Multi-Pollutant Evaluation Method
Technical Document
2016 Update

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Table of Contents

List of Tables

List of Figures

Executive Summary

The Bay Area Air Quality Management District (District) is preparing the Bay Area 2016 Clean Air Plan (CAP) to update its previous 2010 CAP, as required by the California Health & Safety Code. The 2016 CAP will serve as a multi-pollutant plan to protect public health and the climate. The CAP will propose a control strategy designed to reduce ambient concentrations of four types of pollutants: ground-level ozone, particulate matter, air toxics, and greenhouse gases (GHGs). These pollutants differ in fundamental ways in terms of their emission sources, atmospheric formation, chemical composition and health effects.

This document describes a multi-pollutant evaluation method (MPEM) that the District developed to help analyze and compare potential emission control measures on a multi-pollutant basis for the 2016 CAP. Air pollution imposes a range of negative health impacts and economic and social costs on the Bay Area. In developing the CAP control strategy, District staff used the MPEM to help analyze how potential control measures would reduce these negative impacts on public health and the climate (i.e., anticipated impacts from global warming) and to estimate the associated cost savings of the avoided negative impacts.

The MPEM has been used to:

- Estimate how reductions of each pollutant for a given control measure will affect ambient concentrations, population exposures, and health outcomes related to that pollutant;
- Monetize the value of total health benefits of reductions in PM2.5, ozone and certain carcinogens, and the social value of greenhouse gas reductions that would be reduced by each potential control measure; and
- Evaluate and compare the estimated benefit of potential control measures based upon the value of each measure in reducing health costs from air pollutants and environmental/social impacts related to climate change.

MPEM Foundation

The MPEM is based upon well-established studies and methods that have been used by the EPA, ARB, and other entities to quantify and monetize the health benefits associated with:

- The Clean Air Act (US EPA – 1999)
- Attainment of the ozone NAAQS in California (Ostro et al. – 2005)
- ARB Goods Movement Plan, on-road truck rule, etc. (ARB – 2006)
- Attainment of NAAQS in South Coast & San Joaquin Valley (Hall et al. – 2008)
- South Coast 2007 AQMP (South Coast AQMD)

The MPEM also draws heavily from the US EPA BENMAP program, as well as Office of Environmental Health Hazard Assessment (OEHHA) documents, for coefficients, concentration-response (C-R) functions, and uncertainty estimates.

The MPEM builds upon established precedents embedded in these studies, but goes beyond them by:

- Using computer models to estimate how ambient concentrations of each pollutant are affected by changes in emissions of the pollutants or their precursors; and
- Estimating a value for greenhouse gas emission reductions, expressed in cost ($/ton) of CO2-equivalent reduced.

Caveats and Constraints

The multi-pollutant evaluation methodology is meant to serve as a tool to help guide air quality planning and policy. Inevitably, judgment has been exercised in developing the method, balancing completeness against practicality, and being health-protective against the uncertainty in health effects. Key choices in developing this method include:

- which pollutants to include (Section 1.3.1)
- which health endpoints to include (Sections 1.3.2 & 4.6)
- where to set health effects thresholds (Section 1.3.4)

The MPEM does not include all air pollutants. To avoid undue complexity, we limited the pollutants in the methodology to those that EPA analysis of health studies suggests cause the greatest harm. Among the six criteria pollutants, only ozone and PM are included in the MPEM; these are the two criteria pollutants for which the Bay Area does not yet attain all standards. The Bay Area attains all current standards for the other criteria pollutants (CO, SO2, NO2, and lead). It should be noted, however, that for all the criteria pollutants, there may still be health effects at ambient concentrations even below the current standards.

Toxic air contaminants are a separate category of pollutants. Although the California Air Resources Board has identified nearly 200 toxic air contaminants, in the MPEM we focus on five toxic compounds that together account for over 90% of the estimated cancer risk from air toxics in the Bay Area. Likewise, there are dozens of greenhouse gases that contribute to global warming, but we have elected to focus on the "Kyoto Six" GHGs that have been identified by the Intergovernmental Panel on Climate Change (IPCC) as the major culprits in global warming. The "Kyoto Six" GHGs include carbon dioxide, methane, nitrous oxide, hydrofluorocarbons, perfluorocarbons, and sulfur hexafluoride.

District staff believe that the MPEM captures most of the key health effects from Bay Area air pollution. However, it is important to note that some health effects are not included in the MPEM, either because the link between the pollutant and the health effect is not yet clearly established or because we lack the data to complete each of the five steps in the methodology described below. Furthermore, even for the health effects that are included, the per-incidence cost estimates may not fully capture all costs associated with a given illness or impact. Likewise, in the case of greenhouse gases (Section 5.3), we suspect that our estimated value for one ton of greenhouse gas reduced (CO2-e) does not fully capture all potential impacts and costs related to climate change and global warming.

In developing the methodology, District staff grappled with many technical issues that are described in the body of this document. Key simplifying assumptions include the following:

- The emissions reductions for each control measure will be geographically distributed on the same basis as the distribution of emissions of each pollutant in the District's emissions inventory. For example, if we estimate that a control measure would reduce one ton of NOx, we then distribute the NOx emission reductions across Bay Area grid cells in the same proportion as the overall NOx inventory is distributed across those grid cells. (Section 1.5)
- For purposes of estimating population exposure (Step 3 below), full-time (24/7) "backyard" exposure is assumed, even though we realize that people do not spend all of their time at home and in their yards. (Section 3.2)

The Five Key Steps

Although the MPEM is necessarily complex, the basic concept is straightforward. The methodology involves several stages of calculations for each proposed control measure[1]. The steps are:

Step 1. Emissions: We estimate how much a given control measure would reduce (or increase) emissions of each of the pollutants.

Step 2. Concentrations: We estimate how a change in emissions of each pollutant would affect its ambient concentrations and other pollutants related to it. For ozone, PM, and air toxics, we employ photochemical modeling results to calculate pollutant response at the level of each 4 km by 4 km grid square. (Section 2)

Step 3. Population Exposure: We estimate how a change in ambient concentrations would affect the exposure of Bay Area residents to each pollutant, again at the grid square level. (Section 3)

Step 4. Health Impacts: We estimate how a reduction in population exposure would impact various health endpoints, projecting changes in the incidence of endpoints such as asthma emergency room visits, lower respiratory symptoms, and deaths (premature mortality). (Section 4)

Step 5. Health/Social Benefits: We monetize the benefits (i.e. avoided costs) of each control measure by estimating the cost of the health and climate impacts from each pollutant. For each health endpoint, the change in the number of incidents is multiplied by an estimate of the per-incident social cost. For greenhouse gases (GHGs), the change in tons of GHG emissions is multiplied by the estimated social cost per ton of GHGs, expressed in terms of CO_2-equivalent. (Section 5)

The output of the MPEM (Steps 1-5) is an estimated dollar value of the health and social benefits of each potential control measure, based upon the decrease (or increase) in each pollutant.

[1] For ozone, PM2.5, and air toxics, we employ Steps 1 through 5. For greenhouse gases, only Steps 1 and 5 are applied. For discussion of how we consider greenhouse gases for purposes of this methodology, see Section 5.3.

Applications

For purposes of the 2016 CAP, District staff has used the multi-pollutant evaluation methodology to estimate the aggregate value of the health and climate protection benefits of each potential control measure. The MPEM can be particularly useful in helping to evaluate potential trade-offs; i.e., a situation where a control measure may reduce one pollutant, but increase a different pollutant. In addition, District staff used the MPEM to:

- Estimate the total cost of health impacts and monetary costs associated with current emission levels and ambient concentrations;
- Estimate the aggregate benefit of the overall emission reductions for the proposed 2016 CAP control strategy as a whole; and
- Backcast to estimate the health impacts and monetary costs associated with emission levels and ambient concentrations in years past.

Probability Analysis

Uncertainty is inherent in the MPEM. We consider the range of the uncertainty by means of a probability analysis which is described in Fairley (2010). The probability analysis estimates the degree of uncertainty in the assumptions and computations related to each step in the method, and then calculates an overall probability distribution for the results of the methodology as applied to each control measure. The probability analysis enables us to determine whether the potential benefits of one control measure differ significantly from another.

2016 Update

The MPEM used for the 2016 CAP has been updated in several respects. One key update is to incorporate new data in Stage 1, where we estimate how pollutant concentrations change as a function of changes in pollutant emissions. This involves using the results of a 3-D gridded photochemical model (See Section 2 for details). For the previous CAP, the photochemical model was run only for certain times of year, requiring an extrapolation to annual average pollutant concentrations. For this CAP, the model was run for a representative set of days during the year, making extrapolation unnecessary.

Otherwise, the data going into the MPEM were updated to the most recent available: 2016 population projections from the California Department of Finance, 2011-2013 hospital admissions data from the California Department of Public Health, 2011-2013 mortality data, inflation-adjusted valuation data, and updated concentration-response data from BenMAP (US EPA 2012).

Conclusion

The multi-pollutant evaluation methodology summarized above, and described in detail in the body of this document, is a tool developed by Air District staff, based on existing data and studies, to analyze control measures on a multi-pollutant basis. The results of the MPEM

analysis were one of the factors considered by District staff in developing the control strategy proposed in the 2016 CAP.

The MPEM makes use of the tools and technical data currently *available*. In developing the MPEM, we have tried to identify data gaps and technical gaps that should be addressed to improve this methodology for future planning cycles, as discussed in Section 6.

Multi-Pollutant Evaluation Method

1. Introduction

The Bay Area Air Quality Management District (District) is preparing a 2016 Clean Air Plan (CAP) that incorporates an integrated plan to reduce multiple air pollutants. This Technical Document describes the multi-pollutant evaluation methodology (MPEM) developed by Air District staff to help analyze and compare the benefits of potential emission control measures on a multi-pollutant basis.

The MPEM is based upon well-established studies and methods that have been used by the EPA, ARB, and other entities to quantify and monetize the health benefits associated with:

- The Clean Air Act (US EPA – 1999)
- Attainment of the ozone NAAQS in California (Ostro et al. – 2005)
- ARB Goods Movement Plan, on-road truck rule, etc. (ARB – 2006)
- Attainment of NAAQS in South Coast & San Joaquin Valley (Hall et al. – 2008)
- South Coast 2007 AQMP (South Coast AQMD)

The MPEM also draws heavily from the US EPA BENMAP program, as well as OEHHA documents, for coefficients, concentration-response (C-R) functions, and uncertainty estimates.

The MPEM has been used to:

- Estimate how reductions of each pollutant for a given control measure will affect ambient concentrations, population exposures, and health outcomes related to that pollutant
- Monetize the value of total health benefits of reductions in PM2.5, ozone and certain carcinogens, and the social value of greenhouse gas reductions that would be associated with each potential control measure; and
- Evaluate and compare the estimated benefit of potential control measures based upon the value of each measure in reducing health costs from air pollutants and environmental/social impacts related to climate change.

A control measure can affect the emissions of many different air pollutants. However, for the purpose of this study, we considered only the following pollutants:

- Ozone and its precursors, VOCs and NOx
- $PM_{2.5}$ - both primary $PM_{2.5}$ as well as precursors of secondary $PM_{2.5}$ (NOx, SO_2, NH_3)
- Air toxics
- Greenhouse gases (GHGs)[2]

District staff developed the MPEM to assess the impact of potential control measures on the air pollutants listed above, and to evaluate the overall impact of proposed control measures by totaling the estimated health and climate protection benefits. Assessing the impact of

[2] Greenhouse gases are included and evaluated in the methodology on the basis of the overall predicted social and economic impacts of global warming. Direct health effects account for only a small portion of the total estimated cost of global warming impacts.

individual measures provides an opportunity to compare different control measures to one another, as well as to compare benefits versus costs for individual control measures and for the control strategy as a whole.

Although the basic evaluation concept is relatively straightforward, the implementation is complex, requiring a number of assumptions as explained below. We adopted several steps of calculations for simplicity. For ozone, $PM_{2.5}$, and air toxics, we employed steps 1 through 5 below; for greenhouse gases, steps 1 and 5 only. The steps are:

Step 1. Emissions: Estimate how much a given control measure changes emissions of each of the pollutants.

Step 2. Concentrations: Estimate how a change in emissions in each pollutant affects its ambient concentrations and other pollutants related to it.

Step 3. Population Exposure: Estimate how a change in ambient concentrations affects the exposure of Bay Area residents to each pollutant.

Step 4. Health Impacts: Evaluate pollutants based upon their impact on various health effects, estimating changes in the incidence of effects such as asthma emergency room visits, lower respiratory symptoms, and deaths.

Step 5. Health and Climate Benefits: For each health endpoint, multiply the change in the number of incidents of the health endpoint by an estimate of the per-incident social cost. For GHGs, multiply the change in tons of GHG emissions by the estimated social cost per ton of GHGs expressed in terms of CO_2-equivalent.

The result of these steps is an estimated dollar value for the health and climate protection benefits from the changes in emissions due to each control measure.

1.0 Peer Review and Process to Develop the MPEM

In May 2009, the District sent a preliminary draft version of the Multi-Pollutant Evaluation Method (MPEM) Technical Document to leading experts in the analysis of public health impacts of air pollution, including Dr. Jane Hall of Cal State Fullerton, Donald McCubbin of UC Davis, Dr. Bart Ostro of the Office of Environmental Health Hazard Assessment (OEHHA), as well as Dr. Robert Harley of UC Berkeley, an expert on air pollution chemistry and dynamics. Written comments were provided by reviewers Hall, McCubbin, and Harley. Reviewer comments and District staff responses are summarized in the *Peer Review of Draft Multi-Pollutant Evaluation Method* document available on the District website at www.baaqmd.gov/Divisions/Planning-and-Research/Plans/Clean-Air-Plans/Resources-and-Technical-Docs.aspx.

The District issued the Draft MPEM Technical Document for public review in early June 2009. The District provided the document to staff in the Air Quality and Transportation Planning section at the California Air Resources Board. Notice was sent to interested parties

on the CAP email list serve. The District held a public workshop on June 11, 2009 to present the Draft MPEM and to solicit questions and comments. The 2016 update has primarily used the 2009 peer reviewed methodology with the most recent emission and concentration inputs.

1.1 Probability Analysis

Even though District staff has used their best estimates of the values in the calculations of each step, uncertainty exists in the calculations. To estimate the uncertainties, we have designed a Monte Carlo simulation as described in Fairley (2010).

This simulation is based on probability distributions (such as the normal distribution) for each step that represent our best understanding of the difference between calculated and (unknown) true values. We run Monte Carlo simulations that select values from these distributions to generate random repetitions of each step.

The result is a set of simulated values for each control measure, comprising a probability distribution for the benefits of the measure. These distributions can then be used to determine if apparent differences among control measures, or between a control measure and its costs, are real or due to chance.

1.2 Key Inputs to Methodology

Key inputs used in the methodology include the following:

- Estimated emission reductions for each control measure[3]
- Data on ambient concentrations of pollutants derived from the District's ambient air quality monitoring network
- Data on simulated concentrations and estimates on how changes in emissions affect ambient concentrations using outputs from the latest model applications
- Population projections at the census tract level from the California Department of Finance (2015)
- Estimates of the changes in incidence rates from changes in pollutant concentrations from a number of epidemiological studies (US EPA 2012)
- Health endpoint incidence rates for the Bay Area (OSHPD 2015)
- Health endpoint and greenhouse gas cost estimates from several valuation studies

1.3 Discussion of Key Assumptions

This MPEM is meant to provide information for air pollution policy. Inevitably, judgment has been required in developing the method, balancing completeness against practicality, and being health-protective against the uncertainty in health effects. This section discusses the choices made by District staff in developing the MPEM. Key choices in developing this method are:

[3] For stationary source measures, emission reduction estimates are provided by the District's Rule Development Section. For mobile and transportation source measures, emission reduction estimates are provided by the District's Air Quality Planning Section in collaboration with staff at the Metropolitan Transportation Commission.

- which pollutants to include
- which health effects to include
- how to deal with "background" concentrations, and
- where to set health-effects thresholds

1.3.1 Air Pollutants Included in the Methodology

There are hundreds of air pollutants, with a multitude of known and suspected health effects. However, a relatively small set has known health risks. To evaluate the health benefits of control measures, we chose a set that represents most of the known health risks from ambient air, specifically $PM_{2.5}$, ozone, and a small set of carcinogenic air toxics: benzene, formaldehyde, acetaldehyde, 1,3-butadiene and diesel $PM_{2.5}$.

$PM_{2.5}$ and ozone were chosen because they are the two criteria pollutants[4] for which the Bay Area continues to violate air quality standards. The toxic compounds chosen represent almost 90% of the known carcinogenic risk in the ambient air of the Bay Area[5]. Two other pollutants, carbon tetrachloride and hexavalent chromium, which make up much of the remaining risk from air toxics, were not considered for various reasons[6].

Some $PM_{2.5}$ is directly emitted, but a portion of ambient $PM_{2.5}$ derives from reactions of other compounds in the atmosphere. This *secondary* $PM_{2.5}$ is mainly composed of:

- ammonium nitrate, formed from ammonia and nitric acid; nitric acid, in turn, derives from NOx and its interactions with VOCs.
- ammonium sulfate, formed from ammonia and sulfuric acid; sulfuric acid , in turn, derives from SO2 and
- secondary organic aerosol, formed from reactions of various organic gases.

For direct emissions, we consider only carbonaceous $PM_{2.5}$ – the emissions of particles from burning fossil fuels and wood, and from cooking. For this study, we will consider the emissions of NOx, VOCs, sulfur-compounds and ammonia as the key precursors of secondary $PM_{2.5}$. We estimate that ammonium nitrate and ammonium sulfate account for most of the Bay Area's secondary $PM_{2.5}$ both annually and on high $PM_{2.5}$ days. The $PM_{2.5}$ components considered in this study constitute over 90% of the anthropogenic $PM_{2.5}$ in the Bay Area. In line with other health benefit studies, we assume that the impact of the various $PM_{2.5}$ components on health is the same – depending only on mass, not composition or size provided the size is < 2.5 microns.

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[4] The 1970 Clean Air Act set standards for six pollutants, called "criteria pollutants" because the standard-setting process involved compiling detailed scientific analyses about them in criteria documents. The six pollutants were ozone, TSP (now PM2.5), NO2, SO2, CO, and lead. The Bay Area comfortably meets the national (and even the stricter California) standards for the latter four pollutants.

[5] In addition to carcinogenic risks, air toxics may have both acute (short-term) and chronic (long-term) non-cancer health effects. However, for purposes of this methodology, we have chosen to focus on toxic cancer risks only.

[6] Carbon tetrachloride is ubiquitous in the atmosphere. There are virtually no emissions of it anymore in the Bay Area. Hexavalent Chromium was excluded because we do not have reliable estimates of emissions and because we have not developed the modeling to compute its formation in the atmosphere.

Benzene and 1,3-butadiene are largely directly emitted. Both formaldehyde and acetaldehyde are directly emitted but also formed via secondary processes. However, for this study, we consider only the primary (direct) emissions.

Diesel $PM_{2.5}$ is both part of overall $PM_{2.5}$ and also the Bay Area's major known ambient carcinogen (OEHHA 1998). Much of its effect, however, is included in $PM_{2.5}$ mortality, which includes death from lung cancer. We added the endpoint of lung cancer cases not resulting in death so as to include the costs of both fatal and non-fatal lung cancer.

Of the Kyoto 6 greenhouse gases, three constitute 95% of the known GHG potential of the Bay Area (BAAQMD 2015) emissions: CO_2, methane, and nitrous oxide. Ground level ozone and black carbon (soot) may contribute to global warming. However, their global warming impacts are not well-understood and have not been fully confirmed by the Intergovernmental Panel on Climate Change (IPCC), so we did not include them in the methodology.

Table 1 summarizes which emissions and pollutants are evaluated for their health/social impact in the MPEM. The middle column shows the pollutants whose emissions changes are input into MPEM. In MPEM, we only consider the health and social effects of the pollutants in the right hand column of the table.

Table 1 – Pollutants Included in Multi-Pollutant Evaluation Methodology

Category	**Direct or Precursor Emissions**	**Pollutant causing health/social impacts**
Ozone	NOx VOC	Ozone
$PM_{2.5}$	Directly Emitted $PM_{2.5}$ NOx VOC SO_2 Ammonia	$PM_{2.5}$
Toxics	Benzene 1,3-Butadiene Formaldehyde Acetaldehyde Diesel $PM_{2.5}$	Benzene 1,3-Butadiene Formaldehyde Acetaldehyde Diesel $PM_{2.5}$
Greenhouse Gases	Carbon Dioxide Methane Nitrous Oxide Sulfur Hexafluoride Hydrofluorocarbons Perfluorocarbons	GHG in CO_2-equivalent

We note some key omissions. We only considered a limited number of carcinogenic toxics. There are other carcinogens and also toxics that have other serious health effects, e.g., acrolein, lead, mercury, radon. In addition, there are risks that undoubtedly exist but have not

been quantified. An example is the carcinogenicity of woodsmoke, which is very similar chemically to tobacco smoke, a known carcinogen. Another key omission was ultrafine particles (UFP), which recent epidemiological research has demonstrated is likely to have a serious health impact distinct from $PM_{2.5}$ as a whole. We will continue to monitor the health effects literature and update the methodology with some of these omitted pollutants.

1.3.2 Choice of Health Effects

Numerous epidemiological, clinical and animal studies have linked $PM_{2.5}$ and ozone exposure to a wide variety of health effects from shortness of breath through mortality. Several recent studies use virtually the same set of ozone and/or $PM_{2.5}$ health effects (CARB 2006, Hall *et al.*, 2008, Ostro *et al.*, 2006, Stratus 2008). The effects (see Table 2) are chosen because the scientific link to pollution is well-established and because each step in the chain from emissions through health costs can be estimated.

In addition to the effects derived from the studies cited above, we add cancer from several carcinogens -- benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel exhaust.

Table 2. Health effects used in the methodology.

Health Effect	$PM_{2.5}$	Ozone	Toxics
Mortality	x	x	x
Chronic Bronchitis Onset	x		
Respiratory Hospital Admissions	x	x	
Cardiovascular Hospital Admissions	x		
Non-Fatal Heart Attacks	x		
Asthma Emergency Room Visits	x	x	
Acute Bronchitis Episodes	x		
Upper Respiratory Symptom Days	x		
Lower Respiratory Symptom Days	x		
Work Loss Days	x		
Minor Restricted Activity Days	x	x	
School Absence Days		x	
Cancer			x

Except for diesel, which is a constituent of $PM_{2.5}$, we include both fatal and non-fatal cancer. For diesel, we include only non-fatal cancer to avoid double counting $PM_{2.5}$ mortality.

The health impacts of GHGs are not incorporated explicitly, but are implicit in the estimate of overall social cost.

1.3.3 Pollutant Concentrations and Behavior

Once the emissions reductions for a potential control measure have been estimated, the next step is to estimate how these changes in emissions affect ambient concentrations. The expectation for directly emitted pollutants is that concentrations should be reduced in the

same proportion as the reduction in emissions. For example, a 10% reduction in emissions should lead to a 10% reduction in ambient concentrations. The relationship is complicated, however, because 1) not all pollution is locally generated; "background" pollution mixes in from other areas, and 2) locally generated pollution can travel outside the Bay Area.

For secondary pollutants like ozone and ammonium nitrate, the relationship with emissions is further complicated by chemical reactions. The amounts of these secondary compounds formed depend on a host of factors including ambient temperature, sunlight, humidity, the ratios of precursor compounds, and atmospheric ventilations.

To account for these issues, we use the results of computer models that simulate pollutant concentrations from pollutant emissions. These models incorporate both meteorology and chemistry to show the transport and transformation of pollutants. Simulation results used in this study are taken from the available episodic or seasonal simulations. The results from the toxics modeling were available for one week in July and another week in December, 2005; from the PM and ozone modeling for the entire January and December, and 2-week periods from March, May, August, and October, 2012.

The models (Soong, et al., 2015, Tanrikulu et al., 2009) provide an estimate of the relationship between a change in emissions and a change in pollutant concentration for ozone and particulate matter, and toxics respectively.

The health related pollutants, except for diesel[7], are measured at various District air monitoring stations around the Bay Area. These observations were used to validate the ozone and $PM_{2.5}$ concentrations simulated by the models.

Toxics: From a limited trend analysis conducted (Appendix A), we found a linear relation between declines in concentrations and emissions of benzene and 1,3-butadiene. We assume the relationships between concentrations and emissions of diesel, formaldehyde and acetaldehyde are also linear.

We also investigated changes in simulated concentrations for the species above in response to reductions in emissions and found a linear relationship between them. For each of the toxics, regressions using grid-by-grid pairs had slopes of 0.90.

$PM_{2.5}$: For $PM_{2.5}$, ambient trends also indicate a linear relationship between emissions and ambient concentrations, but when projected toward a non-zero background. $PM_{2.5}$ is a complex pollutant, being composed of a number of different components both primary and secondary.

Ozone: A number of studies have been conducted based on ambient data to investigate how ozone concentrations have been responding to changes in emissions.

Ozone concentrations are almost entirely a result of chemical reactions between ozone precursors NOx and volatile organic compounds (VOCs). The model showed a nearly linear

[7] Diesel PM cannot be directly measured.

relation between changes in precursors and changes in ozone concentrations. However, the magnitude of the response of simulated ozone to changes in emissions appears to be smaller than historic ambient change in ozone for changes in NOx and VOCs emissions. (See Appendix X.)

1.3.4 Health Effects Thresholds

Potentially, the harmful effects of a pollutant may diminish or disappear entirely below a certain concentration. But scientifically, it may be very difficult to find such thresholds, and thresholds may vary among individuals. Therefore, we took an approach that balanced health protectiveness and this scientific uncertainty.

Toxics: For carcinogens, the common assumption has been that no threshold exists; even extremely small concentrations can cause cancer, just with correspondingly small probabilities. We use the assumption of a linear effect with no threshold for purposes of the multi-pollutant method.

PM: Most epidemiological studies of the health impacts of $PM_{2.5}$ that tested for a threshold have not detected one. An EPA study (US EPA 2006a) eliciting opinions about the effect of $PM_{2.5}$ on mortality found that the experts were nearly unanimous in rejecting the idea of a population-wide threshold, although some thought it may exist at the individual level. We assume no threshold for $PM_{2.5}$ for the "best" scenario, but take the possibility of a threshold into account in the probability analysis.

Ozone: Ozone represents a special case. Although epidemiological studies looking for a threshold have been inconclusive, clinical studies of exercising individuals have found them. Several studies of ER visits for asthma suggest a population threshold in the range of 0.075–0.110 ppm for 1-hr maxima. Ostro *et al.,* (2006) noted that most epidemiological studied "include very low concentrations in their analysis." Ostro *et al*. used ozone background of 40 ppb as the no-effects threshold for their "best estimate", doing sensitivity analyses for no-effects thresholds between 50 ppb and 70 ppb. For this analysis, we assume a 45 ppb threshold.

1.3.5 Scientific Evidence for Causality

The MPEM is based on an implicit assumption that the connection between air pollution and health and other social effects is causal. The evidence for many of the health effects is epidemiological, whereby an *association* has been demonstrated between a health effect and changes in the concentration of a pollutant. In other cases, a causal connection has been demonstrated in laboratory animals for pollutant concentrations, often at high doses, or reversible changes in physiological pathways or markers have been found in human subjects. Although it is difficult to prove causality between air pollution and health effects, sufficient evidence has been found for the effects used in the MPEM, based on the coherence of the evidence from epidemiological, clinical, animal and occupational studies.

Toxics Judgments of causality have been made at both the national (NTP 2016) and international (IARC 2016) levels. At both levels, benzene, 1,3-butadiene and formaldehyde are judged human carcinogens; the other toxic used in MPEM, acetaldehyde, is considered a likely carcinogen, and diesel is considered a carcinogen by IARC and "reasonably anticipated to be a carcinogen" by NTP.

$PM_{2.5}$ The EPA's Integrated Science Assessment or ISA (US EPA 2009a), is a compendium of facts and scientific understanding about PM. The ISA draws upon the work of dozens of scientists. Chapter 7 of the ISA summarizes the current consensus regarding the relationship between PM and health effects. The chapter concludes that the relationship between long-term PM2.5 exposures and mortality is causal.

Ozone EPA has evaluated the evidence for ozone health effects in its Integrated Science Assessment (US EPA 2013). Humans subjected to elevated ozone concentrations experience cough, chest pain, decreased lung volumes, and airway irritation. Asthmatics are affected more strongly. Causal determinations for short-term effects of ozone, which are the health endpoints considered in MPEM, are "causal" for respiratory effects and likely to be causal for cardiovascular effects and mortality. (US EPA 2013, Table 6-54, pg 6-264.)

GHGs The Intergovernmental Panel on Climate Change (IPCC) is the key international scientific group tasked with pooling the vast array of research on climate change. They conclude:

1. The evidence for "…warming of the (world) climate system is unequivocal." (IPCC 2014, pg 2)
2. "Anthropogenic greenhouse gas emissions have increased since the pre-industrial era, driven largely by economic and population growth, and are now higher than ever. This has led to atmospheric concentrations of carbon dioxide, methane and nitrous oxide that are unprecedented in at least the last 800,000 years. Their effects, together with those of other anthropogenic drivers, have been detected throughout the climate system and are extremely likely to have been the dominant cause of the observed warming since the mid-20th century." (IPCC 2014, pg 4)

Thus, for the reasons described above, the MPEM is based on the assumption that air pollution causes health effects, and that emissions of greenhouse gases cause or contribute to climate change.

1.3.6 Apportioning emissions reductions on a geographic basis

In this study, we assumed that the reductions of a pollutant for a rule are spread proportionately across all emissions of that pollutant. For example, if a control measure reduced NO_x by 1 ton per day, and our emissions inventory shows that a total of 300 tons of NO_x is emitted in the Bay Area per day, we would calculate the impact of a $1/300^{th}$ reduction in NOx emissions by geographically distributing the NO_x reduction the same as the geographic distribution of the entire NO_x inventory.

These assumptions are due to their practicability. Attempting to calculate more focused effects is currently beyond the scope of our work. Even allocating the emission reductions to specific emission inventory source categories, although more accurate, would require many separate resource-intensive model runs.

2. Estimating Concentrations from Emissions

This section explains how the MPEM calculates ambient concentration estimates from emissions. Although the approaches are generally similar, the estimation methods for toxics, ozone and $PM_{2.5}$ are discussed separately.

The District operates computer models that simulate pollutant concentrations from an emissions inventory. The model output is hourly concentrations of the pollutants for a 4 km by 4 km grid for ozone and $PM_{2.5}$ and 1 km by 1 km grid for toxics, covering regions that include the Bay Area, and covering certain periods during the year. The models use the 2012 base year emissions inventory for ozone and PM and the 2005 base year inventory for toxics.

The models were run both for a base case and for various sensitivity runs where the Bay Area emissions of precursors were reduced by a certain percent (10% or 20% were used in different simulations). The difference in concentrations between the base case and these sensitivity runs serves as the basis for estimating how emissions reductions affect pollutant concentrations.

In developing the multi-pollutant evaluation method, we use the modeled and ambient concentration data to develop formulas for each grid square that relate the change in emissions of harmful pollutants or their precursors to the change in the ambient concentrations of these pollutants.

For the case of a *primary* pollutant, the change in a concentration field (Δc_{ri}), resulting from the emission reductions for a given control measure, is estimated using equation 2.1

$$\Delta c_{ri} = c_{0i} (dc_i/de) \Delta e_r \qquad (2.1)$$

Where c_{0i} is its initial concentration in grid square i, Δe_r is a percentage of total annual District emissions of that pollutant that are reduced by the control measure, and dc_i/de is the percent change in concentration of the pollutant in grid square i for a percent change in emissions of the pollutant derived from the model. The initial concentration, c_{0i}, derives from modeled values for toxics and ambient concentrations for directly emitted $PM_{2.5}$.

For the secondary pollutants in the MPEM, analysis of model runs showed that the joint effect of all precursors was approximated well by the sum of impacts of individual precursors, that is, ignoring interactions among the precursors. But the *magnitude* of the impact is affected. For example, the reduction in ammonium sulfate from a given reduction in ammonia considered in isolation is different from the reduction when considered jointly with other precursors. This is analogous to the difference between a simple derivative and a partial derivative. We will term this latter case a *jointly-considered* reduction.

Individual runs that provide estimates of the effects of precursor reductions individually, and a model run where all precursors are reduced jointly available are. These together allow for an approximation of the jointly-considered reduction, as follows:

A regression is run with the joint-model run concentration change, y, as the dependent variable and the individual-model run concentrations as the independent variables, x_1, x_2, ..., x_k. The resulting fitted regression equation:

$$y = f_1x_1 + f_2x_2 + ... + f_kx_k$$

provides factors to convert from the marginal effect to the jointly-considered effects. Symbolically, we have

$$\delta c_i/\delta e_j \approx f_j dc_i/de_j$$

Then the change in secondary pollutant concentration from a change in its precursors is

$$\Delta c_{ri} = c_{0i} [f_1(dc_i/de_1) \Delta e_{1r} + f_2(dc_i/de_2) \Delta e_{2r} + \ldots] \qquad (2.2)$$

where dc_i/de_1 is the percent reduction of the pollutant concentration from a percent reduction in the 1st precursor in the model run reducing the 1st precursor only, dc_i/de_2 is the percent reduction of the pollutant concentration from a percent reduction in the 2nd precursor from the model run reducing the 2nd precursor only, and so on. And Δe_{1r}, Δe_{2r} …are the percent reductions in precursor 1, precursor 2, and so on, of a given control measure.

2.1 Key considerations

There are several considerations that apply to all pollutant categories.

Concentrations and health effects: The health impact formulas that relate pollutant concentrations and exposures to health effects generally require pollutant concentration estimates for the whole year. With this update, we now have sufficient modeling to estimate the annual averages and annual peak values reasonably well.

Concentration averaging time: A related consideration is the concentration averaging time used to relate to the health effects discussed in subsequent sections. For example, for ozone, the focus has been on peak values such as the daily maximum 1-hour average concentration. But for toxics, the focus has been on the annual mean. For $PM_{2.5}$, both the 24-hour concentration and the annual concentration are of interest. In the MPEM, ozone values were estimated on a daily basis; for toxics and $PM_{2.5}$, estimated annual averages were used.

Estimating initial concentrations – use of ambient concentrations: The models are used for estimating the *change* in concentrations. They were also used to estimate initial concentration for toxics, based on an analysis of ambient concentrations that showed that the July+December mean was close to the annual mean. Similarly, for $PM_{2.5}$, the average of

Jan+May+Aug+Oct ambient average concentrations were comparable to the annual. So these averages were used for initial concentrations. For ozone, where an extensive monitoring network of BAAQMD monitoring sites provides good spatial estimates of daily ozone concentrations, daily maximum ozone concentrations for 2011-13 were used, interpolated to each grid square.

Primary or/and Secondary: As discussed in 1.3.1 above, the health-related pollutants may be primary or secondary or sometimes both. Ozone is virtually all secondary. For this analysis, secondary toxics modeling was not available, so only the primary emissions of toxics were considered. But for $PM_{2.5}$, we considered both directly emitted $PM_{2.5}$, largely carbonaceous particles from burning fossil fuels and wood, and secondary $PM_{2.5}$, specifically ammonium nitrate and ammonium sulfate.

2.2 Air Toxics

2.2.1 Concentration averaging times

Unlike for ozone and $PM_{2.5}$, there are no national or California air quality standards for air toxics. Rather, the issues are morbidity effects generally, and cancer risk in particular. Cancer onset is believed to be proportional to long-term exposure, so the averaging time of interest is annual.

We simulated toxics concentrations one week in December, 12/12-12/18, 2005 and one week in July, 7/12-7/18, 2005, using meteorology from those weeks and an hourly emissions inventory. For this analysis, for each toxic compound, the compound's concentration was averaged across hours, days and seasons to get an estimated annual average for each specific grid square. Simulated toxics concentrations were compared against observations where possible.

2.2.2 Relating toxic concentrations to toxic emissions

We conducted sensitivity simulations, each with an across-the-board 10% reduction in the emissions of one of the toxic compounds. The reduction in the annual averaged modeled concentration of that toxic, Δc_{i0}, was found and its ratio to the initial concentration computed: $\Delta c_{i0}/c_{i0}$. This provided a coefficient that relates percent change in concentration to percent change in emissions, dc_i / de.

Figure 1 shows the model results for a 10% reduction in benzene emissions. Benzene concentrations are reduced by amounts ranging from 0.01 ppb, shown in yellow, to 0.08 ppb, shown in red.

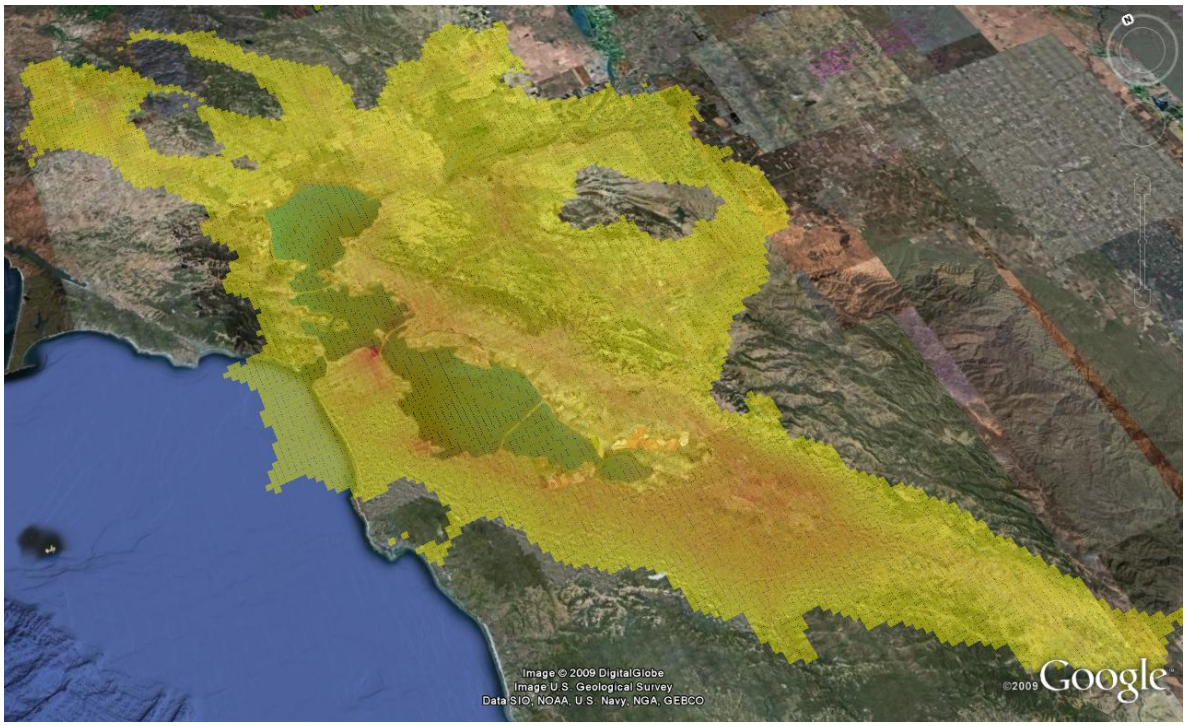

Figure 1. Model results for a 10% reduction in benzene emissions. Grid of model estimated reductions in benzene concentrations for a 10% reduction in benzene emissions. Values range from 0.01 ppb (light yellow) to 0.08 ppb (red).

This was combined with the estimated initial concentrations, c_{0i}, estimated in the next two sections below to yield the values for equation (2.1) above.

2.2.3 Concentrations, except for diesel

Ambient toxics data are collected from a number of Bay Area sites. However, only three BAAQMD sites – Fremont, San Francisco, and San Jose – measure formaldehyde and acetaldehyde, and only these sites have limits of detection for benzene and 1,3-butadiene low enough to get a good estimate of their annual averages. For these sites, an analysis of the ambient data showed that the July+December mean was similar to the annual mean.

Modeled benzene and 1,3-butadiene concentrations agreed well with ambient measurements, but formaldehyde and acetaldehyde were underestimated by the model. The latter two compounds have a substantial secondary component, but it is not clear whether this secondary component is what was underestimated.

2.2.4 Diesel PM concentrations

Unlike the other toxics, diesel concentrations are not monitored. The science of measuring diesel exhaust is still evolving. There is, however, a rough correspondence between diesel PM and elemental carbon, which has been measured at a number of Bay Area sites. Comparisons of modeled diesel concentrations and elemental carbon suggest that the modeled concentrations are reasonable. Thus, for this methodology, we rely on modeled concentration

estimates, that is, for any cell, we take the initial concentration as the mean July+December modeled diesel concentration.

2.3 The CMAQ model for ozone and $PM_{2.5}$

The CMAQ model was run to simulate both ozone and $PM_{2.5}$ concentrations and their sensitivity to precursor reductions for the entire January and December, and 2-week periods for March, May, August and October, 2012. Sensitivity simulations include twenty percent reductions in emissions of: NOx, VOC, ammonia, SOx, and total PM. A run was also made with a 20% reduction in all of these pollutants simultaneously. The model produced hourly average concentrations for a 4x4 grid covering the San Francisco Bay Area.

2.4 Ozone

2.4.1 Ozone concentration averaging times

Ozone standards are specified for peak ozone. The national primary ozone standard is based on daily 8-hour maximum ozone.[8] It states that the 3-year average of the annual 4th highest 8-hour maxima cannot exceed 70 ppb at any site. California has an 8-hour standard of 70 ppb that, in essence, allows at most 1 exceedance per year. It also has a standard that allows the 1-hour maximum ozone to exceed 95 ppb at most once per year.

Epidemiological studies investigating the relationship of ozone and health effects have used both 1-hour and 8-hour averages, and health benefits analyses have generally used one or the other. Because the correlation between 1-hour and 8-hour averages is generally high, it is reasonable to use a conversion from one to the other.

For the health effects analysis below, the 1-hour daily maximum average concentrations are used.

2.4.2 Relating the change in ozone concentrations to the changes in precursor concentrations

The approach here was more elaborate than for the other health-related pollutants. A photochemical model (CMAQ) was run for varying combinations of ozone's precursors, NO_x and VOCs, with all 9 combinations of no reductions, 5% and 10% reductions for NOx and VOCs. The goal was to estimate the change in ozone as a function, f(n,v), of reductions in NOx and VOCs, that included the possibility of non-linear interactions.

After analyzing the results, we found, however, that a simple linear model sufficed, with modeled changes in ozone being a linear combination of modeled changes in NOx and VOCs. Specifically, for each grid cell i, the model

[8] Ozone measurements are collected as 1-hour averages. To compare with the standard, running 8-hour averages are computed for each day starting from midnight-7:59am, 1:00am-8:59am, and so on, then the maximum among these is obtained. These values are computed on a site-by-site basis.

$\Delta y_i = y_i[a_i*\Delta e_n + b_i*\Delta e_v]$

was fit, where Δy_i was the reduction (or increase) in ozone from the base case, y_i was the initial ozone concentration, and Δe_n and Δe_v were the percent reductions in NOx and VOC.

2.4.3 Estimating initial ozone concentrations

Ambient ozone is measured at 21 Bay Area sites. There is considerable geographic variation in ozone concentrations, but interpolation from the site network is sufficient to provide reasonable estimates for other Bay Area locations.

The CMAQ simulations involved modeling four seasons. These runs provide good information on how the relationship between ozone formation and precursor changes spatially and a reasonable estimate of the relationship by season.

In order to compute changes in ozone concentrations, we applied the change functions derived from the model to observed daily 1-hour maximum ozone for 2011-13, interpolated to a concentration c_{0i} for each grid square.

2.4.4 Incorporation of a 45 ppb threshold

Both for air quality and health reasons, we decided to consider only changes in ozone for grid-cell-days where interpolated 1-hour max ozone concentrations exceeded 45 ppb. (See Appendix B for additional discussion.) Keeping track of every such day for every grid cell would have been cumbersome, so an approximation was made that estimated total ppb-days above 45 ppb as a function of r% reduction in ozone. For each grid square i, the function $f_i(r) = c_i + d_i r + e_i r^2$ = estimated ppb-days above 45 ppb for a reduction of r% was fit for values of r = 0, 1, …, 20, where $f_i(r)$ was the summation of $z_j = (1\text{-}r)y_j - 45$ for all 1-hr max ozone values y_j in 2011-13 for which z_i was positive. The fits were generally excellent, with over 95% of the fits having R^2 values > 0.99.

2.4.5 Calculation of ozone above threshold

Combining these factors, if the reduction in NOx emissions from a given control measure (as a percent of total NOx emissions) is Δe_n, and the reduction in VOC is Δe_v, then the average daily 1-hr max ozone above 45 ppb in grid square i is estimated as:

$\Delta f_i = [f_i(0)\text{-}f_i(r_i)]/(3*365)$,

where $r_i = a_i\Delta e_n + b_i\Delta e_v$ = estimated percent reduction in ozone concentration.

2.5 $PM_{2.5}$

2.5.1 $PM_{2.5}$ concentration averaging times

There are both national and California standards for annual average $PM_{2.5}$ concentrations and a national $PM_{2.5}$ standard for peak 24-hour values. The Bay Area currently meets both standards.

Two main kinds of epidemiological studies have been performed to investigate the relation between health effects and $PM_{2.5}$: 1) time series comparisons of daily effects such as emergency room visits or death with daily (and/or lagged daily) 24-hour $PM_{2.5}$ concentrations, and 2) for mortality, comparison of mortality rates in different areas with the annual $PM_{2.5}$ concentrations in those areas, where the mortality rates are adjusted for personal factors such as smoking, age, gender and occupation.

To be consistent, one would estimate the health effects derived from time series using changes in daily 24-hour $PM_{2.5}$. For the sake of simplicity, however, we only consider the annual average. See Appendix B for details on the effect of this approximation.

2.5.2 Components of $PM_{2.5}$

$PM_{2.5}$ is composed of particles from many different sources. In the Bay Area, the key sources of direct $PM_{2.5}$ include emissions of carbonaceous particles from burning fossil fuels, burning wood and other vegetative matter, and cooking; and oceanic background, largely sea salt and sulfate. Geological dust is only a minor component of $PM_{2.5}$ as are tire wear and brake dust. There is also a large secondary component composed primarily of ammonium nitrate and ammonium sulfate. In MPEM, our analysis of $PM_{2.5}$ is limited to three components of $PM_{2.5}$: directly emitted carbonaceous $PM_{2.5}$, ammonium sulfate and ammonium nitrate. For purposes of the MPEM, we consider each of these three major components of $PM_{2.5}$ separately.

2.5.3 $PM_{2.5}$ simulation using the CMAQ model

The CMAQ model was run to estimate the sensitivity of concentrations of components of $PM_{2.5}$ to reductions in various emissions. A base case was run with the originally estimated emissions for 2-30 January and December and 2-15 March, May, August and October. Six sensitivity runs were also made with 20% reductions in: NOx, VOC, ammonia, sulfur gases including SO_2, directly emitted $PM_{2.5}$, and reductions in all 5 categories.

2.5.4 Relating the change in direct carbonaceous $PM_{2.5}$ concentrations to emissions

Two components of direct carbonaceous particles were recorded by the model: elemental carbon (EC) and organic carbon (OC). Both of these components are emitted directly, but organic carbon also forms in the atmosphere through chemical reactions of organic gases. We assumed that the modeled change in EC concentrations resulted from a change in EC emissions, and similarly for OC.

A comparison of the model with 20% reductions in all precursors showed almost the same changes in $PM_{2.5}$ concentrations from the base case as the sensitivity runs for OC and EC reductions alone. Therefore, no adjustment was made to convert from marginal to jointly-considered effects.

2.5.5 Relating the change in precursor emissions to the change in ammonium nitrate concentrations

Although there is some direct emissions of nitrate, virtually all of it is formed in the atmosphere from other compounds, principally the conversion of ammonia and NOx, although other compounds may participate in intermediate reactions. Figure 2 shows the results of sensitivity simulations where Bay Area emissions of ammonium nitrate precursors were reduced by 20% one at a time. Reductions of 20% in NOx resulted in reductions of between 1% and 4% in nitrate, with a median just above 2%. Nitrate reductions from reductions in ammonia tended to be somewhat greater, ranging between 2% and 5% with a median of about 3.5%. Reducing VOC had a much lower impact, less than 1%, and the impact of reducing SOx was negligible.

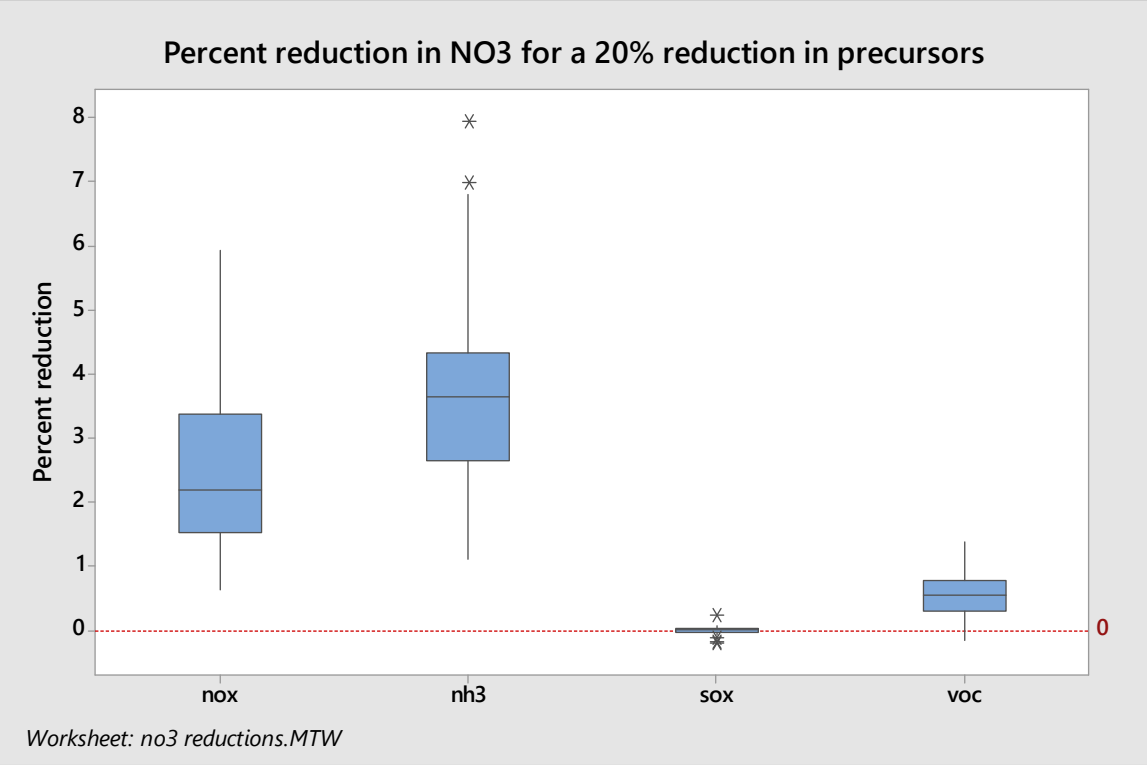

Figure 2. Boxplots of the percent reduction in nitrate for a 20 percent reduction in the emissions of ammonium nitrate precursors. Shown are results for grid squares with ambient air monitoring sites.

2.5.6 Factors for joint effects of precursors

A multiple regression was performed with the nitrate concentration change from a run reducing all precursors as the dependent variable and the reductions from 20% reductions in NOx, NH3 and VOC independent variables. The factors for adjusting from marginal to jointly-considered effects were 1.14 for ammonia, 0.88 for NOx and 1.79 for VOC.

2.5.7 Calculation of the change in ammonium nitrate concentrations

A factor of 80/62 was used to convert from nitrate mass to ammonium nitrate mass.[9]

So, for a given percent reduction in emissions of ammonia, Δe_a, for NO_x, Δe_n, and for VOC, Δe_v, we predict a change in ammonium nitrate concentrations in grid square i of:

$$\Delta c_i = c_i*(80/62)*[\ 1.14(dc_i/de_a)\ \Delta e_a + 0.88\ (dc_i/de_n)\ \Delta e_n + 1.79(dc_i/de_v)\ \Delta e_v]$$

2.5.8 Relating the change in ammonium sulfate concentrations to the change in precursor emissions

Analysis of the modeled changes in ammonium sulfate concentrations as a function of 20% reductions of various precursors showed a reduction of 4%-7% for reductions in directly emitted sulfate, but only about 0.5% reductions for SO2, and essentially zero reductions for ammonia. (See Figure 3.)

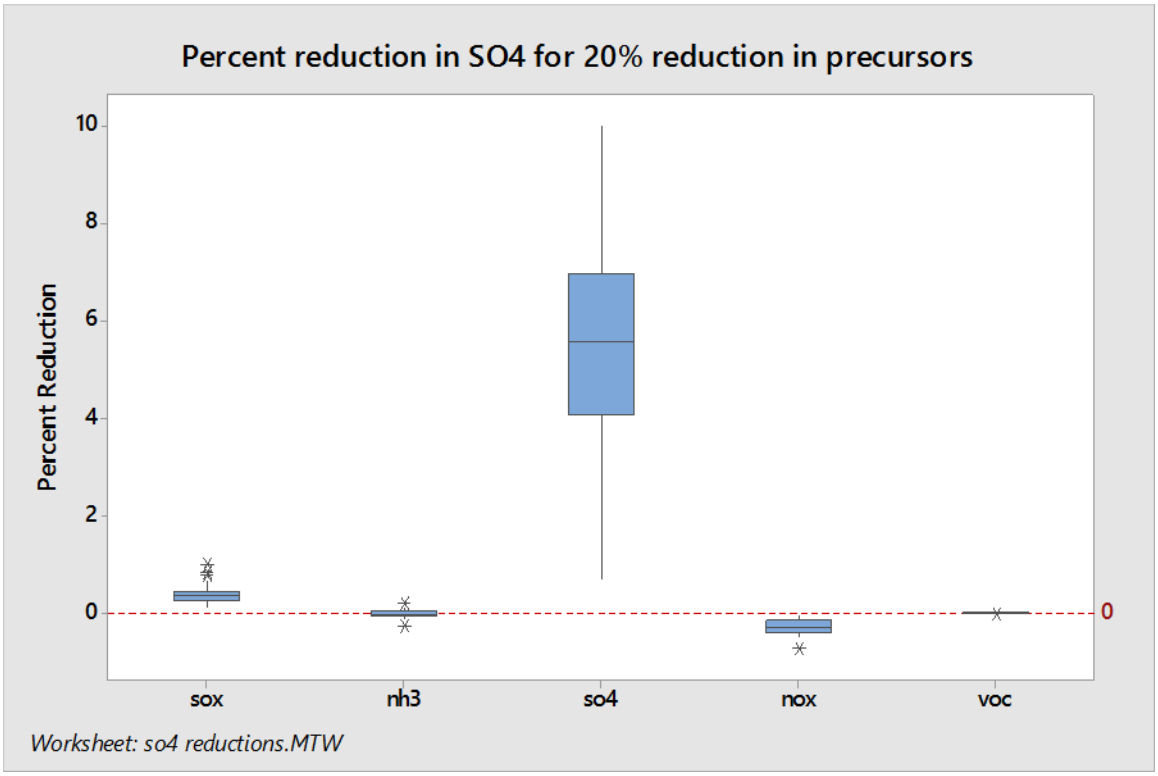

Figure 3. Boxplots of the percent reduction in sulfate for a 20 percent reduction in the emissions of ammonium sulfate precursors. Shown are results for grid squares with ambient air monitoring sites.

2.5.9 Factors for joint effect of precursors

A multiple regression was performed with the sulfate concentration change from the run where all pollutants were reduced as the dependent variable and the reductions from the runs for SO2, ammonia and SO4 as the independent variables. The factors for adjusting from

[9] Nitrate, NO_3, has atomic weight 62. Each nitrate molecule combines with an ammonium (NH4) molecule, for an atomic weight of 62 + 18 = 80.

marginal to jointly-considered effects were 0.85 for ammonia, 0.90 for sulfur gases and 1.03 for sulfate.

2.5.10 Calculation of the change in ammonium sulfate concentrations

The model output is in terms of sulfate, so this value was adjusted to convert from sulfate to ammonium sulfate: (132/96).[10]

So, for given percent reductions in ammonia, Δe_a, and sulfur species, Δe_{ss}, and Δe_{sa}, and we predict a change in ammonium sulfate concentrations in grid square i of:

$$\Delta c_i = c_i*(132/96)*[0.85(dc_i/de_a)\ \Delta e_a + 0.90(dc_i/de_{ss})\ \Delta e_{ss} + 1.03(dc_i/de_{sa})\ \Delta e_{sa}]$$

where c_i is the average sulfate concentration from the base-case model run, and dc_i/de_j is the percent change in concentration in grid square i from a percent change in species j computed from the base case model run and the model run with a 20% marginal reduction in species j.

3. Estimating Population Exposure

3.1 Population and Demographics

The MPEM uses population data in two different ways. One is to compute population-weighted exposures. For this, total population is required on a fine geographic basis. The other use is to compute incidence rates. Many of the health endpoint estimates involve incidence rates for a specific age range, e.g., 5-17 for school absences or ≥ 27 for chronic bronchitis. Here county-level data by age group is utilized.

To obtain spatially disaggregated population data, we used Census data from the American Community Survey (ACS) for the most recent period available, 2009-2013 (US Census 2015). We obtained population estimates at the census block group level, and applied each to the grid square containing the block group's population centroid. This population was assumed exposed to the concentration estimated for that grid square. The product of population times Δconcentration was then summed for each county and divided by the county population, yielding a population-weighted Δconcentration. Figure 4 provides an example.

To estimate incidence of health endpoints, we used data from the California Department of Finance. These data are projections based on sophisticated modeling that includes ACS data. These were available by county for 2015 by for each age 0-100. These were aggregated into the age ranges needed to estimate incidence rates for various age groups.

[10] Sulfate, SO4 has atomic weight 96. Each sulfate molecule combines with 2 ammonium (NH4) molecules, for an atomic weight of 96 + 2 x 18 = 132.

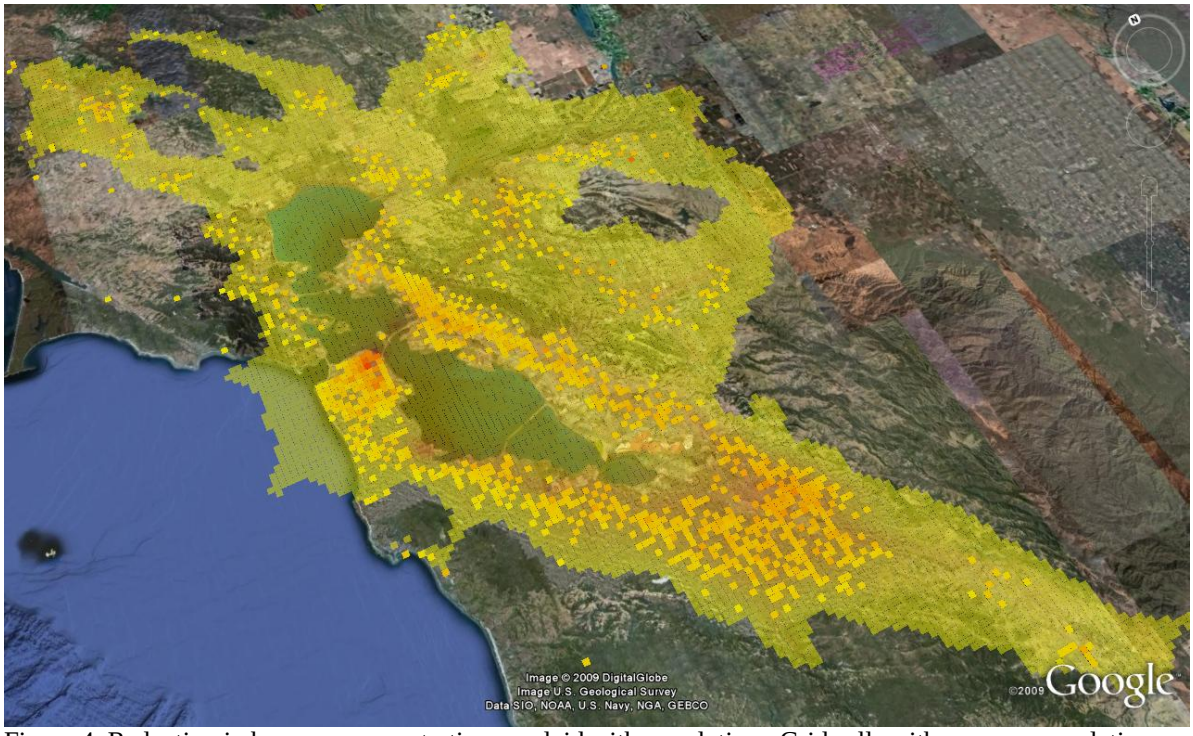

Figure 4. Reduction in benzene concentration overlaid with population. Grid cells with nonzero population shown in solid colors underneath benzene reductions layer.

3.2 Exposure Rationale for Ozone and $PM_{2.5}$

Our method estimates "backyard" exposure, namely assuming that people are at home and outside in their yards all the time (24 hours a day, seven days a week). Although this approach is admittedly simplistic, it is generally consistent with the exposure estimates made in the epidemiological studies that produce the dose-response functions used in MPEM and thus is an appropriate method in combination with steps 4 and 5.

Very few of us spend our entire lives in our backyard. Improvements in estimating real exposures will be of tremendous value, but will require considerable effort to gather data on people's activity patterns and concentrations in various micro-environments. For the current MPEM methodology, however, our simplistic exposure assumptions may be adequate, or even appropriate.

Most of the epidemiological studies used to calculate ozone and $PM_{2.5}$ health effects themselves use only these rough estimates of exposure. Thus, the concentration-response relationships developed are also based on backyard estimates of exposure. In fact, a number of these studies assumed that everyone within a county was exposed to the average monitored value in the county, possibly based on a single monitor. Thus, if anything, our own rough backyard exposure may be a more precise estimate than those used to establish the concentration-response relationships.

What is the effect of this approximation? There are two aspects, both of which suggest that our methods will, if anything, underestimate the pollutant effects on the health effects.

3.2.1 Average ambient concentration

The average ambient concentrations used in the epidemiological studies are not, in general, equal to the average exposure concentration.

For example, suppose average population exposure concentration was 80% of the value measured at monitoring station. Suppose the concentration-response (C-R) slope for, say, hospital admissions, estimated in the epidemiological study were an incidence rate of 0.02 / 10 $\mu g/m^3$ so that there was a 2% change in incidence for a 10 $\mu g/m^3$ change in *monitored* $PM_{2.5}$ concentrations. But actual exposures were not the same, so really, this is a 0.02 change for an 8 $\mu g/m^3$ change in *exposure* concentrations. Thus the C-R slope is 10/8 x (0.002) = 0.0025 per $\mu g/m^3$ in $PM_{2.5}$ exposure.

In the Bay Area, at least, air quality monitors tend to be placed in areas with above-average concentrations. To the extent that this is true in the areas where C-R functions have been calculated, this would cause an underestimation of the response for a given concentration, provided that unbiased estimates of backyard concentrations were used. For $PM_{2.5}$, we used modeled values, which may be unbiased. Thus, for $PM_{2.5}$ C-R functions, the response may be underestimated. For ozone, monitored values were interpolated to backyard values thereby approximately canceling the bias. That is, the backyard ozone values are likely to overestimate actual backyard ozone concentrations, thereby roughly canceling the presumed underestimate in the C-R functions.

It should be noted that if we were able to use the true exposures, the bias would be even stronger because people spend most of their time indoors. The amount of $PM_{2.5}$ that infiltrates is perhaps 70% of the ambient levels (See, e.g., Lurmann & Korc 1994), so this would impart a greater downward bias.

In layman's terms, the concentration-response function (C-R coefficient) is calculated by analyzing the relationship between known health outcomes for a given population compared with their estimated population exposure. So if population exposure is over-estimated (e.g. by using monitored concentrations that are higher than real exposure), this will result in underestimation of the C-R coefficient. If an under-estimated C-R coefficient is then applied to a more accurate (in this case, lower) population exposure, this will result in underestimation of health effects.

3.2.2 Exposure

Exposures were estimated with error. If exposure were estimated without bias[11], but with error, then the C-R coefficient would tend to underestimate the effect of the pollutant on the health endpoint. This is a regression theory result, where if the independent variable, x, is

[11] Bias is a systematic over- or under-estimation, like a scale that always reads 3lbs lighter than you really are. Error means the difference between the measured and true value. So a scale might be unbiased but sometimes read up to 2lbs more than the real weight and sometimes down to 2lbs less than the real weight, so the error is plus or minus 2 lbs.

measured with error: x* = x + error, and the error has zero mean, then the fitted regression slope, b*, of the regression of y on x* will tend to be less in magnitude than the true slope, β, from the regression of y on x (had it been known). That is |b*| < | β |. See Appendix D.

This issue was considered important by the experts in EPA's elicitation of experts' judgement about the true $PM_{2.5}$ – mortality C-R function:

> "Uncertainties in population exposures assessed using central-site monitoring was raised by all experts as an important issue, and in many cases as a major issue, and nine experts took this issue into account when deriving their median effect estimate of the mortality effects of a 1 μg/m3 change in PM2.5…many thought that this issue caused underestimation of the effects of PM2.5 on mortality. The reason cited for this underestimation was the well-known effect of exposure measurement error ("misclassification") in biasing epidemiological effect estimates towards the null." (page 3-18, EPA 2006a)

Thus, this is a second reason why it's likely that the C-R coefficients from epidemiological studies underestimate the true C-R effect.[12] In our case, it is likely that even with backyard exposures we are estimating the true exposure more precisely than simply using the monitored value. Thus, all else being equal, if the same studies had been done using such backyard exposures instead of monitored values, the slopes would likely have been steeper. In other words, this is a second reason that it is likely that we will underestimate the true effect of the pollutants on health effects.

3.3 Exposures and Cancer

The opposite relation may exist with our estimates of cancer effects, although the exposure bias is dwarfed by other uncertainties. The health impacts from toxic air pollutants are estimated from occupational studies or studies of lab animals. In occupational studies, exposure estimates are often very rough. In studies of lab animals, the exposure may be well-controlled, but the low-dose extrapolation and extrapolation from other species to humans introduce large uncertainties.

In addition to these large uncertainties, there is likely to be a modest overestimate of exposure: We believe our models do a reasonable job of estimating backyard exposure. But indoor exposures are likely to be lower, at least for diesel particulate matter (DPM). The assumed lung cancer risk for DPM is 300 in a million per $\mu g/m^3$ (OEHHA 2016) for an average lifetime exposure of 1 $\mu g/m^3$ (a concentration very near the annual average for the Bay Area). As discussed above, however, most people spend most of their time indoors, say 90%, so, assuming that the PM penetration rate is 70%, then their average exposure would be about 0.1(1) + 0.9(1 x 0.7) = 0.73 $\mu g/m^3$, for a true risk of 0.73 x 300 = 219 in a million.

[12] The $PM_{2.5}$ – mortality C-R function is an exception because it is based on the pooled judgement of these experts, who took this bias into account in their estimates.

4. Estimating the Impact of Exposure on Health Effects

4.1 Calculation of Health Impacts: C-R Functions

All the health impact calculations have a similar form, a formula that relates a change in exposure concentration to a change in the number of cases of a particular health endpoint such as an emergency room visit, hospital admission, missed school day, or death:

$$\Delta\text{cases} = \text{baseline incidence} \times \Delta\text{risk} \qquad (4.1)$$

where

- Δcases = the annual increase or decrease in the number of cases of that health endpoint in the population resulting from the change in exposure,
- baseline incidence = the underlying rate of that health endpoint, expressed as a number of cases, and
- Δrisk = change in risk of an incidence of that health endpoint resulting from the change in exposure.

The actual function in 4.1 is termed a *concentration-response function* or *C-R function*.

4.2 C-R Functions for Ozone and $PM_{2.5}$

For ozone and $PM_{2.5}$, the C-R functions are generally derived from epidemiological studies that examine the correlation between a health endpoint and exposure to ozone or $PM_{2.5,}$ in conjunction with other potential factors that might affect the endpoint. These additional factors include other pollutants, such as sulfur dioxide, carbon monoxide and nitrogen dioxide, as well as factors such as extreme temperatures, time of year, day of the week, etc. All the C-R functions in this methodology are of two forms, either log-linear or logistic.

Log-linear: The statistical analysis is often equivalent to a regression on the log of the number of incidents. This implies that the regression coefficient for ozone or $PM_{2.5}$, say b, represents a rate. Converting back to the original scale, the estimated change in incidence rate per a change, c, in exposure concentration (to ozone or $PM_{2.5}$) would be $e^{bc} - 1$, where e is the base of the natural log, e = 2.71828 18284 59045 23536….

Thus, to compute the change in the number of cases from a change, c, in concentration, we compute

$$\Delta\text{cases} = \text{baseline incidence} \times (e^{bc} - 1) \qquad (4.2)$$

The baseline incidence might be the number of annual deaths, for example.

Alternatively, we may know the incidence *rate*, the incidence per so many in the population, in which case the formula becomes:

Δcases = population x (baseline incidence rate) x $(e^{bc} - 1)$ (4.3)

Note that the "population" may be some age-subset. For example, if the endpoint is school absences, then the population are those 5-17 years old. A number of the other health effects are based on subsets of the population.

Logistic: This applies to those health studies that used logistic regression in the analysis. The C-R formula is:

Δcases = population x (baseline incidence rate) x $(e^{bc} - 1)/(1 + f)$ (4.4)

where $f = e^{bc} \times y_0 /(1 - y_0)$ and y_0 is the baseline incidence rate.

4.3 C-R Functions for Cancer

For cancer from toxics, the C-R function is different. The change in cancer rates is expressed as a risk, b, of an individual getting cancer from a compound from a lifetime (70-year) constant exposure to the compound. Thus the annual number of cases caused/reduced by a given change in average concentration, c, would be:

Δcases = population x b/70 x c formula (4.5)

4.4 Population Data

We use population projections by county.

4.5 Incidence and Incidence Rates

For most health effects we require baseline incidence rates, namely the annual population frequency of a particular health outcome. For this methodology, we were able to obtain some Bay Area county-specific data. Otherwise, we relied on incidence rates from previous health-benefit studies.

For mortality, we used county-by-county annual total non-accidental mortality to county residents, averaging 2011-13, the three most recent years available.

For hospitalization rates and asthma emergency room visits, we obtained 2011-13 county-by-county rates from the California Office of Statewide Planning and Development (OSHPD), using the averages of the three years' data.

Rates for non-fatal myocardial infarctions (MIs) were computed at the national level starting with the National Hospital Discharge Survey for 2010, and adjusting for hospital transfers and miscoding. The number of fatal MIs were multiplied by 1.29 to account for the difference

between in-hospital fatality to fatality within 30 days of the event (Coxson 2009). Data were available in 10-year increments. These were multiplied by the appropriate age ranges to get a population incidence rate. Our rates average 2 non-fatal MIs per 1,000, compared with 3 per thousand in BenMAP (Table D-5, US EPA 2012).

For school absence rates, we used a recent San Francisco Unified School District figure of 4.7% per day. (SFUSD 2009)

4.6 Health Effects used in this Methodology

As mentioned in the introduction, a number of health benefit studies have used an approach similar to that used here to investigate the benefits of reducing ozone and $PM_{2.5}$. Among these studies is a near-consensus on which epidemiological studies to use and how to use them. For $PM_{2.5}$, our methodology includes most of those listed in Appendix E of the BENMAP User Manual (US EPA 2012). We also use the BENMAP coefficients, uncertainty estimates, and C-R functions.

4.6.1 $PM_{2.5}$ – Mortality C-R function

The one exception is for mortality, where we use an estimate based on the mean of the median C-R estimates from 12 experts (US EPA 2006a). Specifically, each expert provided a probability distribution that summarized his/her judgement of the magnitude of the $PM_{2.5}$ – mortality C-R effect. Only one, expert K, assumed an effects threshold existed. Several others believed that the C-R effect was somewhat less for lower $PM_{2.5}$ concentrations. To combine these expert probability distributions, we averaged them, using the more conservative (lesser slope) C-R function when an expert provided more than one. We also incorporated the experts' probabilities that the $PM_{2.5}$ – mortality relation was not causal.

The result is a mixture distribution. It has a point mass of 12.5% at zero and a roughly triangular shaped probability density function above that. The median, and also the mode is near a 1% increase in all-cause mortality to persons 30 and older for a 1 $\mu g/m^3$ increase in $PM_{2.5}$. Expert K also placed 100% of the mass of his distribution on values < 0.8% per 1 $\mu g/m^3$. Thus, no matter what probability distribution one uses for this expert, the median of the pooled distribution would not be reduced.

We believe that this C-R coefficient represents a reasonable estimate of the $PM_{2.5}$ – mortality effect. The experts each relied on a range of studies, but they all relied on 2 studies, the Harvard 6-cities study (Dockery *et al.* 1993, reanalyzed in Krewski et al. 2000) and the American Cancer Society study (Pope et al. 2002). The C-R functions from these two studies bracket 1% / 1$\mu g/m^3$. Although the ACS study is based on a huge sample – with participants in the hundreds of thousands – it has limitations. Its participants were self-selected with characteristics that differ systematically from the adult population in general. Another limitation is the error in population exposure estimates, where centrally monitored $PM_{2.5}$ is used as a surrogate. As discussed in section 3.2, this may well lead to a downward bias in the C-R function.

4.6.2 Other C-R functions

For ozone, we use the effects, C-R functions, coefficients and uncertainties from Ostro *et al.* 2006. For ozone and school absences, we used the same study, Gilliland *et al.* (2001), but analyzed it somewhat differently. See Appendix E.

For toxics, we use the unit risk values from OEHHA (2016). We note that these factors are the 95th percentiles of risk, so that the risks, estimated cancer cases and economic values are likely to be overestimated, perhaps by a factor of 2 (Salmon 2009).

Table 3 lists the health effects along with the C-R functions we adopted, the original studies serving as the basis for the functions, the population subset subject to the health effect, and the incidence rates and sources.

Table 3. Health Endpoint C-R Functions and Incidence

Health Effect	Original Study(s)	Population	Beta	Formula*	Incidence	Incidence source
$PM_{2.5}$			**% per 1 μg/m^3**			
Mortality	US EPA 2006a + our own summary	≥ 30	1.0	4.1	all non-accidental deaths by county of residence	California Department of Health Statistics
Chronic Bronchitis Onset	Abbey *et al.* 1995	≥ 27 (w/o bronchitis)	1.32	4.3	0.00378	Abbey *et al.*
COPD Hospital Admissions	Ito 2003 & Moolgavkar 2003	≥ 65	.116(.206) Ito 0.185 (.052) Moolgavkar	4.2	county-specific rates, 2011-13 Bay Area rate 0.0009	OSHPD**
COPD Hospital Admissions	Moolgavkar 2003	18-64	0.218	4.2	county-specific rates, 2011-13Bay Area rate 0.0061	OSHPD**
Pneumonia Hospital Admissions	Ito 2003	≥ 65	0.398	4.2	county-specific rates, 2011-13 Bay Area rate 0.0073	OSHPD**
Cardiovascular Hospital Admissions (less MI)	Moolgavkar 2003	≥ 65	0.158	4.2	county-specific rates, 2011-13 Bay Area rate 0.0282	OSHPD**
Cardiovascular Hospital Admissions (less MI)	Moolgavkar 2003	18-64	0.140	4.2	county-specific rates, 2011-13 Bay Area rate 0.0032	OSHPD**
Non-Fatal Heart Attacks	Peters *et al.* 2001	≥ 18	2.41	4.3	based on national data, 2010. The Bay Area average rate is 0.0021	NHDS public use data files, adjusted for 30 day survival.
Asthma Emergency Room Visits	Norris *et al.* 1999	< 18	1.653	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHPD**
Acute Bronchitis Episodes	Dockery *et al.* 1996	5-17	2.721	4.3	0.043 cases per child per year	American Lung Association 2002
Upper Respiratory Symptom Days	Pope *et al.* 1991	Asthmatic children 5-17	0.36	4.3	124.8	California Center for Health Statistics reported that in 2003, 14.8% of children and adolescents in California had been diagnosed with asthma

Lower Respiratory Symptom Days	Schwartz & Neas 2000	7-17	0.6	4.3	0.438	Schwartz *et al.* (1994,Table 2)
Work Loss Days	Ostro 1987	18-64	0.46	4.2	2.17	Adams *et al.* 1999
Minor Restricted Activity Days	Ostro & Rothschild 1989	≥ 18	0.741	4.2	7.8	Ostro & Rothschild 1989
Ozone			**% per ppb 1-hr max ozone**			
Mortality	Ostro 2006	All ages	0.04	4.2	all non-accidental deaths by zip of residence	California Department of Health Statistics
Hospital Admissions for Respiratory Diseases	Thurston & Ito 1999	All ages	0.16	4.2	county-specific rates, 2007 Bay Area rate 0.0025	OSHPD**
Asthma Emergency Room Visits	Ostro 2006	< 18	0.24	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHPD**
School Loss Days	Gilliland *et al.* 2001	5-17	1.98	4.2	SFUSD rates	SFUSD 2009
Minor Restricted Activity Days	Ostro & Rothschild 1989	≥ 18	0.22	4.2	7.8	Ostro & Rothschild 1989
Toxics			**lifetime risk / $\mu g/m^3$**			
Lung Cancer (DPM)	OEHHA 2005	all ages	.0003	4.4	NA	NA
Leukemia (1,3-butadiene)	OEHHA 2005	all ages	.00017	4.4	NA	NA
Leukemia (benzene)	OEHHA 2005	all ages	.000029	4.4	NA	NA
Cancer – various sites (acetaldehyde)	OEHHA 2005	all ages	.0000027	4.4	NA	NA
Cancer – various sites (formaldehyde)	OEHHA 2005	all ages	.000006	4.4	NA	NA

* See formulas in text above.

** OSHPD = California Office of Statewide Health Planning and Development.

4.7 Calculation of Change in Incidence

Estimates of the changes in incidence of various health effects are made for each grid square, using grid square population and county- or Bay Area-level incidence rates, then summed to get county and Bay Area totals. For example, consider asthma emergency room visits. Suppose a control measure would reduce directly-emitted $PM_{2.5}$. We apply the results of Table 3 and Section 2 as follows. For a given grid square, i, the change in $PM_{2.5}$ concentration, Δc_i, is computed. This is combined with the effect coefficient, 0.0165 to compute the exponential part of formula 4.3, The incidence rates differ by county; for Alameda grid squares for example, the incidence rate is 0.0067. So for an Alameda grid square, we would combine this with the estimated 0-17 year-old population, p_i, to produce

$$p_i * 0.0067 * (e^{0.0165*\Delta ci} - 1)$$

that is, the estimated reduction in the annual number of asthma emergency room visits among 0-17 year-olds with residences within grid square i. These values are then summed by county.

There were several variations on this approach, depending on health endpoint.

4.7.1 School absences.

We follow the approach in Hall (2008) to take into account summer vacations, weekends, holidays, etc. See Appendix E for details.

5. Valuation of Health Effects and Greenhouse Gas Reductions

The last step in the methodology is to estimate the economic value of pollution reductions in terms of decreased health and social costs. The goal is to establish whenever possible not just the direct costs of illness, such as hospitalization and medications, but the value placed by individuals on avoiding the illness. This incorporates concerns such as:

- Loss of productive time (work and school)
- Direct medical costs that result from avoiding or responding to adverse health effects
- The pain, inconvenience and anxiety that result from adverse effects, or efforts to avoid or treat these effects
- Loss of enjoyment and leisure time
- Adverse effects on others (family, friends, caregivers, etc.) resulting from their own adverse health effects (Hall 2008).

The following section, 5.1, discusses the methods applied to value the social benefits of air pollution reduction. It is quoted directly from an excellent discussion in Hall 2008.

5.1 Concepts and Measures of Value

"Ideal measures of value would represent all of the losses that result from adverse health effects. They would also accurately reflect real preferences and decision-making processes similar to those we use to make basic choices every day. Our decisions about which goods or services to buy are based on which items give the most satisfaction, or utility, relative to prices and income. Market prices are therefore accepted as reasonable measures of the value of those items that can be purchased. However, there is no market in which cleaner air (like many other environmental goods) can be bought. Consequently, values for such goods cannot be directly observed from prices. Economists have developed alternatives to market prices to measure the value of environmental improvements, including health benefits resulting from cleaner air.

"Generally accepted measures of the value of changes in well-being due to reducing the adverse health effects of air pollution include the cost of illness (COI) measure and the willingness to pay (WTP) or willingness to accept (WTA) measures. All three measures have limitations but, when taken together, they yield a generally accepted range of values for the health benefits of improvements in air quality. In this study, we use the most appropriate available value for each health endpoint."

5.1.1 Cost of Illness

"The cost of illness (COI) method was the first to be developed and described in the health and safety literature as a basis to value reductions in risk. It requires calculating the actual direct expenditures on medical costs, plus indirect costs (usually lost wages), incurred due to illness. This method is still the primary measure used to value the benefit of avoiding hospital admissions and other medical treatments. The COI method has the advantage of being based on real dollars spent to treat specific health effects and the actual market value of work time. Since it includes only monetary losses, however, and does not include losses associated with the value of leisure time, of school or unpaid work time, or of general misery, it does not capture all of the benefits of better health. The method is therefore generally viewed as limited and representing a lower bound on value. The basic limitation is that it is a measure of the *financial* impact of illness, not the *change in well being* due to illness, since financial loss is only part of the value forfeited by illness and discomfort. Other factors associated with illness, most notably pain, inconvenience, and anxiety, can result in a significant disparity between COI estimates and WTP (or WTA) estimates. As discussed below, the COI approach has been shown to produce a lower-bound value estimate. Overall, COI measures are used when more complete measures are unavailable for a specific effect. While they generally represent a lower bound of value, using them allows the valuation of some adverse effects, such as emergency room visits, which might otherwise not be quantified."

5.1.2 Willingness to Pay and Willingness to Avoid

"Because we know that COI measures undervalue adverse health effects, many studies have been conducted to determine more complete values. For improvements in health, for

example, we use WTP measures, which are both more complete than COI and consistent with accepted economic concepts about markets and individual economic choices. Market choices that reduce risks to health or life indirectly indicate the WTP for lower risks, or the WTA for higher risks. Values derived from these market-based methods are based on relating differences in wages or consumer costs to differing degrees of risk. Those differences indicate the demand for and the WTP for lower risk, or the WTA for greater risk. Because air quality is not a market commodity and has no observable market price, many of the values used in benefit assessments for environmental improvements depend on studies of market-determined wage differentials and consumer expenditures in relation to lower risk of harm from other causes. These differentials and expenditures are then surrogates for the market price for reduced risk of harm from air pollution.

"There is an extensive economics literature assessing the value of reduced workplace risk of death. It is, however, important to control for factors other than risk that can influence wage differentials, such as unpleasant working conditions. Studies conducted in the past 20 years do control carefully for job attributes that are not related to differences in risk (Viscusi 1992, 1993, 2004; Viscusi and Aldy 2003). There is a smaller literature that investigates differences in consumer expenditures relative to risk of injury or death associated with product use. The results for the most carefully conducted work, which controls for product characteristics other than relative risk, are generally consistent with the wage-risk studies (Atkinson and Halvorsen 1990; Viscusi 1992). Finally, there are several “meta-analyses” that assess the value of reduced risk based on statistical amalgamation of multiple underlying studies."

5.1.3 Contingent Valuation

"When values inferred from markets are not available, another means to estimate value involves the use of surveys. This method is referred to as contingent valuation (CV) because people are asked to determine what something would be worth to them *as if* they were able to purchase or sell it. CV has become a significant source of values over the past two decades, as the methodology has matured and become more accepted, and as policy-makers (and the courts) have become more engaged with the application of economic values to decision-making. CV-based values, as with wage-risk based WTA values, are conceptually better than COI because they are more inclusive. Respondents can value loss of enjoyment and discomfort, as well as the direct costs of an adverse health effect. The survey approach is, however, expensive to administer and the validity of values derived from this method depends on careful design and application of the survey instrument. Nonetheless, CV measures are in many cases well supported and add useful information to benefits assessment (Carson et al. 2001)."

5.2 Health Valuations used in this Methodology

Health valuations were combined from several studies: US EPA (2012), Hall (2008), McCubbin and Delucchi (1996), and Stratus (2008). Valuations were adjusted for the metropolitan Bay Area Consumer Price Index, and also for prevailing wage rates, where applicable.

Table 4 lists the valuations by health endpoint. Willingness-to-pay measures were used where possible, otherwise cost of illness. The costs per incident are listed, ranging from a willingness to pay $25 to avoid a day of lower respiratory symptoms, to $8,800,000 to avoid a death.

Table 4. Unit Values Used for Monetary Estimates for Quantified Health Effects (2015 dollars)

Health Effect	Unit Value (Cost per Incident)	Type of Measure	Derivation of Estimate Explanation
Mortality (all ages)	$8,800,000	WTP	Using EPA (2010), the mean value of avoiding one statistical death is assumed to be $6.3 million in 2000 dollars. This unit is the mean value based on meta analyses of the wage risk value of a statistical life (VSL), applying a Weibull distribution to the values in the range. This method is similar to Hall et al (2008) and Stratus (2008). The 2000 value was adjusted to 2014 dollars using the CPI for all Bay Area urban customers. US EPA 2012 assumed a confidence interval between $1.25 million and $12.5 (in 2008 dollars) based on two meta analyses of the wage risk VSL literature. The lower bound estimate is based on Mrozek and Taylor (2002) and the upper bound interval is based on a meta analyses by Viscusi and Aldy (2003).
Chronic Bronchitis Onset	$476,117	WTP	US EPA 2012 provides estimated values for the reduction in risk of CB (I.2.1 -- Appendix I). The best estimates of WTP avoid a case of CB comes from Viscusi et al (1991) and Krupnick and Croper (1990). Their adjusted pooled estimate for an avoidance of CB is $340,482 (in 2000 dollars). Adjusting for the CPI yields $476,117 in 2014 dollars.
Respiratory Hospital Admissions	Age 65 < : $55,305 Age 65 > : $48,901	WTP + Third Part COI	The unit values were derived using Hall et al (2008) and from the estimates by Chestnut et al (2006) and adjusting to region specific CPI.
Cardiovascular Hospital Admissions	Age 65 < : $65,178 Age 65 > : $56,060	WTP + Third Part COI	The unit values were derived using Hall et al (2008) and from the estimates by Chestnut et al (2006) and adjusting to region specific CPI.
Non-Fatal Heart Attacks	$82,580	COI	There are no WTP values for the reduction of nonfatal heart attacks, Hall et al (2008) turn to COI estimates (Eisenstein et all 2001; Russell et al 2001) and opportunity costs estimates (Cropper and Krupnick 1990) in order to derive a value for non-fatal heart attacks. They derive a value of $70,103 (2007$). The value in the table is updated to 2014$ using the CPI for the Bay Area for the wage portion and the medical CPI for the COI portion.
Asthma Emergency Room Visits	$478	COI	Using US EPA 2012, I.3.2 and updating Smith et al (1997) where they report the average cost per emergency room visit made in 1987. Updating to 2014 CPI-U for medical care, the estimate is $478.
Acute Bronchitis Episodes	$598, for a 6 day illness period	WTP	US EPA 2012 reports estimates of WTP based on preventing respiratory symptoms caused by acute bronchitis. They assume a 6 day illness period, with a 6 day WTP of $356 in 2000$. The value shown uses the CPI-U for medical care to adjust to 2014$.
Upper Respiratory Symptom Days	$40	WTP	Hall 2008 estimated $35 per day in 2007$. Adjusted to 2014$ using Bay Area CPI.
Lower Respiratory Symptom Days	$25	WTP	Hall 2008 estimated $22 per day in 2007$. Adjusted to 2014$ using Bay Area CPI.

Work Loss Days	Daily Median Wage by County;	COI	Stratus (2008) note that there are no available estimates of WTP for preventing a day of lost work due to illness. Therefore, the point estimate value is based on county specific median daily wage. All figures are from Employment Development Department *Occupational Employment Statistics Survey(1st quarter 2014 wages)* Alameda, & Contra Costa: $232 Marin, San Francisco, & San Mateo: $205 Santa Clara: $278 Napa: $186 Solano: $196 Sonoma: $191
School Absence Days	$103	COI	Following US EPA 2005, the value of a school absence is the estimated daily lost wages for women over age 25. Using BLS data (Women in the Labor Force: A Databook 2008) we find that the weekly median age for women over 25 in 2007 was $614, thus the estimated daily median wage $123. The labor force participation rate for women over 25 with children under the age of 18 was .713. Thus, .713*$123=$88 (in 2007$) or $91 in 2008$ (Where $91 is the lost productivity at the female's parent's wage. This is with the assumption that if a child stays home from school, a working mother will have to stay home from work to take care of the child.)
Minor Restricted Activity Days	$85	WTP	Values from EPA 2012, Appendix I for 2000$, adjusted for Bay Area CPI increase.
Cancer	$3,700,000	WTP+COI	McCubbin et al (1996) choose $,7 million (2008 dollars) as a lower bound estimate of the cost of a non-fatal cancer, and $2.8 million (2008 dollars) for an upper bound estimate (with a mean of $1.75 million in 2008 dollars). These figures where estimated from a literature review completed by McCubbin et al (1996). The lower and upper bound include all costs of cancer, including medical costs, pain and suffering to both patients and friends, and the loss of production to society.

*All values have been adjusted to 2015 dollars and adjusted to Bay Area values where data are available. (CPI-U for SF-Oakland-San Jose)

5.3 Valuation of Greenhouse Gas Reductions

Gases that contribute to climate change and global warming (GHGs) are one of the four categories of pollutants specifically targeted in the 2016 Clean Air Plan (CAP). Therefore, in addition to the value of health benefits from reducing ozone, PM, and air toxics, we include in the MPEM an estimated value of the social benefit of GHG reductions; specifically, the benefit of reducing one ton of GHG (CO_2-e). This section describes key issues related to valuing GHG reductions, and explains how we went about selecting a reasonable GHG value for the MPEM.

MPEM uses $62 per ton of CO_2 to value reductions in CO_2 or the CO_2 equivalent for the other Kyoto 6 GHGs, developed by the Interagency Working Group on the Social Cost of Carbon (IWG 2015). It is important to note that this value does not necessarily include all potential impacts and costs related to global warming. Moreover, the study of climate change is extremely dynamic; predictions of the potential impacts seem to become more serious and better documented with each passing month. Therefore, it is likely that the GHG value that we use for the MPEM may prove in retrospect to be conservative.

Key issues discussed below include:

- Which GHGs to include?
- The range of uncertainties in estimating the value of GHG reductions
- What GHG valuation method to use?
- What discount rate to use?
- Which value to use?

5.3.1 GHGs included

For purposes of the MPEM, we consider only emissions of the "Kyoto Six" GHGs.[13] These gases vary significantly in terms of the volume (mass) of emissions as well as their specific global warming potential (GWP) expressed on a CO_2-equivalent (CO_2-e) basis. It should be noted that, to the extent that 2016 CAP control measures may reduce emissions of other (non-Kyoto Six) GHGs, or other pollutants such as black carbon that are not included in our calculations, the MPEM may underestimate the benefit of control measures in protecting our climate.

[13] The Kyoto Six GHGs are CO2, methane, nitrous oxide, hydrofluorocarbons, perfluorocarbons, and sulfur hexafluoride.

5.3.2 Uncertainties

The estimated value of GHG reductions is subject to many factors and difficult to establish due to the long time frame and wide range of impacts associated with climate change. Although we are already experiencing some impacts that can be tied directly to human-induced climate change[14], the full range and scale of its effects will not be felt until far into the future. Key questions include:

- At what level should CO2 concentrations be stabilized?
- How great a reduction in GHG emissions would be required to achieve the stabilized level?
- How fast can we and should we move to reduce GHG emissions and radiative forcing? Is it better to front-load the reductions, or defer the deeper reductions into the future?
- How can our moral obligation to future generations be expressed in economic terms?
- What assumptions should we use regarding future economic growth? At what rate should we assume that future technological advances will help to reduce GHG emissions?

In light of these uncertainties, the value of GHG reductions, like other elements of the MPEM, is analyzed as part of the probability analysis (see Fairley 2010) performed to estimate the uncertainty of our estimates for the methodology as a whole.

5.3.3 GHG Valuation Methods

There are three basic approaches commonly used to monetize the value of GHG reductions: market price of carbon,[15] marginal abatement cost of carbon, and social cost of carbon.

The market price of carbon (MPC) uses current market prices in carbon trading schemes, such as the European Emissions Trading Scheme.[16] MPC reflects the current price for carbon trading or carbon emission offset purposes. An advantage to a carbon market price is that it provides a *real* price that can aid business decisions. However, the *price* of carbon has no direct connection to the social *cost*, and thus the MPC is not appropriate to the purposes of the MPEM.

The marginal abatement cost of carbon (MAC) is defined as the cost involved in preventing the emission of one additional unit of carbon (or CO2-e). As in the case of MPC, MAC is based on the cost of reducing a unit of carbon emissions, rather than the social and economic costs of climate change impacts. Therefore, MAC, like MPC, is not an appropriate method for purposes of the MPEM.

[14] The US Science Change Program summarizes many of the changes already taking place, including a rise of 2°F over the US, extreme weather has increased including heat waves, droughts, and hurricane intensity. USGCRP (2009) Studies have documented an increase in wildfires in California. Wildfires in the summer of 2008 caused the Bay Area to violate the national 24-hour $PM_{2.5}$ on several days.

[15] Some studies express the value of GHG reductions in terms of CO2-equivalent; other studies talk in terms of carbon reductions. For purposes of the MPEM, we use CO2-e. CO2-e can be converted to carbon by multiplying the ratio of their atomic masses (12/44). A value of $44 per ton of carbon would equate to $12 per ton for CO2-e.

[16] See http://www.ecx.eu/

The social cost of carbon (SCC) is defined as the "total damage from now into the indefinite future of emitting an extra unit of carbon" (Stern 2006). SCC attempts to monetize the costs to society of a ton of carbon emissions. The key advantage of SCC is that it attempts to capture the total costs to society of a wide range of climate change impacts, including impacts on public health, the environment, and societal disruption such as after a major weather disaster. For this reason, we rely on the social cost of carbon method for the MPEM. It should be noted, however, that the effort to quantify a wide range of climate change impacts does introduce greater uncertainty in SCC estimates compared to MPC or MAC.

5.3.4 Discount Rate

Future generations will bear the burden of the greenhouse gases that we emit today. GHGs vary in terms of their lifespan in the atmosphere and the length of time they will cause climate change. Impacts from current emissions of the primary GHG, CO_2, will not be fully realized for more than one hundred years. Thus, the full costs of today's GHG emissions will not be felt until far into the future.

A crucial issue in determining a value for GHG emissions is how to value, in today's dollars, the benefit of avoiding climate change impacts that will not be fully experienced until centuries to come. Empirical evidence suggests that humans value future benefits less than present benefits. Therefore, economists apply a *discount rate* to put a price in current dollars on goods or benefits that will be consumed at some future date. The selection of a discount rate is a critical factor in determining the value of GHG emissions. The current value of avoiding future climate change impacts can be large or small depending on one's choice of discount rate.

Although discount rates are well suited for projects or analyses with a near or moderate term time frame (say, 50 years or less), it is very difficult to determine an appropriate discount rate for an issue such as climate change with a very long timeframe. Applying typical discount rates (e.g., 3% to 7%) on a constant basis to events in the far future essentially would reduce the value of future benefits to near zero in today's dollars, but this would raise ethical issues since putting a near-zero value on future benefits suggests that as a society we do not care about the future beyond another generation or two. Our valuation of $62 is based on a discount rate of 2.5%.

6. Potential future enhancements to the MPEM

The MPEM was developed within the constraints of the available information, tools, and time. This section discusses a number of ways the method might be improved. Some of these enhancements can be performed in-house, whereas others would require improved information from external sources.

6.1 In-house enhancements

Spatial distribution of emissions reductions: For the 2015 MPEM we assume that emissions reductions from control measures will be geographically distributed on the same basis as the overall emissions inventory. It would require more modeling runs, but it would be more accurate from the standpoint of estimated population exposure and health outcomes to estimate the geographic distribution of emission reductions for key control measures based upon the location of the sources that would be impacted by the measure.

Temporal distribution of emissions reductions: For the 2015 MPEM we assume that emissions reductions from control measures are constant throughout the year. Some controls (e.g., wood burning controls) vary considerably by season. This would require evaluating emission reductions by season.

Distribution of emissions by source: Currently, the emissions used in the model are pooled by species. It would be more accurate to disaggregate emissions by source and reduce precisely the sources affected by the key control measures. Again, this would require separate modeling runs for individual source categories.

Population exposure: For the 2015 MPEM we assume population exposure based upon "backyard" exposure; i.e., we assume that people are at home, outside in their yards on a 24/7 basis. One approach for making more realistic exposure estimates would be to develop (or find) more accurate data as to daily individual activity patterns by, for example, having a random sample of Bay Area residents fill out diaries of daily activities. This approach would also require monitoring and modeling of micro-environments such as in homes, offices, cars, parking garages, schools, etc. An alternative approach would be to outfit a random sample of residents with personal monitors to measure the pollutants of interest. Ideally, these more accurate exposure values would be used to estimate new health endpoint values.

Wider range of exposure: Expand the population domain to areas outside the Bay Area that are affected by Bay Area pollution.

Wider range of pollutants: For the 2015 MPEM we considered ozone, PM, air toxics, and greenhouse gases (GHGs). For toxics, we included only the five toxics that collectively account for an estimated > 90% of the cancer risk from air toxics. For GHGs, we included only the "Kyoto 6" gases. Recent research suggests that black carbon (soot) may be a major contributor to global warming. Studies indicate that ultrafine particles (UFP) may have a large impact on health independent of PM2.5 as a whole.

Morbidity from toxics: For the 2015 MPEM, we considered key toxic carcinogens, but did not include non-carcinogenic effects. For example, there is a significant amount of ambient acrolein, an eye nose and throat irritant, in the Bay Area.

Improved modeling: Modeling results are crucial to the MPEM. Modeling refinements that would improve the accuracy of the MPEM include:

- Use decoupled direct method to obtain more accurate sensitivities (especially for small changes in emissions).
- Current models offer averages within areas of a square kilometer or greater. Potentially, neighborhood-scale models could be developed that estimate pollution levels for individual street blocks.

Speciate VOCs: There are many volatile organic compounds and these VOCs vary in terms of their reactivity; i.e., their ozone-forming potential. We could speciate the VOCs for each inventory source category and apply the speciation to the emission reduction estimates in order to more accurately estimate the ozone reduction potential for each control measure.

Bay Area-specific health studies: The dose-response values used in the MPEM are based on studies from other areas and only rough approximations of exposures. If the District conducts studies that make major improvements in population exposure estimates, these might be used to provide input for new studies of dose-response values that would be Bay Area-specific and tailored to the enhanced exposure estimates.

6.2 Enhancements based on External Information

Wider range of health effects: For the 2015 MPEM we used a limited set of health effects from established, peer-reviewed studies. As additional studies are performed that better document the full range of health effects from air pollution, we should incorporate these additional health effects in the MPEM. For air toxics in the 2015 MPEM, we included only cancer-related costs. It would be more accurate to include acute and chronic non-cancer (as well as cancer) effects for air toxics in the future.

More specificity on PM health effects: Recent analysis suggests ultrafine PM may affect health more than larger particles. There is also current research on the relative health impact of different components of PM, such as elemental carbon (EC), organic carbon (OC), ammonium nitrate, and ammonium sulfate. We will monitor this research and incorporate new results as the scientific consensus warrants.

Environmental and ecosystem impacts: We estimated costs and benefits in the 2015 MPEM for certain health effects, as well as for the social cost of greenhouse gas emissions. While these costs are very important, they do not capture the full range of impacts from air pollution. We should attempt to include costs for a wider range of environmental and ecosystem impacts in the future, including water pollution, the impacts of reactive nitrogen on ecosystems, etc.

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Appendix A

Trend analysis for toxics, $PM_{2.5}$ and ozone concentrations

A key step in the MPEM is to estimate how a given change in emissions will affect concentrations. The grid models used here provide an estimate for a single (and relatively large) reduction in emissions (10% on some runs or 20% in others). But what is needed is a function that relates changes in emissions from various rules to changes in concentrations, that is, a formula that says how the concentrations will change if the emissions are reduced by 7% or 2% or 0.6% or increased by 3%. One way to get additional information and provide a dose of reality is to see how concentrations have changed historically with changes in emissions.

The simplest assumption about how emissions reductions affect concentrations is a proportional rollback where, for example, if emissions of a pollutant were reduced 10%, then its concentrations would also be reduced by 10%. More general is a rollback model where concentrations are reduced linearly, but not by the same percentage. Except for ozone, which is discussed below, the pollutants considered here have experienced trends that are consistent with a linear rollback scenario.

Pollutants do not always behave simply for several reasons. First, the concentrations of a pollutant may include natural background concentrations. There is a global background for ozone ranging from 20 ppb to 40 ppb that is at least partly natural – the result of intrusion of ozone from the stratosphere. There is natural $PM_{2.5}$ also, including windblown dust and organic $PM_{2.5}$ formed from gases like benzo-a-pyrene emitted by certain plants and trees. In addition, the Bay Area gets significant amounts of oceanic background $PM_{2.5}$, namely sea salt and sulfate. Although CARB estimates California $PM_{2.5}$ background at 2.5 $\mu g/m^3$ (Motallebi et al. 2003), oceanic sea salt increases the background for the Bay Area to perhaps 3.5 $\mu g/m^3$.

Second, anthropogenic pollutants may be transported from other areas. It is likely, for example, that during some winter periods when the winds are easterly, the Bay Area receives various types of pollution from the Central Valley.

Third, pollutants like ozone and ammonium nitrate are formed through complex atmospheric transformations from other precursor compounds where transformation rates depend on the relative amounts of the precursors, as well as atmospheric and meteorological conditions.

Fortunately, it appears that, except for ozone and secondary PM, the pollutants considered here have experienced trends that are consistent with a simple rollback scenario.

Shown below are plots that depict how a whole distribution has changed over time. Sometimes called a *quantile-quantile* or *q-q plot*, two distributions are plotted against each other, with the percentiles of one matched to the percentiles of the other.

Trends in Benzene and 1,3-Butadiene at San Jose

In Figure A1, San Jose's benzene concentration data for 2003-07 has been compared with its 1987-91 benzene data. Due to the limited number of data points*, every 5th percentile is plotted: 5th, 10th, 15th, and so on up through 95th. For example, the 5th percentiles were roughly 0.1 ppb for 2003-07 vs. 0.9 ppb for 1987-91. The 95th percentile was reduced from about 8.7 ppb to 1.5 ppb between the two periods. If there had been no change in the distribution, then the percentiles would have fallen near the line y=x (shown). Instead, the percentiles fall near the line y = 0.155x, i.e., the 2003-07 percentiles are around 15.5% of the 1987-91 percentiles. In other words, there has been an across-the-board reduction in benzene concentrations by a factor of between 6 and 7. Thus, despite the caveats discussed above, the trend is consistent with linear rollback.

Modeling results back this assumption. A regression based on a grid cell-by-grid cell comparison of the effects of a 10% reduction in benzene resulted in a slope of 0.900 and an adjusted R^2 of 100%.

Figure A2 shows a similar plot for 1,3-butadiene. Complete data started more recently so the base years were 1990-94 rather than 1987-91. The trend is again explicable with simple rollback. The slope in y = 0.23x implies an annual rate of decrease similar to that of benzene.

Again, modeling backs the rollback hypothesis, with a 10% reduction in 1,3-butadiene resulting in a regression with slope 0.900 and an R^2 of 100%.

$PM_{2.5}$ Trends at Livermore

Figure A3 shows the trend in $PM_{2.5}$ concentrations at Livermore, comparing measurements using a BAM (Beta Attenuation Monitor) from its first three years of operation, 2001-03, to 2006-08. Because there were more than 1,000 observations in each period, every percentile was computed from the 1st through the 99th.

The picture for $PM_{2.5}$ is somewhat more complex than for benzene and 1,3-butadiene. The $PM_{2.5}$ reduction does appear linear, but the rollback is not to zero because $PM_{2.5}$ has a natural background from vegetation, dust and sea salt from the ocean.

$PM_{2.5}$ values are also available from a site in the Point Reyes National Seashore. The local emissions are low, but analysis of its components shows that Point Reyes gets its $PM_{2.5}$ from the ocean, which includes sea salt and some ship emissions. Although not pristine background concentrations, the $PM_{2.5}$ concentrations at the Point Reyes site illustrate that it is not possible to reduce $PM_{2.5}$ concentrations to zero. They also illustrate that a natural background would itself not be constant, but rather would display a range of concentrations.

* The data are collected on a 1-in-12 day schedule. There were 152 observations in the 2003-07 period and 126 observations in the 1987-91 period.

The percentile lines for both Livermore 06-08 and Point Reyes 04-07 meet the line y=x at 1.1 $\mu g/m^3$. Thus, it appears that $PM_{2.5}$ is indeed being rolled back; however, it is being rolled back not to zero but to a background distribution.

Ozone Trends at Los Gatos

Figure A4 shows the trend in the distribution of Los Gatos May-October hourly ozone from 1991-95 to 2004-08* as a curve with diamonds. Note how the curve falls above the y=x line for percentiles up to the 90th. In other words, for 90% of May-October hours, Los Gatos ozone is higher today than in the early 1990s. However, consider the second curve, which is for Davenport, whose measurements represent the approximate oceanic background**. This curve lies above the other curve until almost the 70th percentile. In other words, 2/3 of Los Gatos hourly ozone has been and continues to be *below background*.

* May-October is the ozone season, the only months with ozone that might exceed national or California standards. The 1991-95 period was chosen because, before that, ozone had been recorded only to the nearest 10 ppb. This would have made comparing percentiles, which differ by only parts per billion, problematic.

** Measurements from Davenport, a coastal site in a tiny town north of Santa Cruz. Shown are hourly values for 2002-2006, the last year the data were available.

Figure A1. Trend in San Jose Benzene Distribution 2003-07 vs 1987-91

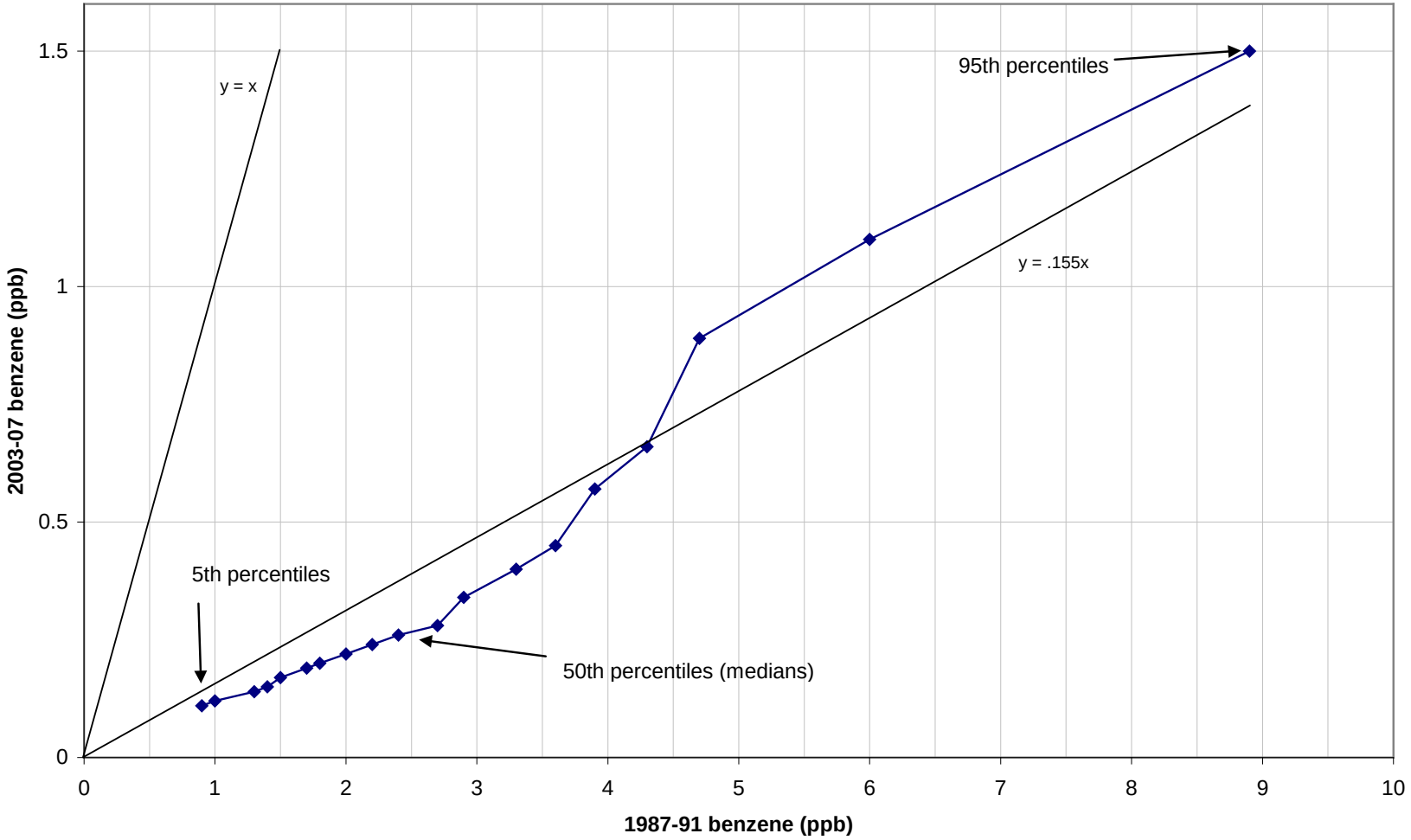


Figure A2. Trend in San Jose 1,3-Butadiene Distribution 2003-07 vs. 1990-94

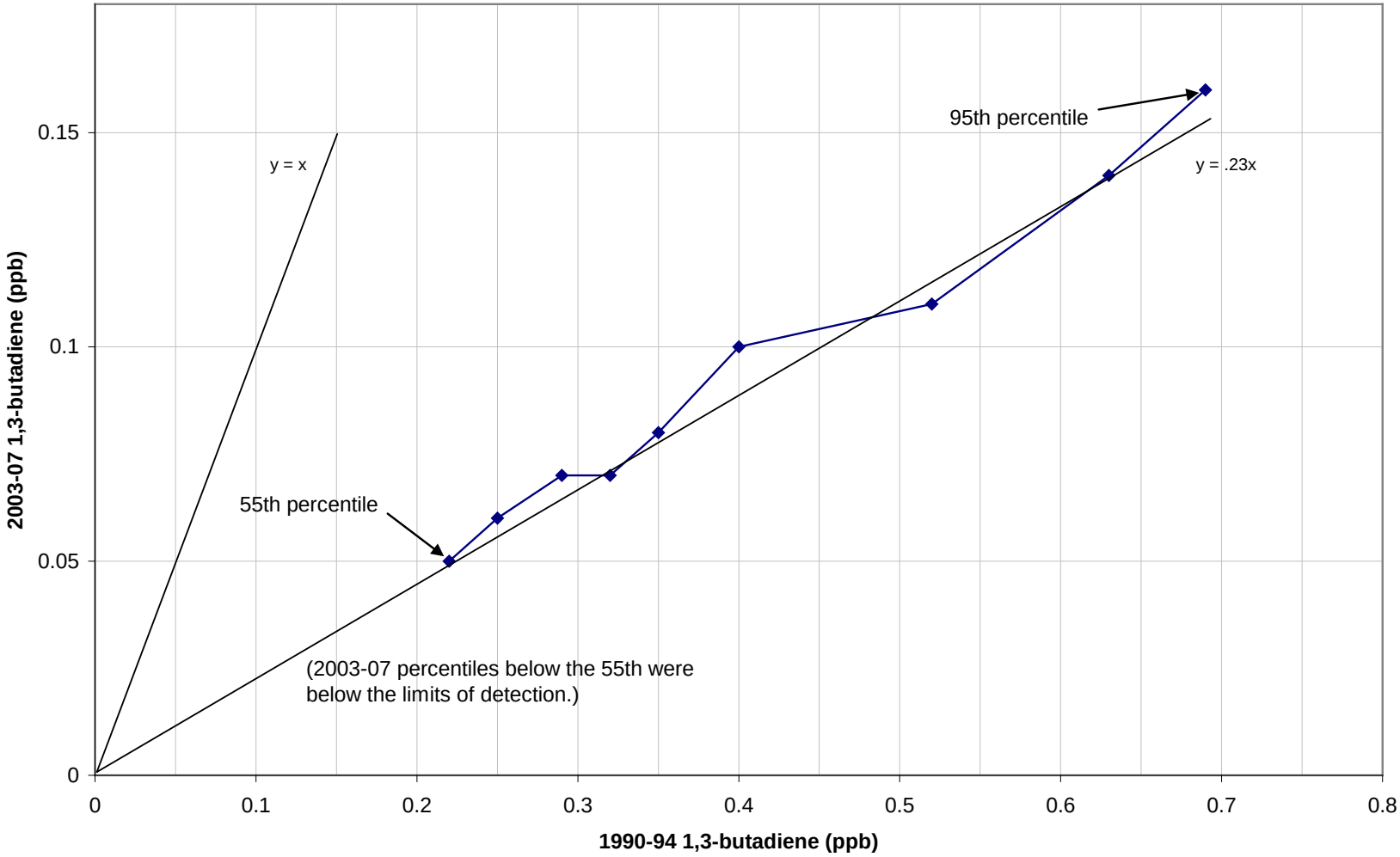
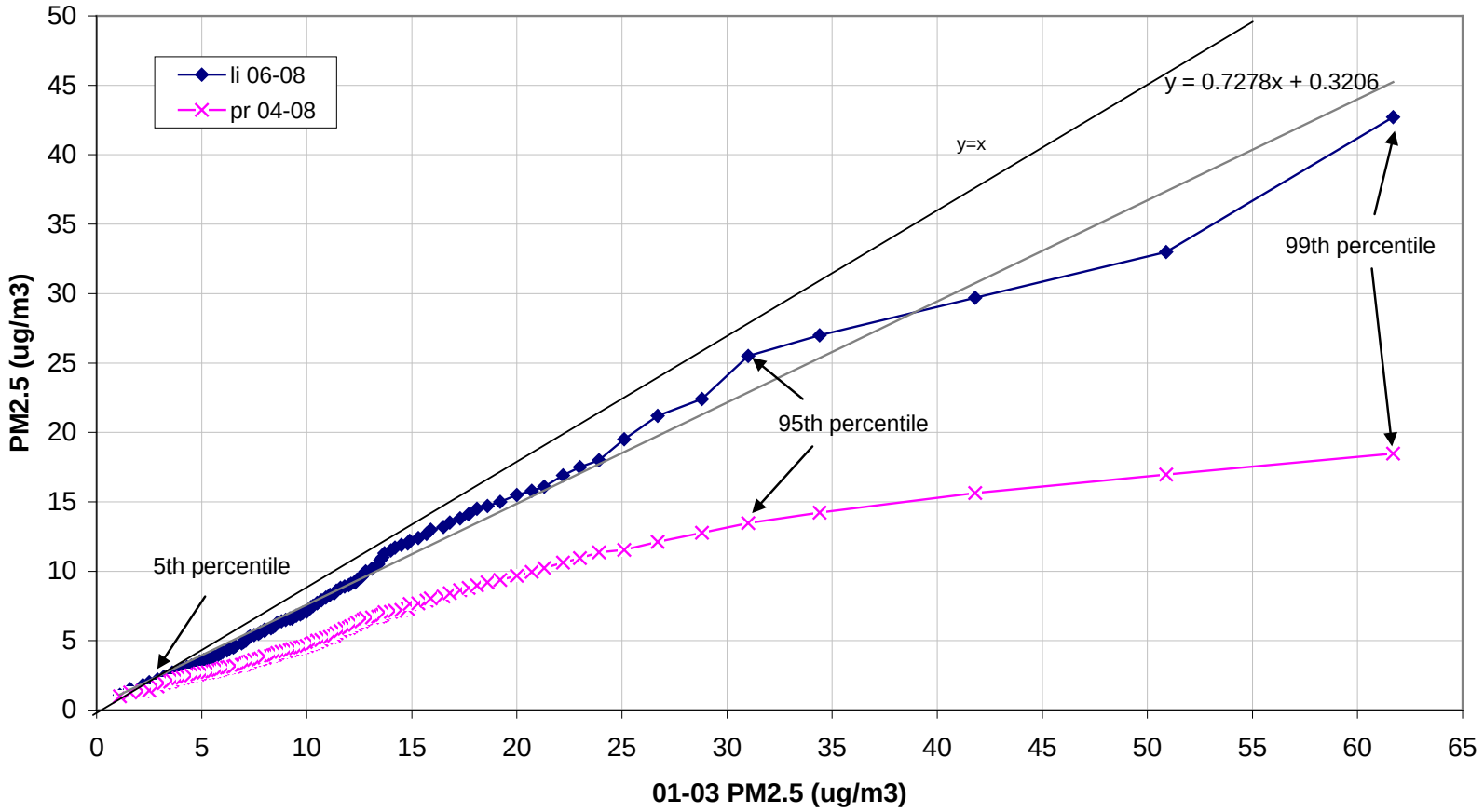
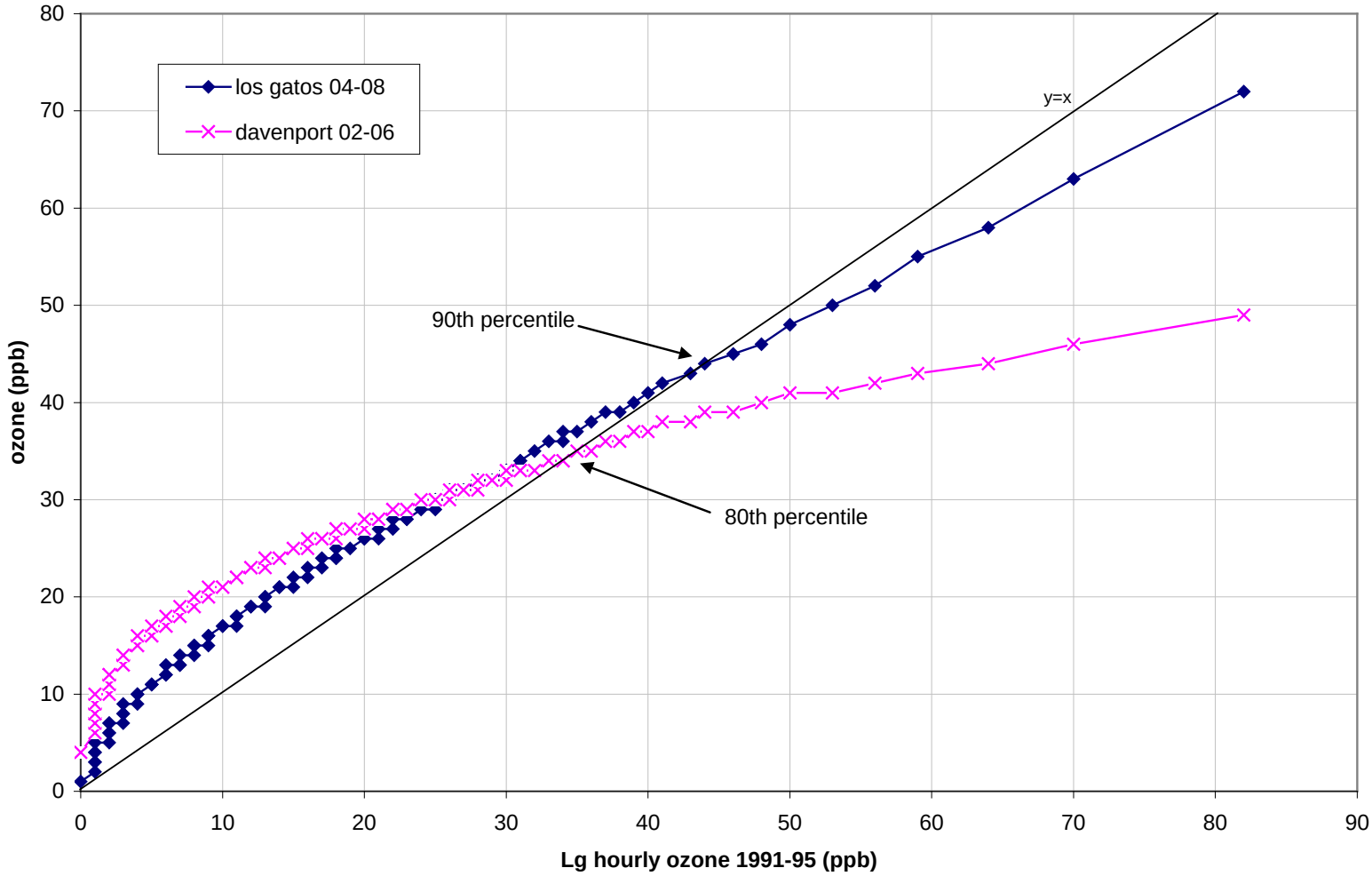


Figure A3. Trend in Livermore PM2.5 2006-08 vs 2001-03, and vs. Point Reyes "background"



Trends in the percentiles of Livermore PM2.5 concentrations. Shown are the 1st through 99th percentiles of the Livermore's 2006-2008 24-hour PM2.5 concentrations (as measured with its BAM unit) plotted vs. the corresponding 1st through 99th percentiles of its 2001-03 24-hour BAM concentrations. A y=x line is drawn to show where the %iles would lie if there were no trend. A trend line is shown. Also shown are the percentiles of the Point Reyes background site PM2.5 concentrations for 2004-08.

Figure A4. Trends in percentiles of Los Gatos and Davenport "background" May-October hourly ozone



Appendix B

Using an annual average to approximate the average effect of 24-hour values

Analyses investigating the relationship between $PM_{2.5}$ and health effects have largely used either daily time series, where an effect like daily hospital admissions is compared with daily 24-hour $PM_{2.5}$ concentrations, or annual mean $PM_{2.5}$ where, for example, mortality rates from different regions are compared against the corresponding annual $PM_{2.5}$ concentrations after the mortality rates have been adjusted for other factors such as age distribution, smoking rates, and so on.

In this methodology, we are using some of each kind of analysis. The mortality and chronic bronchitis effects are based on the latter studies using annual $PM_{2.5}$ concentrations. But the other effects are based on using daily concentrations. Thus, ideally, the impact of changes in $PM_{2.5}$ would be evaluated by summing over the changes in daily health effects.

However, for practical reasons we have not made estimates of $PM_{2.5}$ concentrations for individual days, but only for an annual average. For those health effects based on daily 24-hour concentrations, we make the approximation that the average of the daily effect (C-R) functions is equal to the daily effect function evaluated at the annual average $PM_{2.5}$ concentration. Symbolically, if $x_1, x_2, \ldots, x_{365}$ are the daily $PM_{2.5}$ concentrations and f(x) is the effect function, our assumption is:

$$\frac{1}{365}\sum_{i=1}^{365} f(x_i) \approx f(\bar{x}) \qquad \text{(C1)}$$

In most cases, $f(x) \propto e^{cx} - 1$. Although the goodness of approximation C1 depends on the magnitude and spread of the x_i's, we can say with certainty that it is an *under*-estimate, invoking Jensen's inequality.

As an example, consider acute MI. Using Peters (2001), the C-R function is proportional to $e^{.02412x} - 1$. Using $PM_{2.5}$ measured at San Jose, the values for 2007 for the left and right sides of equation C1 were .3834 and .3423, respectively. For 2008, the values in equation C1 were .3694 and .3407 respectively. These represent underestimates of about 11% in 2007 and 8% in 2008.

For the MPEM, however, the changes in $PM_{2.5}$ are much less than the total $PM_{2.5}$. As the values of the x_i approach 0, the approximation gets better. For example, if we divide San Jose's 2008 $PM_{2.5}$ values by 10, then the left side of C1 is 0.02995 compared with 0.02975 on the right, an underestimate of less than 1% and similarly for 2007. This is because $e^x \cong 1 + x$ for x near 0.

Appendix C

Ozone threshold and the adjustment of the regression slope

Statistically significant relationships have been found between ozone concentrations and a number of health effects, but questions remain about whether the effect is constant over the entire ozone range or if there may be an effects threshold. For the MPEM, we in fact assume a threshold of 50 ppb. This appendix briefly discusses the evidence for a threshold and a method to adjust C-R functions to account for a threshold for studies where none was assumed.

A number of studies have found that ozone effects are greater for higher ozone concentrations. Ostro *et al.* examined several studies (Stieb *et al.* 1996, Tolbert *et al.* 2000, Romieu *et al.* 1995) and estimated the potential impact of thresholds in several sensitivity analyses, including one where they analyzed the effect of a threshold of 50 ppb (in 8-hr max ozone) and a 100% increase in health effects coefficients. Analysis of the impact of ozone on mortality shows that statistical models with thresholds near 50 ppb have stronger correlations with mortality than non-threshold models for the Bay Area (Fairley 2003). In a recent article, Jerrett *et al.* (2009) found marginal evidence for a mortality threshold of 56 ppb in a longitudinal study of US metropolitan areas.

Threshold estimation and adjustment

The straightforward method to estimate the effect of an ozone threshold in an analysis of ozone health effects would be to incorporate it into the statistical model explicitly, for example maximizing the likelihood under a range of thresholds and choosing the corresponding beta. For the MPEM, however, we depend on health effects studies where thresholds were not considered.

How can betas found fitting a no-threshold model be adjusted under the assumption that a given threshold exists? The typical C-R models are multivariate and non-linear, where a closed-form solution for beta does not exist. What follows shows the relationship for a simple linear regression.

Suppose that a threshold x = t exists for a given health effect. Let y be the response (e.g., daily number of hospital admissions for asthma), and let x be the 1-hour max ozone. The threshold model can be written:

$$y_i = y_0 + b(x_i - t)^+ + e_i \qquad \text{(D1)}$$

for i = 1, 2,...,n where the e_i are error terms assumed to have mean zero and constant variance, and the function $z^+ = z$ if $z>0$ and 0 if $z<= 0$.

Suppose we fit a simple linear regression to this model. Then the estimated slope b is:

$$b = \sum_{i=1}^{n}(x_i - \bar{x})y_i / \sum_{i=1}^{n}(x_i - \bar{x})^2$$

Substituting D1 and taking expectations:

$$E(b) = \sum_{i=1}^{n}(x_i - \bar{x})[y_0 + \beta(x_i - t)^+] / \sum_{i=1}^{n}(x_i - \bar{x})^2 = \beta \sum_{x_i > t}(x_i - \bar{x})(x_i - t) / \sum_{i=1}^{n}(x_i - \bar{x})^2 = \beta r$$

where

$$r = \sum_{x_i > t}(x_i - \bar{x})(x_i - t) / \sum_{i=1}^{n}(x_i - \bar{x})^2$$

So, an unbiased estimate of β would be b/r.

For Bay Area sites in 2008, the values of r ranged from (essentially) 0 to 0.63, with a median of 0.38. Of course, the appropriate values would be those for the years and locations that were used in the health effects studies that serve as the basis for the C-R functions. For the MPEM, we will use a value of r = 0.5, that is, doubling the assumed ozone-response relation, but assuming it only applies for changes in 1-hour max ozone greater than 50 ppb.

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Appendix D

Regression Bias Induced by Measurement Error in a Predictor

Measurement error may arise in a host of situations, but for this analysis, the context is the potential bias in C-R functions. The C-R functions used in this analysis generally are based on epidemiological studies where monitored pollutant values are used as a surrogate for population exposure. We have assumed for these studies that the link between pollution and health effects is causative. Thus, we presume that there is a true, or at least more accurate, model, where the response, y, such as a heart attack, is linked to being exposed to a concentration, x, of a pollutant. But x is typically not measured, rather x*, the monitored value. This appendix considers simple linear regression to illustrate that the effect on the estimation of the regression slope, β, from regressing y on x* rather than y on x is to attenuate the slope estimate, that is, to estimate a slope that is less in absolute value.

Basics

How does measurement error affect the basic statistics – means, variances, covariance, and correlation? Let $\mu_x = E(X)$, $\mu_y = E(Y)$, σ^2_x = variance of X, σ^2_y = variance of Y, $\sigma_{xy} = E(X-\mu_x)(Y-\mu_y)$ = covariance of X and Y, and $\rho = \sigma_{xy}/(\sigma_x\sigma_y)$ = correlation of X and Y.

Suppose we measure $X^* = X + \delta$, where δ is a random variable independent of X and Y with mean 0 and variance σ^2_δ. Then the mean of X* is μ_x, the same as X. The covariance of X* and Y = $E(X^*-\mu_x)(Y-\mu_y) = E(X- \mu_x + \delta)(Y-\mu_y) = E(X-\mu_x)(Y-\mu_y) + E[\delta(Y-\mu_y)] = \sigma_{xy} + 0$ = covariance of X and Y. So the covariance of X* and Y is the same as the covariance of X and Y. But the variance of X* is $E(X - \mu_x + \delta)^2 = E(X-\mu_x)^2 + E[\delta(X-\mu_x)] + E(\delta^2) = \sigma^2_x + \sigma^2_\delta$. So the variance of X* is greater than X. Then the correlation of X* and Y is $\rho^* = \sigma_{x^*y}/(\sigma_{x^*}\sigma_y) = \sigma_{xy}/(\sigma_{x^*}\sigma_y) < \sigma_{xy}/(\sigma_x\sigma_y) = \rho$, provided $\sigma^2_\delta > 0$. The regression slope should be an increasing function of the correlation, all else being equal, so lowering the correlation should reduce the slope.

Bivariate Normal Case

Consider a theoretical case where the pair (X,Y) has a bivariate normal distribution with means (μ_x, μ_y), variances (σ^2_x, σ^2_y), and covariance σ_{xy}. For simplicity, assume $\sigma_{xy} > 0$. The regression analogue is the expected value of Y given X=x:

$$E(Y \mid X = x) = \mu_y + (\sigma_{xy}/ \sigma^2_x)(x - \mu_x) = \alpha + \beta x,$$

where $\alpha = \mu_y - (\sigma_{xy}/ \sigma^2_x)\mu_x$, and

$$\beta = \sigma_{xy}/ \sigma^2_x \qquad (1)$$

where $\beta > 0$, by assumption.

(See, e.g., Introduction to the Theory of Statistics, by A. M. Mood, F. A. Graybill, and D. C. Boes, page 167 in the 1974 [Third] Edition).

Suppose we measure $X^* = X + \delta$, where δ is an independent Normal random variable with mean 0 and variance σ^2_δ, in other words, we measure X with error, and the measurement is unbiased. Then $\mu_{x^*} = E(X^*) = E(X) + E(\delta) = \mu_x$, $\sigma^2_{x^*} = Var(X^*) = Var(X) + Var(\delta) = \sigma^2_x + \sigma^2_\delta$, and

$\sigma_{x^*y} = E(X^*Y) - \mu_x\mu_y = E(XY) + E(\delta Y) - \mu_x\mu_y = E(XY) - \mu_x\mu_y = \sigma_{xy}$.

So, $E(Y \mid X^* = x) = \mu_y + (\sigma_{x^*y}/ \sigma^2_{x^*})(x - \mu_{x^*}) = \mu_y + [\sigma_{xy}/ (\sigma^2_x + \sigma^2_\delta)](x - \mu_{x^*})$.

If the covariance between X and Y is positive, then $\beta^* = \sigma_{xy}/ (\sigma^2_x + \sigma^2_\delta) < \sigma_{xy}/ \sigma^2_x = \beta$.

Simple linear regression

In the simple linear regression model, we have

$y_i = \alpha + \beta x_i + \varepsilon_i$,

$i = 1, 2, \ldots, n$, where we assume the x_i are fixed constants and the ε_i are independent with mean 0 and variance σ^2_ε. Under these assumptions, the fitted least squares regression slope, $\hat{\beta}$, is an unbiased estimator of β, where

$$\hat{\beta} = s_{xy} / s_x^2,$$

with $s_{xy} = \sum_i (x_i - \bar{x}) y_i$, and $s_x^2 = \sum_i (x_i - \bar{x}) x_i$. Note the similarity with equation (1).

Suppose instead of observing the x_i, we observe $x_i^* = x_i + \delta_i$, where the δ_i have mean 0 and variance σ^2_δ, and are independent among themselves and also from the ε_i.

The simple linear regression fit yields

$$\hat{\beta}^* = s_{x^*y} / s_{x^*}^2$$

The expectation of $\hat{\beta}^*$ given the δ_i is

$$E(\hat{\beta}^* \mid \delta_i) = E\sum_i (x^*_i - \bar{x}^*) y_i / s^2_{x^*} = \sum_i (x^*_i - \bar{x}^*) E(y_i) / s^2_{x^*} = \sum_i (x^*_i - \bar{x}^*)(\alpha + \beta x_i) / s^2_{x^*}$$
$$= \beta \sum_i (x^*_i - \bar{x}^*) x_i / s^2_{x^*}$$

So, again assuming that $\beta > 0$,

$$E(\hat{\beta}^* \mid \delta_i) < \beta$$

$$\Leftrightarrow \quad \sum_i (x_i^* - \bar{x}^*) x_i < \sum_i (x_i^* - \bar{x}^*) x^*_i$$

$$\Leftrightarrow \quad -\sum_i (x_i - \bar{x}) \delta_i < \sum_i (\delta_i - \bar{\delta})^2$$

plugging $x^*_i = x_i + \delta_i$ and $\bar{x}^* = \bar{x} + \bar{\delta}$ in the second inequality to get to the third.

At least asymptotically, the last inequality will hold, provided there are some regularity conditions on the x_i, like assuming that they are bounded or that the mean sum of squares converges to a finite quantity. Under these conditions, dividing each side by n, each side converges in probability to its expected value, which is 0 for the left-hand side and $\sigma^2_\delta > 0$ for the right-hand side.

The inequality doesn't always hold. For example, suppose $\delta_i = - x_i/2$. Then the left side of the last inequality is double the right. But it does become almost certain, provided the measurement error is relatively large and the sample size is more than minimal because of the Law of Large Numbers.

Actually, if we can divide the last inequality by $s_x * s_\delta$, then the left side represents the negative of the sample correlation, -r, between the x_i and the δ_i, and the right side becomes s_δ / s_x, the ratio of the measurement standard error to the standard error of the observations; in other words, essentially the measurement error as a fraction of the total.

The Central Limit Theorem implies that the distribution of $r + s_\delta/s_x$ can be reasonably well approximated by assuming x_i and δ_i are two sequences of independent and identically distributed normal random variables. Here are the results of simulating $r + s_\delta / s_x$ using this normal assumption for several values of sample size, n, and error fraction $f = \sigma_\delta/\sigma_x$.

Probability regression slope underestimates true slope for various sample sizes and error fractions. Each cell is based on 1,000 simulated sets of x_i and δ_i.

	Sample Size	
Error Fraction, f	n = 25	n = 100
10%	0.68	0.82
25%	0.88	0.99
50%	0.99	1.00

The table shows that if the measurement error is as large as 50%, then the fitted regression slope is almost certain to underestimate the true slope (the slope of y on x, where x is measured without error). Even with a more modest 25% error, the fitted slope will very likely be an underestimate. For an error fraction as small as 10%, an underestimate is more likely than an overestimate, but there is still a substantial possibility of an overestimate unless the sample size is large.

Slopes in C-R functions

C-R functions for ozone and $PM_{2.5}$ are typically derived from a general linear model, not even multiple linear regression let alone simple linear regression. Thus, the above results certainly do not apply directly. However, some of the basics should remain the same, namely that the correlation between the response and the pollutant concentration measured at a monitoring station will be less than the correlation between the response and true exposure concentration of that pollutant. All else being equal, the slope for that pollutant in the C-R function will be an increasing function of the correlation so that lowering the correlation should reduce the slope.

Appendix E

School Absence Calculations

For several reasons, the calculation of the impact of ozone exposure on school absences requires additional analysis. First, the key study, Gilliland *et al* (2001), used an unusual ozone summary statistic that requires conversion to the one in the MPEM. Second, the C-R function is for illness-related absences but we have incidence data only for total absences. Third, unlike other effects such as hospital admissions, the impact of ozone on school absences can only occur on days when kids are in school.

1) Gilliland *et al* (2001) found a 62.9% increase in illness-related school absences for a 20 ppb increase in 10am – 6pm ozone.[17] Our impact function looks at changes in daily maximum 1-hour exposure. Thus, we need to estimate a relation to predict the former from the latter.

Figure E1 shows the relationship of the two sets of ozone values for Livermore, 2008. Also shown is a least squares linear regression line for 1-hour ozone values (x) greater than 50 ppb. The relationship in this range does look approximately linear, with a predicted value of 10am-6pm average ozone (y) as 0.63x + 9.376. So, for example, if the 1-hour maximum were 100 ppb, the 10am-6pm average would be predicted to be 0.63 * 100 + 9.376 = 72.376.

17 They found a 63% increase in illness-related absences, but our data on school absences is limited to the total.

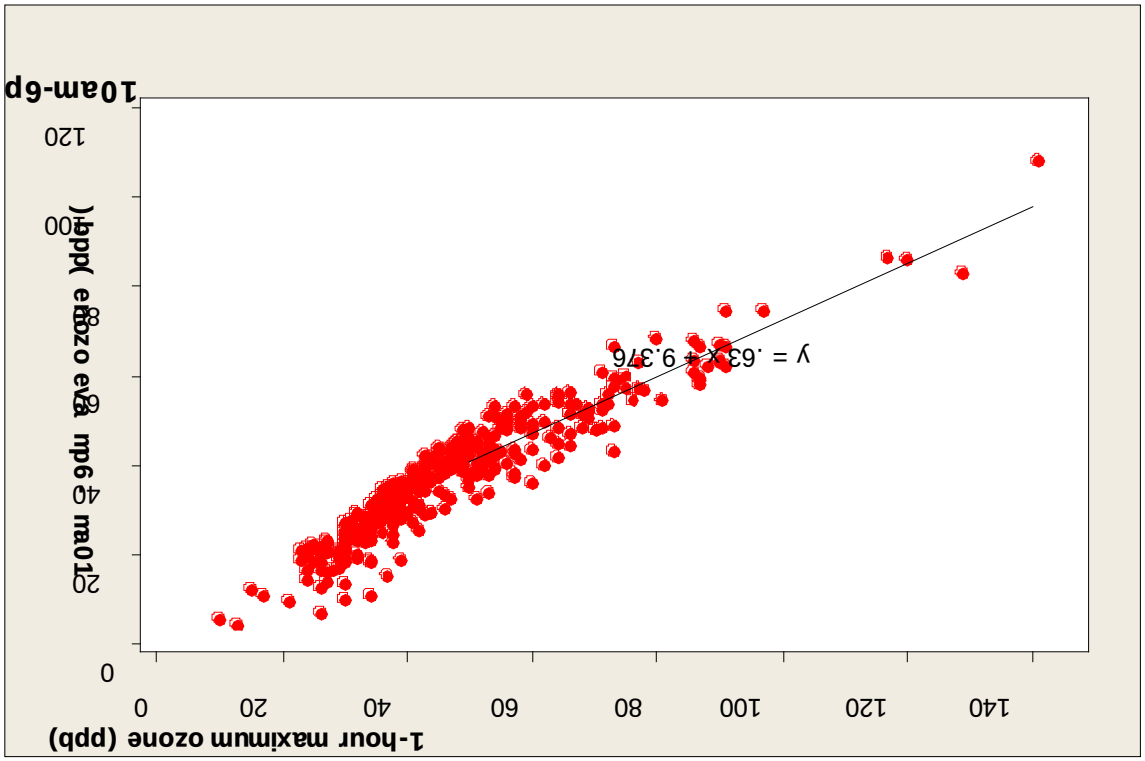

Figure E1. Relationship of 10am-6pm mean ozone to the 1-hour maximum at Livermore, 2008.

But the key issue is how a *change* in 1-hour maximum ozone relates to a *change* in 10am-6pm ozone. If the 1-hour maximum ozone were reduced from 100 ppb to 90 ppb, the 10am-6pm would be predicted to be reduced by 6.3 ppb, that is 0.63 times the 1-hour maximum reduction. So this is the factor to apply to the C-R coefficient.

In the MPEM, we have used C-R coefficients related to a 1 ppb change in 1-hour maximum ozone. Thus, a 62.9% change in school absences per 20 ppb change in 10am-6pm ozone would become:
(0.629 / 20) (0.63) = 0.0198 change per 1 ppb 1-hour maximum ozone.

2) Gilliland *et al* (2001) found an adjusted 1.34 non-illness absence rate and a 1.64 illness absence rate (rates per 100 children-days). Thus, the fraction 1.64 /(1.64+1.34) = 0.55 of all absences are illness-related.

3) Effects of ozone on school absences need to account for school schedules. Schools do not operate on weekends, and they are closed for national holidays. Most students have summer vacations, coincidentally during the time of highest ozone levels. But some students attend year-round schools and others attend summer school. Hall et al. (2008) estimated that 21% of San Joaquin Valley children 5-17 attended school during the summer. We did not obtain figures for the Bay Area, so we use the Hall estimates.

To account for intersection of ozone exposure and school attendance, we computed the average of Bay Area ozone exposures weighted by the fraction of students attending

school on those days. This represented 40% of exposure. Thus, we use a 0.4 factor for the incidence term in computing change in school absences from ozone.

Summary

Combining the results of (1), (2) and (3), the formula for the impact of ozone on school absences is:

y_0 x 0.0047 x 0.55 x [exp(0.0198z) – 1] x (0.4 x 365)

where y_0 is the number of school-aged children and 0.0047 is the absence rate (from the San Francisco Unified School District).

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Appendix I

Estimation of the rate of conversion of SO2 to ammonium sulfate based on ambient data

It is undoubtedly true that much of the sulfate in the air of the Bay Area comes from atmospheric conversion of SO2. There is a good correlation between measured sulfate and SO2 at various sites, and reductions in sulfate over the past 25 years have paralleled reductions in SO2. But an investigation of the CMAQ-modeled effect on sulfate from reductions in SO2 showed a low response. Specifically, for an across-the-board 20% reduction in SO2, the CMAQ model produced a reduction in sulfate of only 0.4%.