51	62	74
San Diego County	390	470	560
San Francisco	440	540	640
San Joaquin Valley	910	1,100	1,300
South Central Coast	120	150	170
South Coast	3,200	3,800	4,500
Statewide Total	5,500	6,800	7,900

Cardiopulmonary Mortality

Air Basin	Low	Mean	High
Great Basin Valley	1	1	1
Lake County	2	3	4
Lake Tahoe	1	2	2
Mojave Desert	100	130	160
Mountain Counties	51	65	80
North Central Coast	20	26	32
North Coast	18	24	29
Northeast Plateau	14	17	21
Sacramento Valley	510	640	780
Salton Sea	65	83	100
San Diego County	550	700	850
San Francisco	650	840	1,000
San Joaquin Valley	1,200	1,500	1,900
South Central Coast	160	200	250
South Coast	3,900	4,900	6,000
Statewide Total	7,300	9,200	11,000