

**Multi-Pollutant Evaluation Method
Technical Document**

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Executive Summary

The Bay Area Air Quality Management District (District) has prepared the draft Bay Area 2010 Clean Air Plan (CAP) to update its previous ozone plan, the 2005 Ozone Strategy, as required by the California Health & Safety Code. In addition to updating the ozone plan, the 2010 CAP will serve as a multi-pollutant plan to protect public health and the climate. The CAP will propose a control strategy designed to maximize reductions in 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 2010 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 and greenhouse gas reductions for all pollutants 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 impacted related to climate change.

MPEM Foundation

The MPEM is based upon well-established studies and methods that have been used by the U.S. 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 its precursors; and
- Including an estimated 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 science and health data suggest 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, hydroflourocarbons, perflourocarbons, and sulfur hexafluoride.

District staff believe that the MPEM captures most of the key health effects from 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:

- We assume that the emissions reductions for each control measure will be geographically distributed on the same basis as the emissions of each pollutant are distributed in the District's emissions inventory. For example, if we estimate that a control measure would reduce one ton of NO_x, we then distribute the NO_x emission reductions across Bay Area grid cells in the same proportion as the overall NO_x inventory is distributed across those grid cells. (Section 1.5)
- For purposes of estimating population exposure (Step 3 below), we assume full-time (24/7) "backyard" exposure, even though we realize that in reality 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 measure¹. 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, i.e., the sensitivity between emissions and concentrations. For ozone, PM, and air toxics, we employ photochemical modeling results to calculate sensitivities 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 CO₂-equivalent.

¹ For ozone, PM_{2.5}, and air toxics, we employ Steps 1 through 5. For greenhouse gases, only Step 1 and Step 5 are applied; the intermediate stages, 2-4, are not performed. For discussion of how we consider greenhouse gases for purposes of this methodology, see Section 5.3.

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.

Applications

For purposes of the 2010 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 2010 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.

Conclusion

The multi-pollutant evaluation methodology summarized above, and described in detail in the body of this document, is a tool developed by 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 draft 2010 CAP.

The MPEM makes use of the tools and technical data *available at this time*. 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) has prepared a draft 2010 Clean Air Plan (CAP) that proposes an integrated plan to reduce multiple air pollutants. This Technical Document describes the multi-pollutant evaluation methodology (MPEM) developed by District staff to help analyze and compare the benefits of potential control measures on a multi-pollutant basis.

The MPEM is based upon well-established studies and methods that have been used by the U.S. 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 and greenhouse gas reductions for all pollutants that would be reduced by each potential control measure
- 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 impacted 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 NO_x
- PM_{2.5} both - primary PM_{2.5} as well as precursors of secondary PM_{2.5} (NO_x, SO₂, NH₃)
- Air toxics
- Greenhouse gases (GHGs)²

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. For discussion of how we consider greenhouse gases for purposes of this methodology, see Section 5.3.

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 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 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 CO₂-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, and 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.

1.1 Probability Analysis

Even though District staff has made 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 measure³
- 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
- Population projections at the census tract level from the Association of Bay Area Governments (ABAG)
- Estimates of the changes in incidence rates from changes in pollutant concentrations from a number of epidemiological studies
- Health endpoint incidence rates for the Bay Area
- 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

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

choices made by District staff in developing the MPEM. Key choices in developing this method are:

- 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. To evaluate the health benefits of control measures, we chose a set that represents most of the known health risks. Specifically, we consider PM_{2.5}, ozone, and a small set of carcinogenic air toxics: benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and diesel PM_{2.5}.

PM_{2.5} and ozone were chosen because they are the two criteria pollutants⁴ for which the Bay Area continues to violate national air quality standards. The toxic compounds chosen represent almost 90% of the known carcinogenic risk in the ambient air of the Bay Area⁵. Two other pollutants, carbon tetrachloride and hexavalent chromium, which make up much of the remaining risk from air toxics, were not considered for various reasons⁶.

The pollutants chosen are not all emitted directly. Thus, we need to consider two lists – the list of *precursor* pollutants emitted, and the list of *health-related* pollutants whose health effects are evaluated. There is considerable overlap, but some pollutants, like ozone, have health effects but are not directly emitted. Conversely, others, like VOCs, are emitted directly but are not evaluated as health threats in themselves. Rather, we include them because they transform in the atmosphere to produce pollutants that are health threats (both ozone and PM_{2.5} in the case of VOCs). Ozone is almost entirely a secondary compound, formed largely from reactions of NO_x and VOCs. Thus, we will consider emissions of VOCs and NO_x.

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 NO_x 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.

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 criteria documents. The six pollutants were ozone, TSP (now PM_{2.5}), NO₂, SO₂, CO, and lead. The Bay Area comfortably meets the national (and even the stricter California) standards for the other 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 any more 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.

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 NO_x, 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.⁷

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 99% of the known GHG potential of the Bay Area (BAAQMD 2006): CO₂, 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.

Table 1 – Pollutants Included in Multi-Pollutant Evaluation Methodology

Category	Direct or Precursor Emissions	Pollutant causing health/social impacts
Ozone	NO _x VOC	Ozone
PM _{2.5}	Directly Emitted PM _{2.5} NO _x 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}

⁷ The question of what components, sizes and aspects of PM are more harmful is an area of current research. There is mounting evidence that ultra-fine particles are more harmful than larger particles, but the results are not yet definitive.

Greenhouse Gases	Carbon Dioxide Methane Nitrous Oxide Sulfur Hexafluoride Hydrofluorocarbons Perfluorocarbons	GHG in CO ₂ -equivalent
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For every pollutant included in the above list there are literally dozens that are not included. The main reasons for limiting the pollutants to those in Table 1 is pragmatic – to include dozens more would require a major effort both in developing the methodology and in rule development.

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. We will continue to monitor the health effects literature and, we hope, 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 simplest assumption 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 a grid of 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; from the PM modeling for two winter months, December and January; and from the ozone modeling for two mid-summer episodes of about a week each.

The models (Soong, et al., 2007, Tanrikulu et al., 2009, Beaver et al., 2009) provide an estimate of the relationship between a change in emissions and a change in pollutant concentration for ozone, toxics and particulate matter, respectively.

The health related pollutants, except for diesel⁸, are measured at various District monitoring stations around the Bay Area. These observations were used to extend the model results beyond the modeled periods, assuming the background fraction and atmospheric dynamics are constant year-round.

Toxics: From a limited trend analysis conducted (Appendix A), we found a linear relation between decline in concentrations and emissions of benzene and 1,3-butadiene. Although diesel concentrations have not been monitored, we assume that the trend for this pollutant is also linear.

⁸ Diesel PM cannot be directly measured.

Formaldehyde and acetaldehyde are both primary and secondary, but we are considering only primary emissions. Therefore, we also assume that the relation between emissions and concentrations for these species is 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. Grids with large concentrations showed reductions close to 10%. For grids with lower initial concentrations, the results were more variable. For diesel, these reductions averaged less than 10%; for grids with diesel concentrations $< 0.5 \mu\text{g}/\text{m}^3$, the median reduction was 6% with half of the reductions between 3.5% and 7.5%.

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

The PM_{2.5} emission reduction simulation was available only for the winter season. The simulated trends may vary by season. How calculations are made for this species is explained in detail in Appendix E.

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

Modeling for the Bay Area indicates that reductions in NO_x may actually cause increases in ozone in some areas during some episodes. (Appendix B shows an example.) Conversely, due to the NO_x quenching or ozone titration, high NO_x concentrations can cause ozone concentrations to fall below natural background levels in some areas, as shown in Figure 4 of Appendix A where Los Gatos peak ozone has been reduced, but its lower percentiles have increased.

The ozone model does, in fact, capture the counter-intuitive behavior described above. However, ozone modeling is performed only for a few episodes.

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. From a policy perspective, it is important to balance health protectiveness against 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: For PM_{2.5}, most epidemiological studies looking for a threshold have not found 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 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. Ostro *et al.*, (2006) reported that they did not find a threshold in the literature. Several studies of ER visits for asthma suggest a population threshold in the range of 0.075–0.110 ppm for 1-hr maxima." They assumed 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. A study done here for the Bay Area suggests that at concentrations around 50 ppb, the health impact diminishes, see Appendix D. For this analysis, we assume a 50 ppb threshold.

1.3.5 Scientific Evidence for Causality and Public Policy

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 casualty between air pollution and health effects, for most of the effects used in the MPEM, there is a scientific consensus that the effects are causal or "likely causal".

Toxics Judgments of causality have been made at both the national and international levels. At both levels, benzene is judged a human carcinogen; the other toxics used in MPEM are considered likely carcinogens.

PM_{2.5} The EPA recently completed its Integrated Science Assessment or ISA (US EPA 2009a), 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's conclusion is: "Collectively, the evidence is **sufficient to conclude that the relationship between long-term PM_{2.5} exposures and mortality is causal.**" (Emphasis in the original)

Ozone EPA has evaluated the evidence for ozone health effects in its Ozone Criteria Document (US EPA 2006b) and a more review of more recent evidence (US EPA 2009b). Ozone (O₃) is unusual in that human health studies have been performed to assess the impact of exposures. Human subjects subjected to elevated ozone concentrations experience cough, chest pain, decreased lung volumes and airway irritation. Asthmatics are affected more strongly. Long-term exposure to high ozone levels leads to permanent reduction in lung function. Their conclusions are not as emphatic as with PM_{2.5}: it is "highly suggestive that

O3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality." For the ozone respiratory effects: "the overall evidence supports a causal relationship between acute ambient O3 exposures and increased respiratory morbidity resulting in increased ED visits and hospitalizations during the warm season" (U.S. EPA, 2006)

GHGs The Intergovernmental Panel on Climate Change (IPCC) is 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."
2. "Global atmospheric concentrations of CO₂, methane and nitrous oxide have increased markedly as a result of human activities since 1750 and now...exceed by far the natural range in over 650,000 years."
3. "There is very high confidence that the net effect of human activities has been one of warming."

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 500 tons of NO_x is emitted in the Bay Area per day, we would calculate the impact of a 1/500th reduction in NO_x emissions by geographically distributing the NO_x reduction the same as the geographic distribution of the entire NO_x inventory.

The reason for this assumption is practical. Attempting to calculate more focused effects is currently beyond our capacity. 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 estimates ambient concentrations 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. Currently, the models for ozone, PM_{2.5} and toxics are run separately, covering different time periods. 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 2000 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 (5%, 10% or 20% were used in different models). The difference in concentrations between the base case and these sensitivity runs served as the basis for estimating how emissions reductions would affect pollutant concentrations.

In developing the multi-pollutant evaluation method we use the models 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 simpler case of a *primary* pollutant, one that is emitted directly, let c_{0i} be its initial concentration in grid square i , and let Δc_{ri} be the change in this concentration resulting from the emission reductions for a given control measure, Δe_r (considered as a percentage of total annual District emissions of that pollutant). So for the concentrations of primary pollutants, the following relation exists:

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

where 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 well-approximated 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.

Generally, we want to consider joint effects, but the model does not provide these directly. What is available are individual runs that provide estimates of the effects of precursor reductions individually, and a model run where all precursors are reduced jointly. 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, \dots, x_k . The resulting fitted regression equation:

$$y = f_1x_1 + f_2x_2 + \dots + f_kx_k$$

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

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

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. But the District's computer simulations of pollutant concentrations are all run for only part of the year.⁹ Thus, there is a need to extrapolate concentration estimates to other times not covered by the simulations.

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. For PM_{2.5}, modeled concentrations were adjusted from winter to annual based on ratios of winter to annual concentrations found in the ambient data. For ozone, where an extensive monitoring network of BAAQMD monitoring sites provides good spatial estimates of daily ozone concentrations, daily maximum ozone concentrations for 2005-07 were used, interpolated to each grid square.

⁹ These simulations require extensive effort to develop. The requirements for dense and accurate meteorological data and realistic simulations of wind fields continue to limit the time that models represent. The PM_{2.5} model was run for a December-January period; the toxics model for a week in July and a week in December; and the ozone model for two summer episodes of about a week each.

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 Toxics

2.2.1 Concentration averaging times

Unlike for ozone and PM_{2.5}, there are no national or California air quality standards for 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.

The toxics model simulates toxics concentrations with meteorology from one week in December, 12/12-12/18 and one week in July, 7/12-7/18, using meteorology from those weeks and a 2005 inventory that varies by time of day, weekend/weekday and by season. 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.

2.2.2 Relating toxic concentrations to toxic emissions

Sensitivity analyses were run, 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 % change in concentration to % 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.

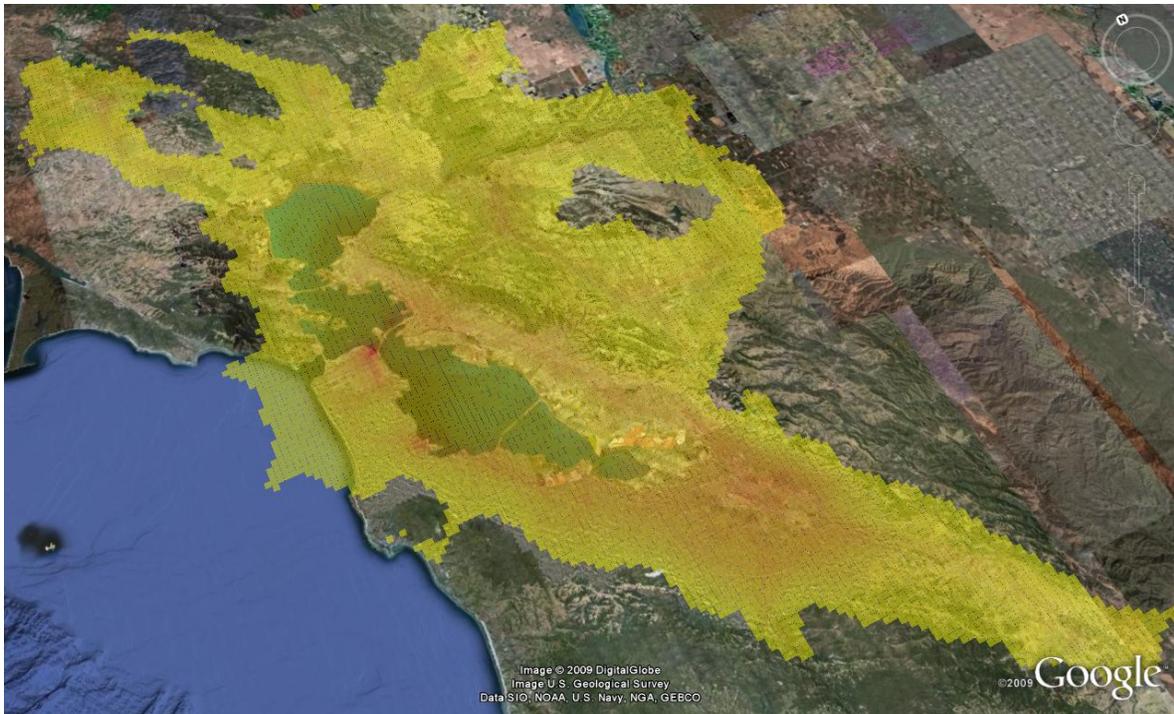


Figure 1. Grid of model estimated reductions in benzene concentrations for a 10% reduction in benzene emissions. Values range from 0.01 ppb (light yellow) to 0.08 ppb (red).

This was combined with the estimated initial concentrations, c_{0i} , estimated in the next two sections below to yield the values for equation (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 elemental carbon and diesel PM, 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 Ozone

2.3.1 Ozone concentration averaging times

Ozone standards are specified for peak ozone. The national primary ozone standard is based on daily 8-hour maximum ozone.¹⁰ It states that the 3-year average of the annual 4th highest 8-hour maxima cannot exceed 75 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 concentration, *c*, used is the daily maximum 1-hour ozone.

2.3.2 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 (CAMx) was run for varying combinations of ozone's precursors, NO_x and VOCs, with all 9 combinations of reductions of 0%, 5% and 10% for NO_x and VOCs. The goal was to estimate the change in ozone as a function, *f*(*n*,*v*), of reductions in NO_x 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 NO_x and VOCs. Specifically, for each grid cell *i*, the model

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

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 NO_x and VOC.

Details are provided in Appendix B.

2.3.3 Estimating initial ozone concentrations

¹⁰ 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.

Ambient ozone is measured at 23 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 ozone simulations involved modeling two *ozone episodes*, periods with ozone violating that national standard. Specifically, two runs were made that included a total of five episode days: July 11-12, 1999 and July 31-August 2, 2000. These runs provide good information on how the relationship between ozone formation and precursor changes spatially, but they do not provide a concentration distribution seasonally or annually.

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 2005-07, interpolated to a concentration c_{0i} for each grid square.

2.3.4 Incorporation of a 50 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 50 ppb. (See Appendix C 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 50 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 50 ppb for a reduction of $r\%$ was fit for values of $r = 0, 1, \dots, 20$, where $f_i(r)$ was the summation of $z_j = (1-r)y_j - 50$ for all 1-hr max ozone values y_j in 2005-07 for which z_j was positive. The fits were generally excellent, with over 95% of the fits having R^2 values > 0.99 .

2.3.5 Calculation of ozone above threshold

Combining these factors, if the reduction in NO_x emissions from a given control measure (as a percent of total NO_x emissions) is Δe_n , and the reduction in VOC is Δe_v , then the average daily 1-hr max ozone above 50 ppb in grid square i is estimated as:

$$\Delta f_i = [f_i(0) - f_i(r_i)] / (3 \cdot 365),$$

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

2.4 PM_{2.5}

What follows is a summary of our method. Details are provided in Appendix E.

2.4.1 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 the annual standards, but violates the national 24-hour standard, which allows roughly 7 days exceeding $35 \mu\text{g}/\text{m}^3$ per year at any site.

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 totally 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 C for details on the effect of this approximation.

2.4.2 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. 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.

2.4.3 PM_{2.5} simulation using the CMAQ model

The District, like most air quality agencies, has more limited experience with PM modeling than ozone modeling. The reduction in the national 24-hour PM_{2.5} standard has caused the District to devote much greater focus to modeling of PM formation and transport in northern California. This includes analysis of meteorological conditions conducive to high PM levels, PM transport between air basins, and effects of emission reductions on ambient concentrations. This PM modeling and data analysis is still at a preliminary stage and will continue over the next several years.

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 the two modeled December-January periods. Six sensitivity runs were also made with 20% reductions in: 1. NO_x, 2. VOC, 3. ammonia, 4. sulfur gases including SO₂, 5. directly emitted PM_{2.5}, and 6. reductions in all 5 categories.

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

Bay Area PM_{2.5} concentrations vary considerably on a seasonal basis, with much higher peak concentrations occurring in the late fall and winter, especially December and January, than in the summer months. Thus, the highest 24-hour PM_{2.5} concentrations occur almost exclusively the winter season. Because the Bay Area violates the national 24-hour standard but not the annual standard, our PM_{2.5} modeling efforts to date have focused on winter months.

The PM_{2.5} model simulated two December-January periods, with meteorology from December 2000 through January 2001, and December 2005 through January 2006, and a 2005 emissions inventory.

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, run 6, showed almost the same changes in PM_{2.5} concentrations from the base case as run 5. Therefore, no adjustment was made to convert from marginal to jointly-considered effects.

2.4.5 Extension to annual PM_{2.5} concentrations

Some of the carbonaceous PM_{2.5} sources, notably wood burning, have distinct seasonal components, so that estimating annual concentrations from modeled concentrations covering December and January would be highly inaccurate. Moreover, Bay Area PM_{2.5} concentrations are highly seasonal because meteorology varies by time of year (wind speeds and direction, and atmospheric stability in particular). Thus, here we need to adjust winter modeled concentrations to annual average concentrations.

The District measured elemental carbon (EC) and organic carbon (OC) on PM₁₀ filters for 2005-07 for 5 sites, and 2006-07 at 3 additional sites. The averages of these measurements were used to adjust the modeled values. For EC, the ratio of annual to January+December concentrations was 0.524. For OC, the ratio was higher, but is complicated because measured OC includes carbon from both directly emitted and secondary sources. Adjusting for this produced a ratio of 0.57. The OC measurements and modeled values include the carbon mass only, whereas the original PM_{2.5} includes oxygen and hydrogen. A 1/0.833 factor was used to adjust from the mass of carbon to the total mass.

Finally, the emissions values will not be broken down into EC and OC, so a single ratio of 0.56 was used for the sum of the two.

Thus, the estimated change in concentrations of directly emitted carbonaceous particles from a reduction of x% in carbonaceous PM_{2.5} emissions is:

$$0.56 [(EC_{\text{base}} - EC_{\text{pm}}) + (OC_{\text{base}} - OC_{\text{pm}})/0.833](x/20)$$

where EC_{base} and OC_{base} are the EC and OC carbon concentrations from the base case model run, and EC_{pm} and OC_{pm} are the EC and OC carbon concentrations from the run with 20% PM_{2.5} reductions.

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

Analysis of the changes in ammonium sulfate concentrations as a function of reductions of various precursors showed that not only was ammonium sulfate sensitive to changes in ammonia and sulfur-gas emissions, but also to changes in directly emitted PM_{2.5}. The latter is likely the influence of directly emitted sulfate.

2.4.7 Factors for joint effect of precursors

A multiple regression was performed with the sulfate concentration change from run 6 as the dependent variable and the reductions from the other runs as the independent variables. The factors for adjusting from marginal to jointly-considered effects were 0.85 for ammonia, 0.90 for sulfur gases and 1.03 for sulfate.

2.4.8 Estimating annual ammonium sulfate concentrations

Extensive PM_{2.5} measurements have been made for a number of years at a site in Point Reyes. This site is near the ocean and has few nearby anthropogenic PM_{2.5} sources. The PM_{2.5} measurements made for Point Reyes include sulfate and a range of other compounds and elements. These measurements allowed for estimation of the PM_{2.5} sources at various times of the year and under different wind conditions (Fairley 2009). The analysis showed that under westerly winds, sulfate from marine air increases while sulfate from other sources stays about constant.

Sulfate measurements are collected at a number of other Bay Area sites and show higher averages for the full year than for the winter. But given the results from Point Reyes, it was concluded that this increase was largely due to an increase in sulfate from marine air, that is, from a non-anthropogenic source. Therefore, we assumed that the annual anthropogenic sulfate concentrations were equal to the December-January concentrations.

2.4.9 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: $(132/96)^{11}$.

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.

¹¹ Sulfate, SO₄ has atomic weight 96. Each sulfate molecule combines with 2 ammonium (NH₄) molecules, for an atomic weight of $96 + 2 \times 18 = 132$.

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

Modeling showed that VOC had a substantial impact along with ammonia and NO_x in ammonium nitrate concentrations. The effect of directly emitted nitrate was negligible.

Preliminary modeling showed a potential disbenefit to NO_x reductions for certain episodes in certain parts of the Bay Area, including several urban areas. The potential disbenefit is not supported by observations and may be caused by the use of modeling inventories from prior years (2000 and 2005). This issue will be evaluated for multiple episodes with a more recent inventory, and the use of direct modeling results will be possible upon conclusion of this work.

For this reason, in lieu of modeling, the impact on ammonium nitrate from changes in NO_x emissions is based on an analysis of ambient data. This analysis found that reductions in NO_x were correlated with annual reductions in nitrate. This included a comparison of weekend to weekday NO_x and nitrate, where weekend ambient nitrate was found to be lower than weekday, whereas the corresponding modeled values showed an increase. From this analysis, we concluded that the effect of a given reduction, x , in NO_x emissions will reduce ammonium nitrate by $0.032x$. See Appendix F for details. We used this factor in the MPEM to estimate the change in ammonium nitrate from a change in NO_x emissions, rather than the modeling results.

Modeling shows benefits for ROG and ammonia reductions everywhere in the Bay Area. This does comport with expectations based on modeling elsewhere. Ambient ROG and ammonia data are unavailable, so an analysis similar to that presented in Appendix F was not possible. Therefore, to estimate the effect of reductions in emissions of ROG and ammonia, we use modeling results.

2.4.11 Factors for joint effects of precursors

A multiple regression was performed with the nitrate concentration change from run 6 as the dependent variable and the reductions from the other runs as the independent variables. The factors for adjusting from marginal to jointly-considered effects were 0.93 for ammonia, 1.00 for NO_x and 1.12 for VOC.

2.4.12 Extension to annual concentrations

The information for Bay Area ammonium nitrate concentrations is much more limited than for ammonium sulfate. Nitrate is measured on PM_{2.5} filters at a single Bay Area location, the BAAQMD San Jose-Jackson site. It is also measured at Point Reyes. The California Regional Particulate Air Quality Study (CRPAQS) study provided PM_{2.5} measurements at Bethel Island, Livermore and San Francisco for 1999-2001.

Statistical analysis of the ratio of annual to December-January concentrations showed a potential range of 0.35 to 0.65.¹² We used the midpoint of this range, assuming that the annual mean ammonium nitrate concentration was half that of December-January.

2.4.13 Calculation of the change in ammonium nitrate concentrations

A factor of 80/62 was used to convert from nitrate mass to ammonium nitrate mass.¹³

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:

$$\Delta c_i = c_i * 0.5 * (80/62) * [0.93(d c_i / d e_a) \Delta e_a + 1.00(0.032) \Delta e_n + 1.12(d c_i / d e_v) \Delta e_v]$$

3. Estimating Population Exposure

This section explains how we estimate population and population exposure.

3.1 Population and Demographics

Population projections for 2005 and 2010 for each Bay Area census tract were obtained from ABAG Projections 2007. We developed estimates for 2009 by interpolating the estimates for 2005 and 2010 i.e., 0.2 times the 2005 estimate plus 0.8 times the 2010 estimate.

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. The ABAG data includes projections for ages 0-4, 5-19, 20-44, 45-64 and over 65. Other age ranges were estimated combining these projections with the 2000 counts, which were available for each age. For example, to get the 2009 estimate of the number of 5-17 year-olds, we took the 2009 estimate for 5-19 year-olds and multiplied by the ratio of 5-17 year-olds to 5-19 year-olds in the 2000 census.

The census tract was then assigned to a grid square using its centroid, and the estimated population values for each grid square totaled. This population was assumed exposed to the concentration estimated for that grid square. The product of population times Δ concentration was then summed for each county and divided by the county population, yielding a population-weighted Δ concentration.

Figure 2 is the same as Figure 1 with population data overlaid.

¹² Bethel Island was an exception, with the upper bound of its 90% confidence interval less than 0.5. But this site is atypical of Bay Area sites, lying as it does in the Central Valley Delta.

¹³ Nitrate, NO_3 , has atomic weight 62. Each nitrate molecule combines with an ammonium (NH_4) molecule, for an atomic weight of $62 + 18 = 80$.

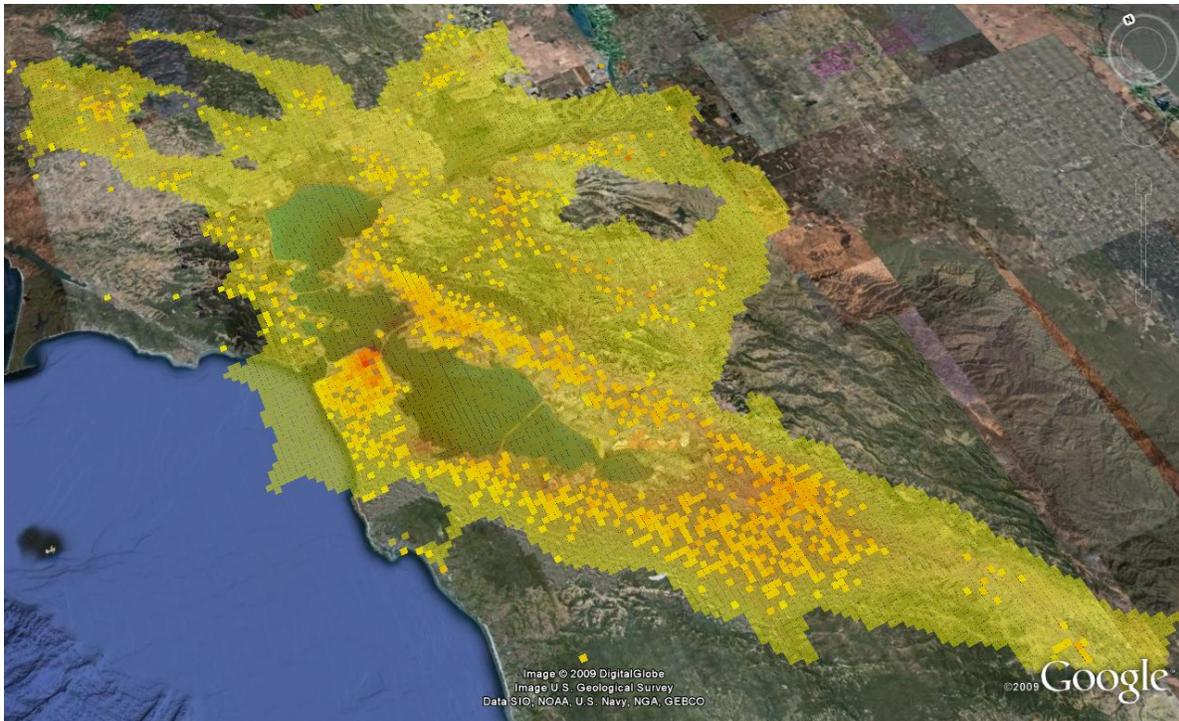


Figure 2. Reduction in benzene concentration overlaid with population. Grids with population shown with solid color.

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 used in the epidemiological studies and thus is an appropriate method in combination with steps 4 and 5.

Very few of us spend our entire lives in our backyards. 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 rough estimates of exposure. Thus, the concentration-response relationships developed are also based on rough 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.

i. Average ambient concentration

The average ambient concentrations used in the epidemiological studies is 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 / $10 \mu\text{g}/\text{m}^3$ so that there was a 2% change in incidence for a $10 \mu\text{g}/\text{m}^3$ change in *monitored* $\text{PM}_{2.5}$ concentrations. But actual exposures were not the same, so really, this is a 0.02 change for an $8 \mu\text{g}/\text{m}^3$ change in *exposure* concentrations. Thus the C-R slope is $10/8 \times (0.002) = 0.0025$ per $\mu\text{g}/\text{m}^3$ in $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$, we used modeled values, which may be unbiased. Thus, for $\text{PM}_{2.5}$ 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 $\text{PM}_{2.5}$ 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 to 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.

ii. Exposure

Exposures were estimated with error. If exposure were estimated without bias¹⁴, 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

¹⁴ 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 2lbs more than the real weight and sometimes 2lbs less than the real weight, so the error is plus or minus 2 lbs.

measured with error: $x^* = x + \text{error}$, 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 $|b^*| < |\beta|$. See Appendix G.

This issue was considered important by the experts in EPA's elicitation of experts' judgement about the true $\text{PM}_{2.5}$ – 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 $1 \mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$...many thought that this issue caused underestimation of the effects of $\text{PM}_{2.5}$ 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.¹⁵ 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 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 very rough. In studies of lab animals, the exposure may be well-controlled, but the low-dose extrapolation and extrapolation from other species to human introduces 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 $\mu\text{g}/\text{m}^3$ (OEHHA 2005) for an average lifetime exposure of $1 \mu\text{g}/\text{m}^3$ (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 $0.1(1) + 0.9(1 \times 0.7) = 0.73 \mu\text{g}/\text{m}^3$, for a true risk of $0.73 \times 300 = 219$ in a million.

¹⁵ The $\text{PM}_{2.5}$ – 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

This section discusses the selection and calculation of health effects caused by air pollution exposures to ozone, PM_{2.5} and the toxics listed in Table 1.

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:

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

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}, b say, 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, $e = 2.71828\ 18284\ 59045\ 23536\dots$

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

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

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:

$$\Delta \text{cases} = \text{population} \times (\text{baseline incidence rate}) \times (e^{bc} - 1) \quad \text{formula (4.3)}$$

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:

$$\Delta \text{cases} = \text{population} \times (\text{baseline incidence rate}) \times (e^{bc} - 1)/(1 + f) \quad \text{formula (4.4)}$$

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:

$$\Delta \text{cases} = \text{population} \times b/70 \times c \quad \text{formula (4.5)}$$

4.4 Population Data

We use population projections by census tract. See section 3.1 for details.

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 2005-07, the three most recent years available.

For hospitalization rates and asthma emergency room visits, we obtained 2005-2007 county-by-county rates from the California Office of Statewide Planning and Development (OSHPD), using the averages of the 3 years' data.

Rates for non-fatal myocardial infarctions (MIs) were computed at the national level starting with the National Hospital Discharge Survey for 2005, 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 estimates average around 0.00256, whereas the US EPA uses 0.00365, based on western states and 1999 data.

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} effects, our methodology includes most of those listed in Appendix F of the BENMAP Users Manual, US EPA (2008). 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 median 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 pointmass 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 µg/m³ increase in PM_{2.5}. Expert K also placed 100% of the mass of his distribution on values < 0.8% per 1 µg/m³. 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% / 1µg/m³. 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.

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 H.

For toxics, we use the risk values from OEHHA (2005). 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 4.1 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 4.1. 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 <i>et al.</i> 1995	≥ 27 (w/o bronchitis)	1.32	4.3	0.00378	Abbey <i>et al.</i>
COPD Hospital Admissions	Ito 2003 & Moolgavkar 2003	≥ 65	.116(.206) Ito 0.185 (.052) Moolgavkar	4.2	county-specific rates, 2007 Bay Area rate 0.0009	OSHPD**
COPD Hospital Admissions	Moolgavkar 2003	18-64	0.218	4.2	county-specific rates, 2007 Bay Area rate 0.0069	OSHPD**
Pneumonia Hospital Admissions	Ito 2003	≥ 65	0.398	4.2	county-specific rates, 2007 Bay Area rate 0.0126	OSHPD**
Cardiovascular Hospital Admissions (less MI)	Moolgavkar 2003	≥ 65	0.158	4.2	county-specific rates, 2007 Bay Area rate 0.0373	OSHPD**
Cardiovascular Hospital Admissions (less MI)	Moolgavkar 2003	18-64	0.140	4.2	county-specific rates, 2007 Bay Area rate 0.0040	OSHPD**
Non-Fatal Heart Attacks	Peters <i>et al.</i> 2001	≥ 18	2.41	4.3	county-specific. The Bay Area average rate is .00256	NHDS public use data files, adjusted for 30 day survival.
Asthma Emergency Room Visits	Norris <i>et al.</i> 1999	< 18	1.653	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHPD**
Acute Bronchitis Episodes	Dockery <i>et al.</i> 1996	5-17	2.721	4.3	0.043 cases per child per year	American Lung Association 2002
Upper Respiratory Symptom Days	Pope <i>et al.</i> 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	Schwartz & Neas	7-17	0.6	4.3	0.438	Schwartz <i>et al.</i> (1994, Table 2)

Symptom Days	2000					
Work Loss Days	Ostro 1987	18-64	0.46	4.2	2.17	Adams <i>et al.</i> 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	OSHDP**
Asthma Emergency Room Visits	Ostro 2006	< 18	0.24	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHDP**
School Loss Days	Gilliland <i>et al.</i> 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 / $\mu\text{g}/\text{m}^3$			
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 4.1 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 0.0086. So for an Alameda grid square, we would combine this with the estimated 0-17 year-old population, p_i , to produce

$$p_i * 0.0086 * (e^{0.0165 * \Delta c_i} - 1)$$

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.¹⁶

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

School absences. We follow the approach in Hall (2008) to take into account summer vacations, weekends, holidays, etc. See Appendix H 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.

¹⁶ The population figures are actually based on census tracts, each of which is assigned to a county. If a grid square covers census tracts from more than one county, then the population from each county is recorded separately, so that the health effect gets apportioned appropriately.

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 (2005), 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 5.1 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 \$22 to avoid a day of lower respiratory symptoms, to \$6,900,000 to avoid a death.

Table 5.1. Unit Values Used for Monetary Estimates for Quantified Health Effects*

Health Effect	Unit Value (Cost per Incident)	Type of Measure	Derivation of Estimate Explanation
Mortality (all ages)	\$6,900,000	WTP	Using US EPA (2005, the mean value of avoiding one statistical death is assumed to be \$6.9 in 2008 dollars. This unit is the mean value based on meta analyses of the wage risk value of a statistical life (VSL). This method is similar to Hall et al (2008) and Stratus (2008). US EPA 2005 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	\$409,189	WTP	The estimated unit value for CB was completed similar to Hall et al (2008). The EPA (2005) gave estimated values for the reduction in risk of CB (see Sec.4.1.5.4.2). The best estimates of WTP avoid a case of CB comes from Viscusi et al (1991) and Krupnick and Croper (1990). US EPA (2005) adjusts the both estimates and the WTP mean for an avoidance of CB is \$409,189 (in 2008 dollars).
Respiratory Hospital Admissions	Age 65 < : \$35,228 Age 65 > : \$33,375	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 < : \$43,889 Age 65 > : \$38,759	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	\$84,076	COI	There are no WTP values for the reduction of nonfatal hear 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. This method is similar to US EPA 2005 (except no discount rates were used in this derivation). Combining results from Eisenstein et al (2001) and Russell et al (1998); where Eisenstein calculates the first year direct acute medical costs of \$29,234 (2008 dollars) and Russell et al (2001) calculate the first year direct medical cost of \$22,835. Averaging the two with the updating 2008 dollars gives us \$52,069. Updating Cropper and Krupnick estimate to 2008 dollars and adding the average direct medical cost gives us \$84,076.
Asthma Emergency Room Visits	\$468	COI	Using US EPA 2005 and updating Smith et al (1997) where they report the average cost per emergency room visit made in 1987. Updating to 2008 CPI-U for medical care, the estimate is \$468. Updating US EPA (2008) to CPI-medical care dollars, distribution was derived (see Table H-7).
Acute Bronchitis Episodes	\$534, for a 6 day illness period	WTP	US EPA 2005 reports estimates of WTP based on preventing respiratory symptoms caused by acute bronchitis. US EPA 2005 assumes a 6 day illness period, where the unit value for avoiding one day is \$89 (2008\$). A 6 day WTP is \$534 (using CPI-U for medical care). Please see US EPA 2005 for distribution derivation.
Upper Respiratory Symptom Days	\$35	WTP	U.S. EPA (2005) reports an average dollar value for WTP to prevent days of three upper respiratory symptoms. Adjusting for inflation, an upper respiratory symptom is valued at \$35. Please see USEPA 2005 for distribution derivation.

Lower Respiratory Symptom Days	\$22	WTP	US EPA (2005) estimates the dollar value for WTP to avoid a LRS symptom. The value of LRS is average of 11 different types of LRS. Adjusting for inflation, the WTP value for LRS is \$22. Please see US EPA 2005 for distribution derivation.
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 <i>Occupational Employment Statistics Survey</i> (1 st quarter 2008 wages) Alameda , & Contra Costa : \$202 Marin , San Francisco , & San Mateo : \$228 Santa Clara : \$243 Napa : \$174 Solano : \$168 Sonoma : \$179
School Absence Days	\$91	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 wage 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	\$61	WTP	Applying similar methods to US EPA 2005 and Stratus 2008, using Tolley et al (1986) estimates of WTP for avoiding a three symptom combination of coughing, throat congestion and sinusitis. The estimate of WTP is \$117 (\$2008). Following US EPA 2005 and US EPA 2008, we arrive at a triangular distribution by applying the following: by definition, MRAD does not result in loss of work. Thus, MRAD WTP should not exceed a Work Loss Day (Alameda County Daily Median Wage \$202). WTP for MRAD should be higher than the WTP for a single symptom (for eye irritation).
Cancer	\$1,750,000	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 were 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 lost of production to society. One recent study, Industrial Economics (2008), derived a similar value.

*All values have been adjusted to 2008 dollars and adjusted to Bay Area values where data is available. (CPI-U for SF-Oakland-San Jose)

5.3 Valuation of Greenhouse Gas Reductions

Greenhouse gases (GHGs) that contribute to climate change and global warming are one of the four categories of pollutants specifically targeted in the 2010 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; i.e., the benefit of reducing one ton of GHG (CO₂-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.

After reviewing the literature of studies that have been performed to estimate the cost or value of GHG emissions, we conclude that \$28 per metric ton of CO₂-e represents a reasonable value for GHG reductions for purposes of the MPEM. 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.¹⁷ These gases vary significantly in terms of the volume (mass) of emissions as well as their specific global warming potential (GWP) expressed on a CO₂-equivalent (CO₂-e) basis. It should be noted that, to the extent that 2010 CAP control measures may reduce emissions of other (non-Kyoto Six) GHGs, or other pollutants such as black carbon¹⁸ that are not included in our calculations, the MPEM may underestimate the benefit of control measures in protecting our climate.

¹⁷ The Kyoto Six GHGs are CO₂, methane, nitrous oxide, hydroflourocarbons, perflourocarbons, and sulfur hexafluoride.

¹⁸ Although there is strong evidence black carbon or “soot” contributes to radiative forcing, it has not yet been formally designated as an agent of climate change by the Intergovernmental Panel on Climate Change (IPCC). District staff will continue to monitor the research in this area.

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 change¹⁹, 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,²⁰ 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.²¹ 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.

¹⁹ The US Science Change Program summarizes many of the changes already taking place, including a rise of 2oF 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.

²⁰ 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.

²¹ 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 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 (SCC) 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, CO₂, 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 time frame. 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. To avoid this scenario, Weitzman (1998) recommends that the far distant future should be discounted at a very low rate. For this reason, we apply a discount rate of 1% to estimate the present value of benefits from reducing GHGs for the MPEM.

5.3.5 Value Assigned to GHG Emissions

For the purpose of MPEM we use a recent meta-analysis by Tol (2008) which synthesizes the results of a wide range of individual studies. Tol (2008) is an update to an earlier meta-analysis (Tol 2005) of the social cost of carbon (SCC) in which 211 estimates of the social cost of carbon are analyzed and more advanced statistical analysis is performed. Using Tol 2008 results and updating to 2008 dollars, the median of a Fisher-Tippet probability distribution²² from peer reviewed estimates with 1% discount rates gives \$28 per ton of CO₂-equivalent emissions (this equates to \$102 per ton of carbon emissions).

²² This type of distribution is used in assessing risk for highly unusual events.

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.

In-house enhancements:

Spatial distribution of emissions reductions: For the 2009 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 2009 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 both evaluating emission reductions by season and also running the models for a full year.

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 2009 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 2009 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. It would be desirable to include a wider range of air toxics and GHGs.

Morbidity from toxics: For the 2009 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 direct decouple method to obtain more accurate sensitivities (especially for small changes in emissions).
- Perform modeling for longer periods in order to provide better estimates on annual average basis. Ideally, several full years would be modeled.
- Current models offer averages within areas of a square kilometer or greater. Potentially, neighborhood-scale models could be developed that estimated 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.

Enhancements based on External Information:

Wider range of health effects: For the 2009 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 2009 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 ultra-fine 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 2009 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 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 and 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%, say, 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 µg/m³ (Motallebi et al.2003), oceanic sea salt increases the background for the Bay Area to perhaps 3.5 µg/m³.

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 1, San Jose's benzene concentration data for 2003-07 has been compared with its 1987-91 benzene data. Due to the limited number of datapoints*, every 5th percentile is plotted: 5th, 10th, 15th, and so on up through 95th. So, 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. 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, roughly speaking, been an across-the-board reduction in benzene concentrations of 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 2 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 explainable with simple rollback. The slope $y = .23x$ 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 3 shows the trend in PM_{2.5} concentrations at Livermore, comparing measurements using a BAM monitor (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 $1.1 \mu\text{g}/\text{m}^3$. Thus, it appears that $\text{PM}_{2.5}$ 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 4 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, for Davenport, measurements that 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, which 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

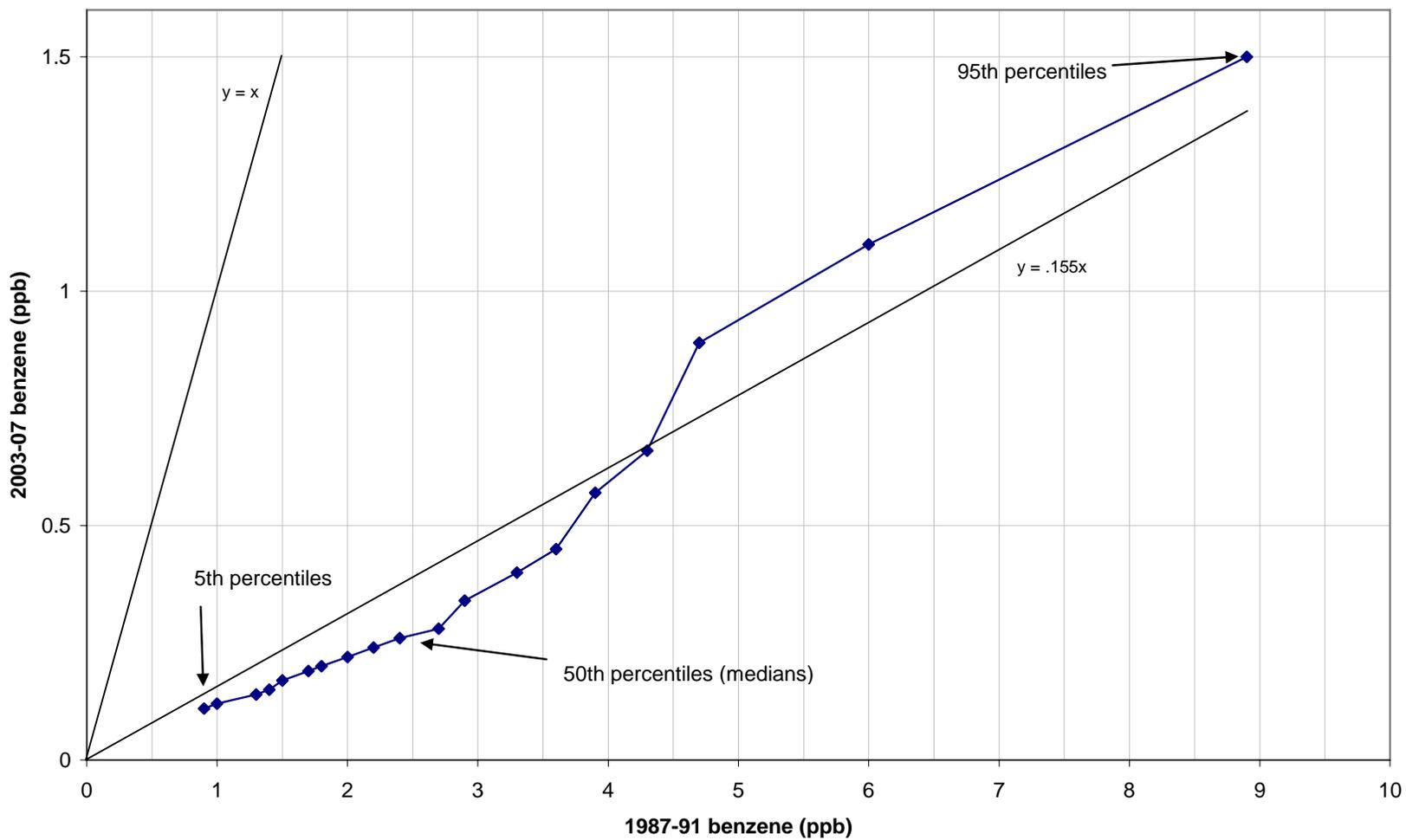


Figure A2. Trend in San Jose 1,3-Butadiene Distribution 2003-07 vs. 1990-94

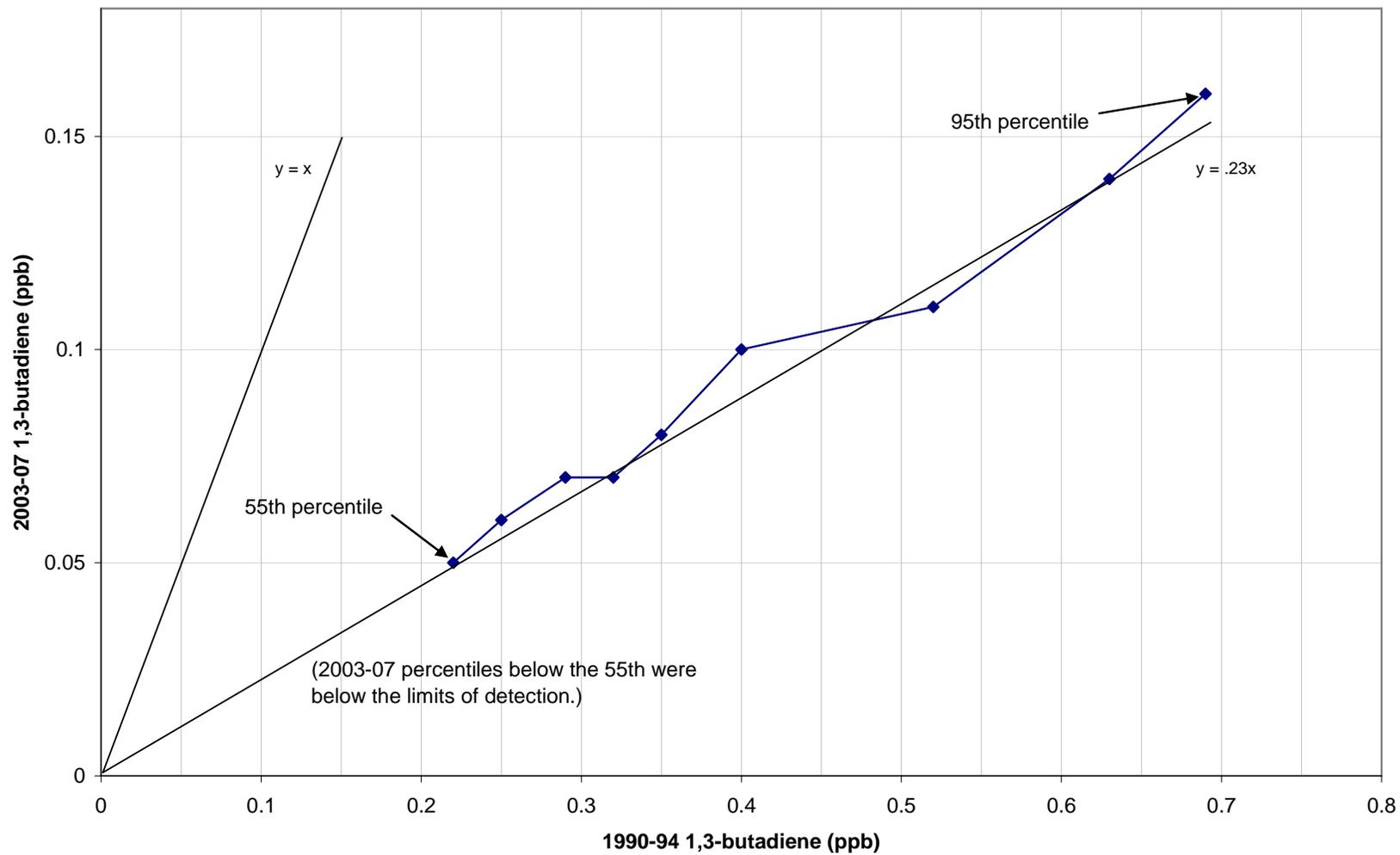
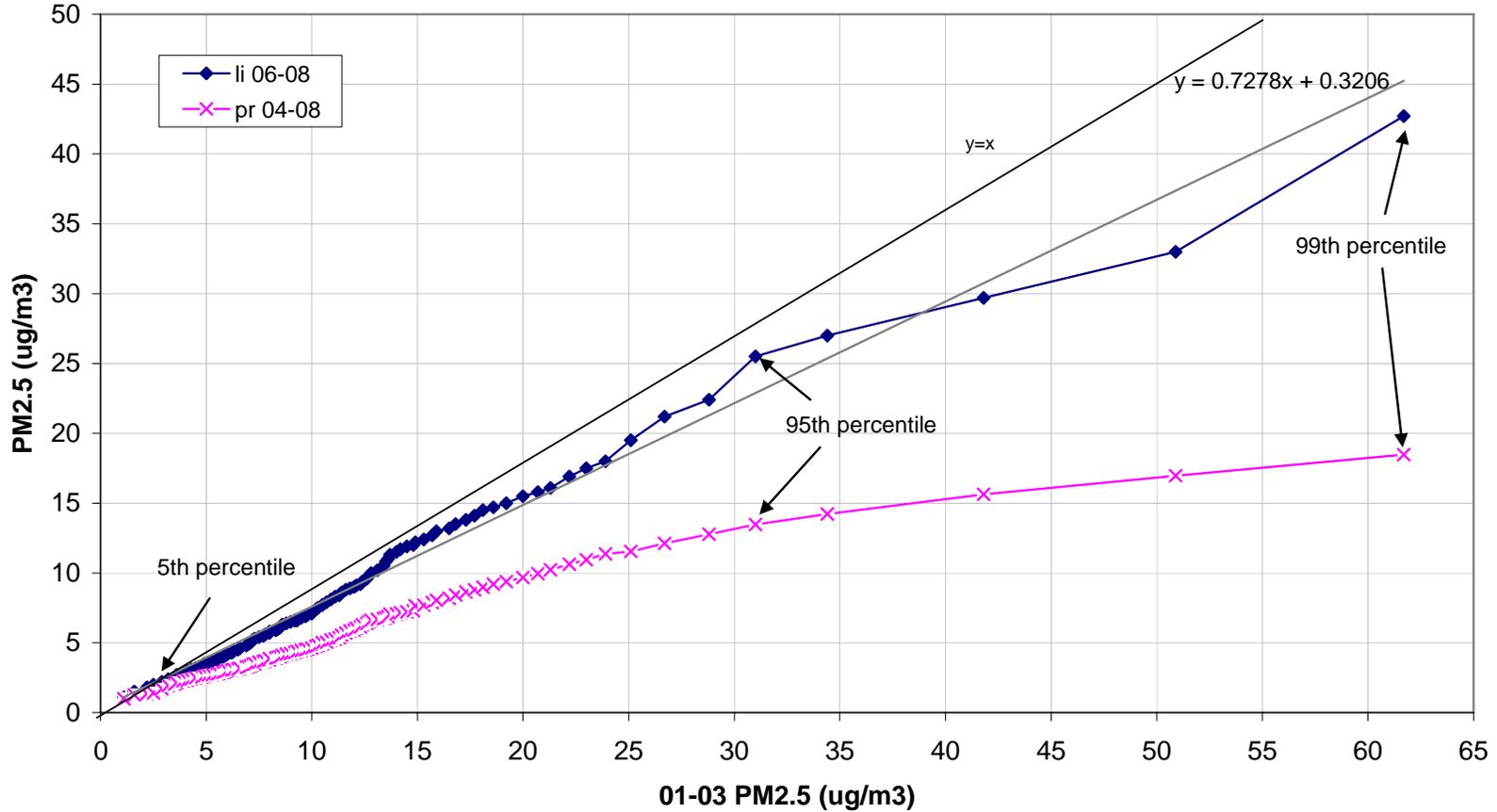
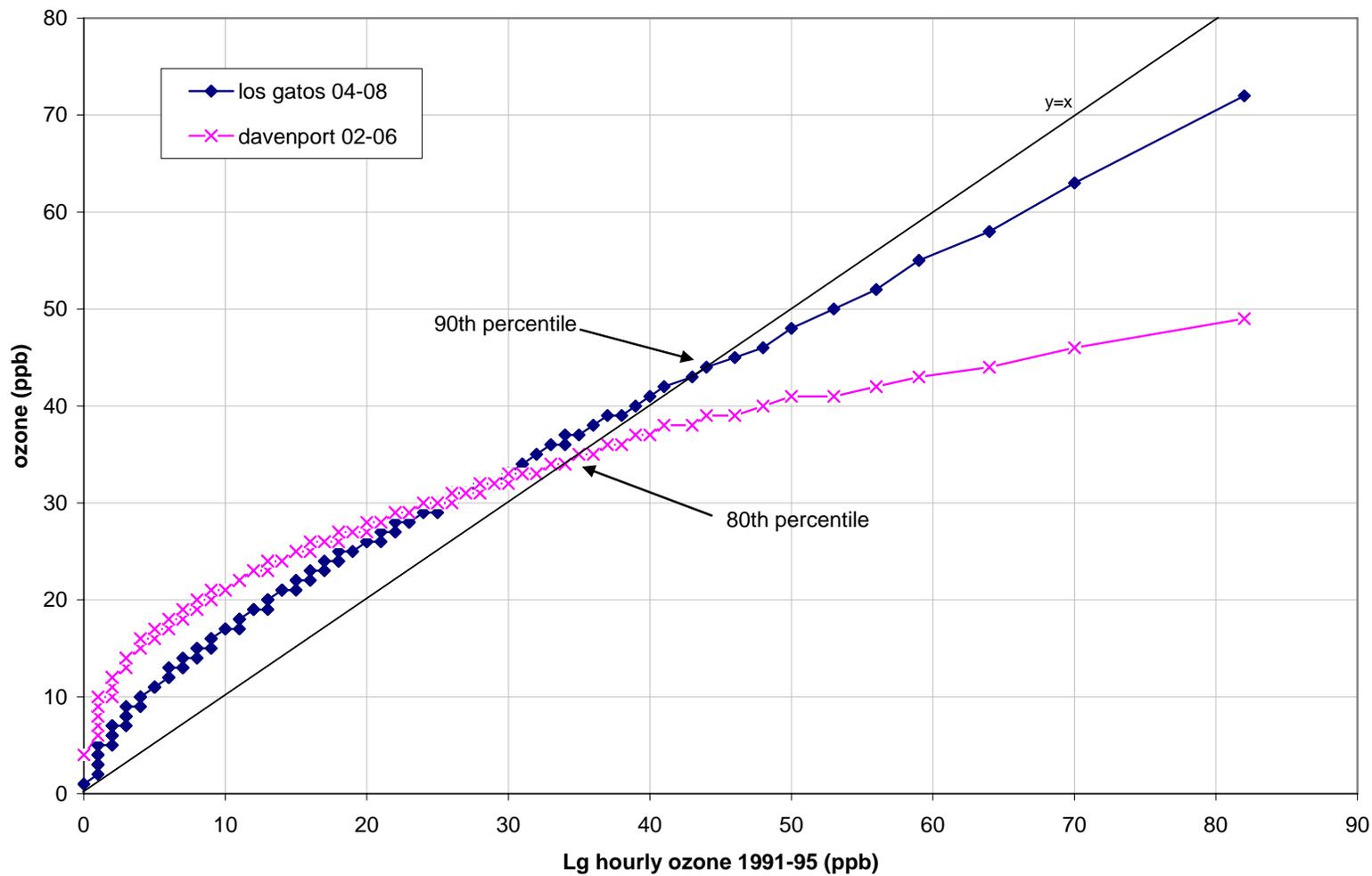


Figure A3. Trend in Livermore PM2.5 2006-08 vs 2001-03, and vs. Point Reyes "background"



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

Estimating the ozone benefit of VOC and NOx reductions

Reducing the concentrations, population exposure, and health effects related to ozone is an important goal of the Bay Area 2010 Clean Air Plan. But ozone is not emitted directly, instead forming from complex atmospheric chemical reactions that include precursors NOx and VOC. Thus the connection between a rule's effect on ozone precursors and the resulting ozone concentrations must be established.

The connection is complex. Reaction rates depend not just on the amounts of these precursors, but also their relative amounts. The effect of changes in NOx can be particularly complex: Reductions in NOx can sometimes lead to increases in ozone. Also, the ratio of NOx to VOC varies substantially around the Bay Area, as does the weather. Thus, the production of ozone varies dramatically, with some areas such as Livermore and San Martin (south of San Jose) violating national standards while other areas such as San Francisco meeting not just the national standards, but also the tougher California standards. Moreover, VOC/NOx ratios vary substantially around the Bay, so that the efficacy of reductions in NOx and VOC also varies from place to place.

Thus, it is valuable to take advantage of the District's photochemical model capabilities to provide estimates of how reductions in ozone precursors affect ozone in different locations in and near the Bay Area. The picture it provides is complex with some areas showing that reductions in NOx may result in estimated increases in ozone.

This analysis investigates the model's estimates of the effects on ozone concentrations of various combinations of 0%, 5% and 10% reductions in NOx and VOC. Regression equations were fit to each grid square in the Bay Area to estimate the effects. The equations were applied to estimate the effects of 10% VOC and NOx reductions using actual recent ozone data from Bay Area monitoring sites. Changes in population exposures were also computed.

Methods

The CAM_x photochemical model was run for 2 episodes, July 11-12, 1999 and July 31-August 2, 2000, under various scenarios: all combinations of reductions of 0%, 5% and 10% in either NOx and/or VOC. Model-predicted ozone was produced for each hour for a grid of 2x2 km squares that included the San Francisco Bay Area. The data were reduced by selecting the maximum 1-hour ozone for each day/grid square/scenario, and also limiting the data to only grid squares in the 9-county Bay Area that had population in the 2000 census.

Results

Figure B1 shows histograms of the distribution of the percent change in ozone resulting from different NO_x and VOC reduction scenarios, for one day from each episode. The two histograms in the first row show the change in ozone from a 10% reduction in NO_x only; the two histograms in the second row show the ozone change from a 10% reduction of VOC only; the third row is like the second, except that the histograms show the effect of a 10% reduction in VOC once NO_x had been reduced 10%, that is, a change from (0 VOC, -10% NO_x) to (-10% VOC, -10% NO_x); the two panels in the fourth row show the change if both NO_x and VOC are reduced by 10%.

Key observations about the histograms are as follows:

- The VOC-only reduction leads to reductions in ozone virtually everywhere in the Bay Area.
- In contrast, the NO_x-only reduction leads to ozone reductions in some areas and substantial increases in other areas.
- The reduction of VOC in the presence of a 10% reduction in NO_x, leads to very similar reductions in ozone as the reduction of VOC with no reduction in NO_x. (Row 3 vs. Row 2)
- The reduction of both VOC and NO_x leads to changes in ozone that are close to additive. If these reductions (y) are regressed on the NO_x only (n) and VOC only reductions (v), the resulting regression equation is: $y = .94n + .89v - .07$, with an R² of 99%.

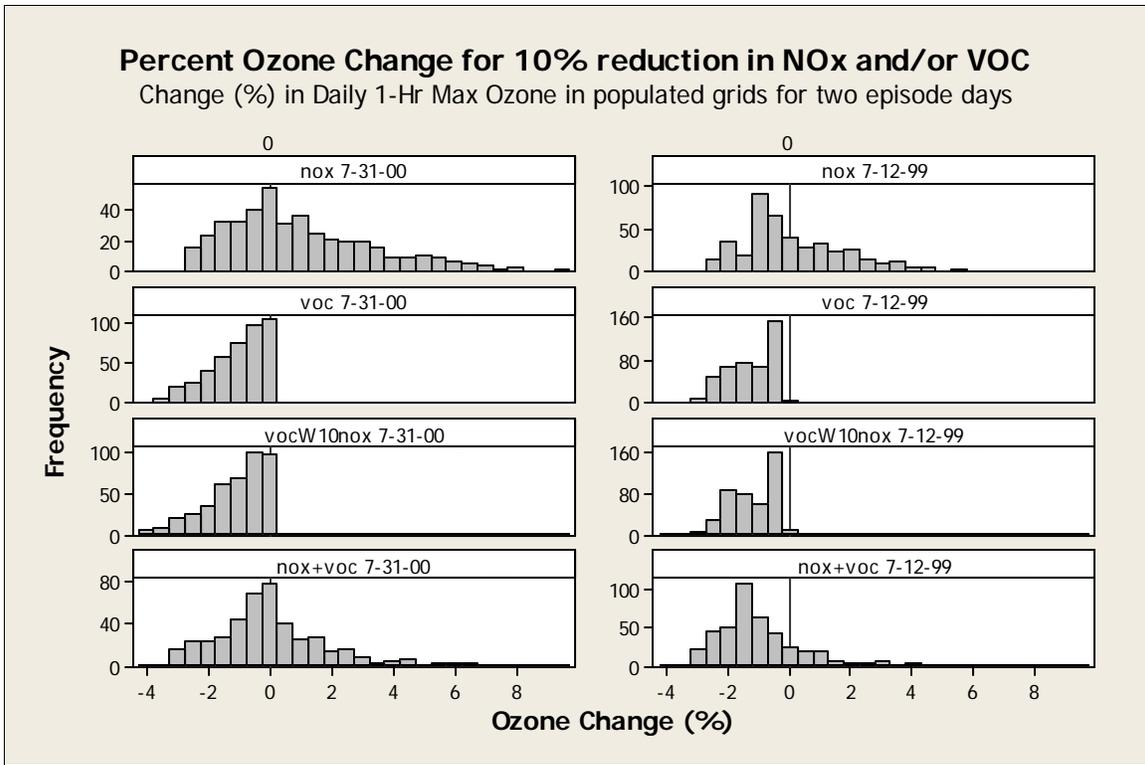


Figure B1. Percent change in 1-hour daily max ozone for various NOx and VOC 10% reductions: NOx only, VOC only, VOC reduction with 10% lower NOx, and reductions in both NOx and VOC.

Relation of the effect on ozone from joint NOx and VOC reductions to the marginal effects of NOx and VOC reductions

Generally speaking, control measures being considered for adoption may affect both NOx and VOC. Thus, it seems reasonable to estimate the joint effect of the reductions of these precursors on ozone. The goal is to estimate a surface, $dx = f(dn, dv)$, where dx is the amount of ozone reduction for a given reduction in NOx, dn , and VOC, dv .

One approach to estimating $f(dn, dv)$ would be to fit a quadratic surface in dn and dv . However, Figure B2 shows that this doesn't appear necessary. The figure compares the modeled reduction in ozone from reducing both NOx and VOC by 10% with the sum of the ozone reductions from reducing NOx by 10% (and VOC 0%) plus the ozone reductions from reducing VOC by 10% (and NOx 0%).

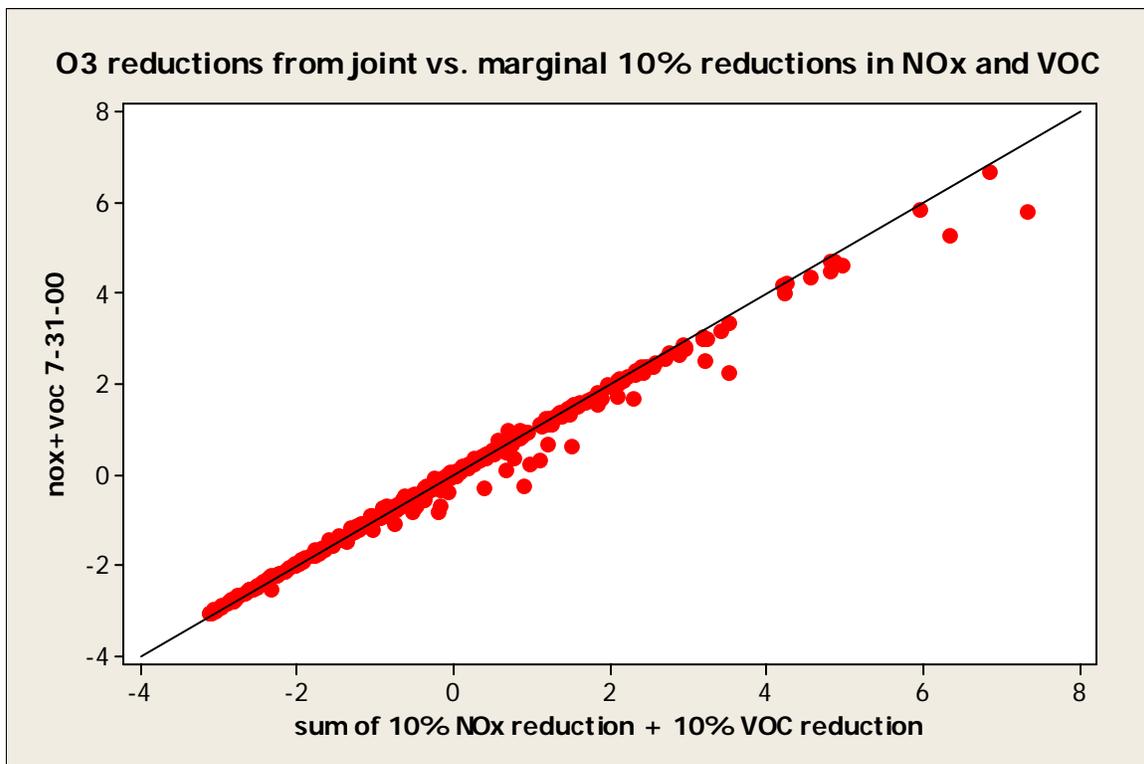


Figure B2. Ozone reductions from joint vs. marginal 10% reductions in NOx and VOC. A $y = x$ line is shown for comparison.

The figure shows a relationship that is not only highly linear, but also very close to the one-to-one line. Thus, to a good approximation, we have $y = g(dn) + h(dv)$.

Background Ozone and the regression model

Background ozone is a key complication both in predicting how ozone is affected by its precursors, and also in ozone policy. Specifically, there is a global background of ozone, partly natural, that would leave the Bay Area with substantial ozone concentrations,

peaking in the range of 40 – 50 ppb, even in the total absence of local emissions of NOx and VOCs.

NOx quenching (ozone titration) actually reduces ozone concentrations locally, so that eliminating NOx pollution can paradoxically increase ozone, as demonstrated in Appendix A.

This led us to consider a model where the magnitude of the impact of reductions in NOx and VOC emissions was proportional not to the ozone concentration itself but to its distance from background. Specifically, we fit the model:

$$y = (x - 50) (a \, dn + b \, dv),$$

where y was the change in ozone (ppb), x was the initial ozone concentration (ppb), and dn and dv were the percent reductions in NOx and VOC.

Results

The regression was performed on the data pooled from all 9 model runs for the 5 modeled episode days, so that each regression had 45 observations. The median R² value was 0.61, and 90% of the R² values were at least 0.30.²³

The VOC coefficients averaged 0.26, with an interquartile range of 0.12 to 0.37. Over 90% of the slopes were statistically significant. For NOx, the mean and median were actually negative, -0.12 and -0.16, respectively, with an interquartile range of -0.40 to +0.25. Over 90% of the NOx coefficients were statistically significant.

²³ Technically, the values computed were not R², because the regressions were fit without an intercept. If intercepts were included, the R² values would be somewhat larger.

Appendix C

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, \dots, x_{365} are the daily PM_{2.5} concentrations and $f(x)$ is the effect function, our assumption is:

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

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 the acute MI. Using Peters (2001), the C-R function is proportional to $e^{0.2412x} - 1$. Using PM_{2.5} measured at San Jose, the values for 2007 for the lh and rh 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 MPPEM, 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 rhs of C1 is 0.02995 compared with 0.02975 on the rhs, an underestimate of less than 1% and similarly for 2007. This is because $e^x \cong 1 + x$ for x near 0.

Appendix D 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, one where they analysed 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). 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:

$$y_i = y_0 + b(x_i - t)^+ + e_i \quad (D1)$$

for $i = 1, 2, \dots, n$ 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 $z \leq 0$.

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

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

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 E

PM Concentrations as a Function of Emissions: CMAQ Model Results

This appendix explains how changes in PM concentrations for changes in emissions are estimated in the MPEM. These estimates are based on runs of the CMAQ model where PM was simulated first from a base-case inventory and then from other inventories where emissions of one or more categories were reduced by 20%.

E1. Introduction

Ambient PM is a complex mixture of compounds from many sources and composed of many different things. In the MPEM we focus on three components of PM that encompass about 90% of the anthropogenic PM in the Bay Area: directly emitted combustion-related PM, ammonium nitrate, and ammonium sulfate.

The emission-concentration relationship for directly emitted PM appears straightforward. But ammonium nitrate and sulfate are for the most part formed in the atmosphere in processes that are complex both chemically and also in how the particles are formed. Here the model springs some surprises.

E1.1 The CMAQ Model

The CMAQ model simulated two wintertime periods: 12/17/00 – 1/11/01 and 12/26/06 – 1/11/07. The model covers a large area that includes the San Francisco Bay Area and is divided into a 185x185 grid of 4 x 4 km cells. There are also many vertical layers, but for this analysis we only consider the surface layer. The model provides hourly output. For this analysis, we considered 24-hour averages.

The inventory emissions are broken down into over 40 categories of compounds shown in Table E1.

Six sensitivity runs were made, five with 20% reductions in NO_x, VOC, ammonia, particulates, sulfur gases, individually, and a sixth with 20% reductions in all categories simultaneously. Table E1 shows which compounds were included in which sensitivity runs.

The output of the CMAQ model is also speciated into a number of compounds. For the MPEM, we are interested in concentrations of the following: elemental carbon (EC), organic carbon (OC), nitrate (NO₃), sulfate (SO₄), and all fine PM (PM_{2.5}).

E2. PM Concentrations and Directly Emitted PM_{2.5}

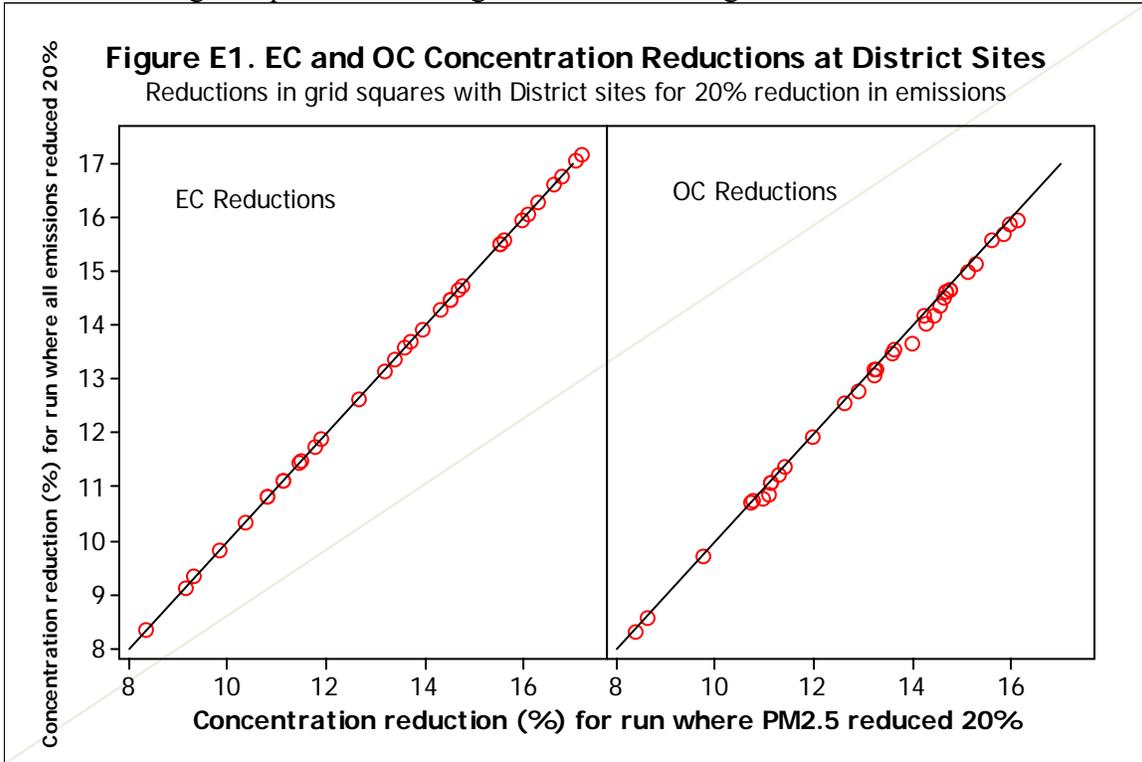
Although there are a number of directly emitted PM_{2.5} compounds, we confine ourselves to carbonaceous compounds, which are mostly formed from the combustion of carbonaceous compounds, and also from char-broiling meat.

Table E1. CMAQ model emissions inputs and groups for sensitivity runs.

Abbreviation	Emissions Category	Sensitivity Run
ACET	Acetone	2
ALK1	Alkanes, non-aromatics, $kOH < 5e2$ (ppm*min) ⁻¹	2
ALK2	Alkanes, non-aromatics, $5e2 < kOH < 2.5e3$ (ppm*min) ⁻¹	2
ALK3	Alkanes, non-aromatics, $2.5e3 < kOH < 5e3$ (ppm*min) ⁻¹	2
ALK4	Alkanes, non-aromatics, $5e3 < kOH < 1e4$ (ppm*min) ⁻¹	2
ALK5	Alkanes, non-aromatics, $kOH > 1e4$ (ppm*min) ⁻¹	2
ARO1	Aromatics, $kOH < 2e4$ (ppm*min) ⁻¹	2
ARO2	Aromatics, $kOH > 2e4$ (ppm*min) ⁻¹	2
BALD	Aromatic aldehydes	2
CCHO	Acetaldehyde	2
CCO_OH	Acetic acid	2
CH4	Methane	2
CO	Carbon monoxide	
CRES	Cresols	2
ETHENE	Ethene	2
GLY	Glyoxal	2
HCHO	Formaldehyde	2
HCOOH	Formic acid	2
IPROD	lumped isoprene produces	2
ISOPRENE	Isoprene	2
MACR	methacrolein	2
MEK	Ketones, non-aldehyde oxygenates $kOH < 5e-12$ cm ³ /mol/sec	2
MEOH	Methanol	2
MGLY	Methyl glyoxal	2
NO	nitric oxide	1
NO2	nitrogen dioxide	1
OLE1	Non-ethene alkenes, $kOH < 7e4$ (ppm*min) ⁻¹	2
OLE2	Alkenes, $kOH > 7e4$ (ppm*min) ⁻¹	2
PEC	PM elemental carbon	4
PHEN	Phenol	2
PM10	PM10	4
PM2_5	PM2.5	4
PMC	PM coarse fraction	4
PMFINE	PM fine fraction	4
PNO3	PM nitrate	4
POA	PM primary organic anthropogenic	4
PROD2	Ketones, non-aldehyde oxygenates $kOH > 5e-12$ cm ³ /mol/sec	2
PSO4	PM sulfate	4
RCHO	Lumped aldehydes with 3+ carbons	2
RCO_OH	Higher organic acids	2
SO2	Sulfur dioxide	5
SULF	Sulfates (SO ₃ or H ₂ SO ₄)	5
TRP1	Terpenes	2

Sensitivity Run: 1 NOX
 2 VOC
 3 Ammonia
 4 Particulates
 5 Sulfur (gas)
 6 All

One sensitivity run (4 in Table E1), reduced all directly emitted PM_{2.5}. There was also a run where all emissions except for CO were reduced 20%. Figure E1 shows the percent reductions for grid squares containing District monitoring sites.



The concentration reductions range between 8% and 17%. There is a near-perfect correlation and 1-1 relation between the reductions from the 20% PM_{2.5} reduction run (run 4) and the 20% run where all emissions were reduced (run 6), so that reductions in other species have no impact on changes in EC and OC concentrations, for all intents and purposes. In other words, for the purposes of the MPEM, we will assume that a 20% reduction in EC causes the modeled reductions in EC concentrations and ditto for OC.

E2.1 Comparisons with ambient EC and OC

Ambient EC/OC data overlapping the sampled period is limited. The District has made EC and OC measurements for 2 days at 8 sites within the dates of the 2006-07 simulation. The correlations between ambient and modeled values are not high, but the average values are similar, although not much can be concluded from such a limited sample. Thus, in the absence of more information, we assume that the modeled EC and OC concentrations provide unbiased estimates of the ambient EC and OC concentrations.

Table E2. Comparison of ambient and modeled OC and EC

	OC ($\mu\text{g}/\text{m}^3$)		EC ($\mu\text{g}/\text{m}^3$)	
	12/31/2006	1/6/2007	12/31/2006	1/6/2007
Ambient*	8.3	5.5	2.7	1.9
Modeled	8.0	4.7	1.9	1.1

* Average of 8 site concentrations.

Extending modeled concentrations to a full year

The health effects used in MPEM require annual PM data, or at least an annual average. Ideally, CMAQ modeling would be done for a full year or at least for representative weeks in each season. But CMAQ modeling simulated PM only for wintertime, the period when PM averages are highest.

EC/OC data are available for the full year, however, so that a rough conversion can be made. Table E3 shows EC, OC and TC means for January+December vs. the full year for 2006-07.

Table E3. Winter/Full Year ambient EC/OC comparison

	n	EC($\mu\text{g}/\text{m}^3$)	OC($\mu\text{g}/\text{m}^3$)	Estimated Anthropogenic OC	Estimated Anthropogenic Carbonaceous PM _{2.5}
Full Year	150	1.09	3.87	0.8x3.87	1.09 + 0.8x3.87/.833 = 4.81
January+December	23	2.09	6.02	0.9x6.02	2.09 + 0.9x6.02/.833 = 8.59
FY/J+D ratio		.524	.643	0.57	0.56

The simplest method would be to assume that the wintertime relationships between EC and OC carried over to the rest of the year. But it's likely that secondary biogenic and anthropogenic OC is a larger percentage of all OC in the summer than winter, because there are considerably more emissions of organic carbon precursors, such as alpha pinene²⁴. If EC were a marker for primary anthropogenic carbonaceous particles, then we might assume the same ratio applied to organic carbon, so that for the full year, we'd expect $.524 \times 6.02 = 3.15 \mu\text{g}/\text{m}^3$ of the $3.87 \mu\text{g}/\text{m}^3$ was anthropogenic, or about 80% of OC. The model estimate for wintertime primary anthropogenic OC is 90%, so this doesn't seem unreasonable.

The next step is conversion OC and EC to carbonaceous PM_{2.5}. Both EC and OC measurements are literally the estimated micrograms per cubic meter of the element carbon. EC comes in this elemental form. But OC is derived from particles made of molecules where carbon is bound with other elements, especially oxygen and hydrogen. The CMAQ model computes that OC represents 60% of biogenic organic carbon particles and 83.3% of directly emitted anthropogenic organic carbon particles. Thus, dividing anthropogenic OC by .833 provides an estimate of the concentration of the carbonaceous molecules it was a part of.

The last column of Table E3 shows estimates of January+December average and full year average anthropogenic carbonaceous PM_{2.5} concentrations. The ratio is $r_0 = 0.56$. Thus,

²⁴ Another piece of supporting evidence is an unpublished analysis of individual PM_{2.5} hydrocarbon compounds measured on filters from San Jose-Jackson. The analysis showed a seasonal pattern of higher concentrations of secondary organic compounds in the summer months and lower concentrations in the winter.

for this version of the MPEM, the factor r0 is applied to the modeled estimates of anthropogenic carbonaceous PM_{2.5}:

Estimated reduction in anthropogenic carbonaceous PM_{2.5} for a 20% reduction in EC and OC

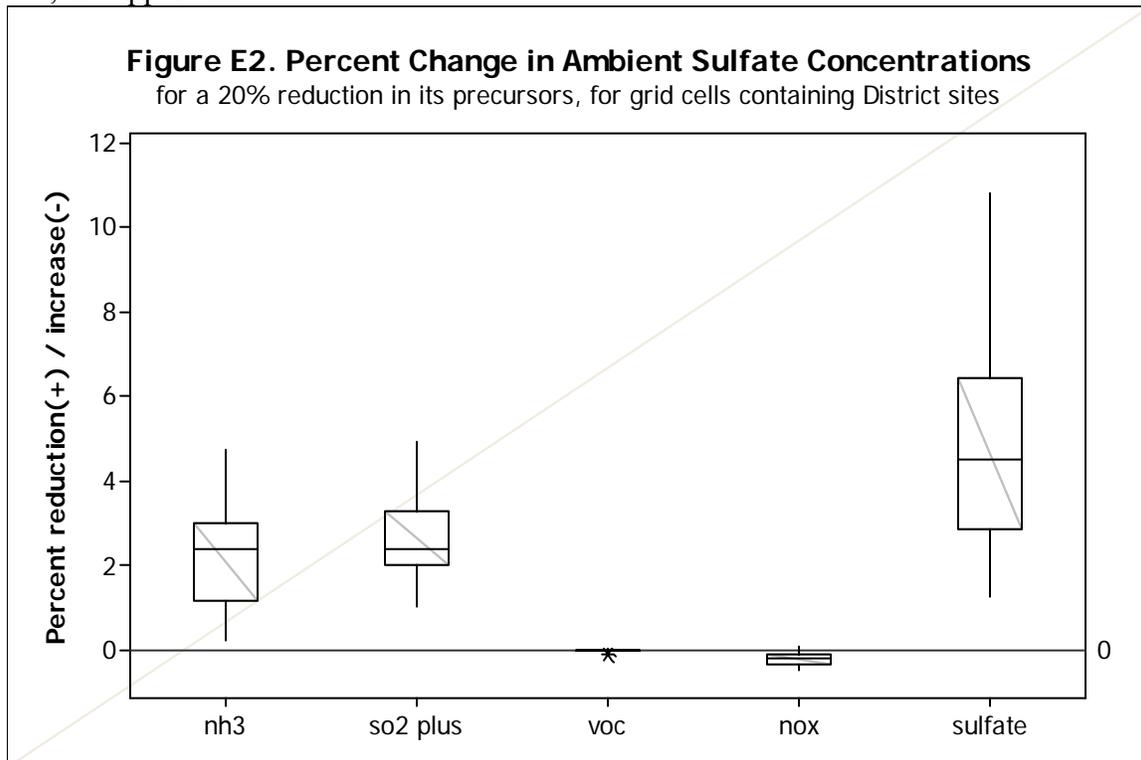
$$= 0.56 \times [EC_{\text{base}} + .9 \times OC_{\text{base}} / 0.833 - EC_{20} - .9 \times OC_{20} / 0.833]$$

Where EC_{base} and OC_{base} are the EC and OC concentrations from the base case and EC₂₀ and OC₂₀ are the EC and OC concentrations from the run with 20% lower direct PM_{2.5} emissions.

E3. Ammonium Sulfate and its Precursors

Ambient ammonium sulfate derives both from direct emissions and chemical reactions in the atmosphere. The principal reactions involve ammonia and SO₂ on water droplets, although ammonium sulfate can also be formed photochemically in gas-phase reactions.

Figure E2 shows boxplots of the reduction (or increase) in sulfate with 20% reductions in ammonia (nh3); sulfur gases, mainly SO₂ (so2 plus), VOC, NO_x, and directly emitted sulfate. The boxes show the distribution of changes in sulfate concentrations. The middle of the box is the median so, for example, the median reductions in sulfate for 20% reductions of nh3 or so2 plus are both slightly above 2%. The lower and upper sides of the box are the 25th and 75th percentiles, respectively. E.g., about 1% and 3% for nh3. The "whiskers" represent the 5th and 95th percentiles. For nh3, the lower is slightly above 0%, the upper almost 5%.



The median reduction in sulfate for a 20% reduction of all precursors is 9.6%, suggesting that much of the Bay Area's ambient ammonium sulfate is imported. Ammonium sulfate is a significant component of marine air and also of ship emissions. Measurements from Point Reyes show considerable quantities of ammonium sulfate when winds are westerly, i.e., when the air is coming off the ocean.

Individually, 20% reductions in ammonia and sulfur gases reduce ammonium sulfate modestly, 2.4% each, whereas 20% reductions in direct sulfate result in reductions averaging 4.5%. VOC has essentially no impact, while NOx reductions cause a small increase, of 0.17%.

A regression of the reductions in sulfate in run 6 vs the reductions in nh3, sulfur-gases, and direct-sulfate yields an R² of 99.9%. The regression equation is:

$$y = 0.85*nh3 + 0.90*sulfur-gases + 1.03*directSO4$$

where y is the so4 reduction in sulfate under model 6, and nh3, sulfur-gases and directSO4 are the reductions in sulfate for the individual runs: nh3, sulfur gases, and directly emitted PM_{2.5}, with all reductions in µg/m³.

The near-perfect R² indicates that the reductions are additive to a good approximation, that is, the reduction from all the pollutants is the sum of individual contributions. The nh3 and so2 slopes less than 1 indicate that the so4 reduction from reduction in these is somewhat less if considered jointly with the other factors than considered alone. Since we are considering joint impacts, the lesser slopes are appropriate, that is, for an x% reduction in nh3, we would predict a reduction in so4 of 0.85*nh3*(x/20).

E3.1 Extension the full year

Sulfate data are available from 15 Bay Area sites, sufficient to provide good geographic coverage. The data were collected on PM₁₀ filters, but comparisons with PM_{2.5} sulfate show a nearly 1-1 correspondence, suggesting that most sulfate occurs in the fine PM fraction. Data are collected on a 1-in-6 day schedule. Because of this relatively sparse data, 2 years, 2006 and 2007, were aggregated, and all January and December observations were taken to represent the modeled period.

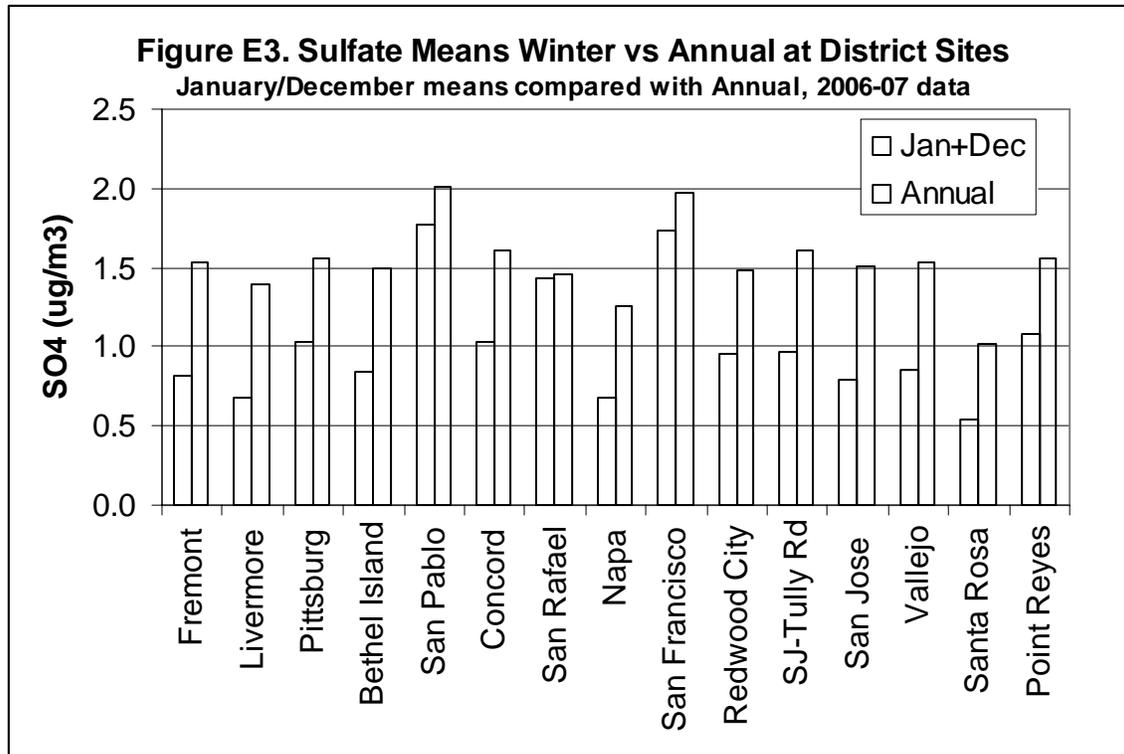


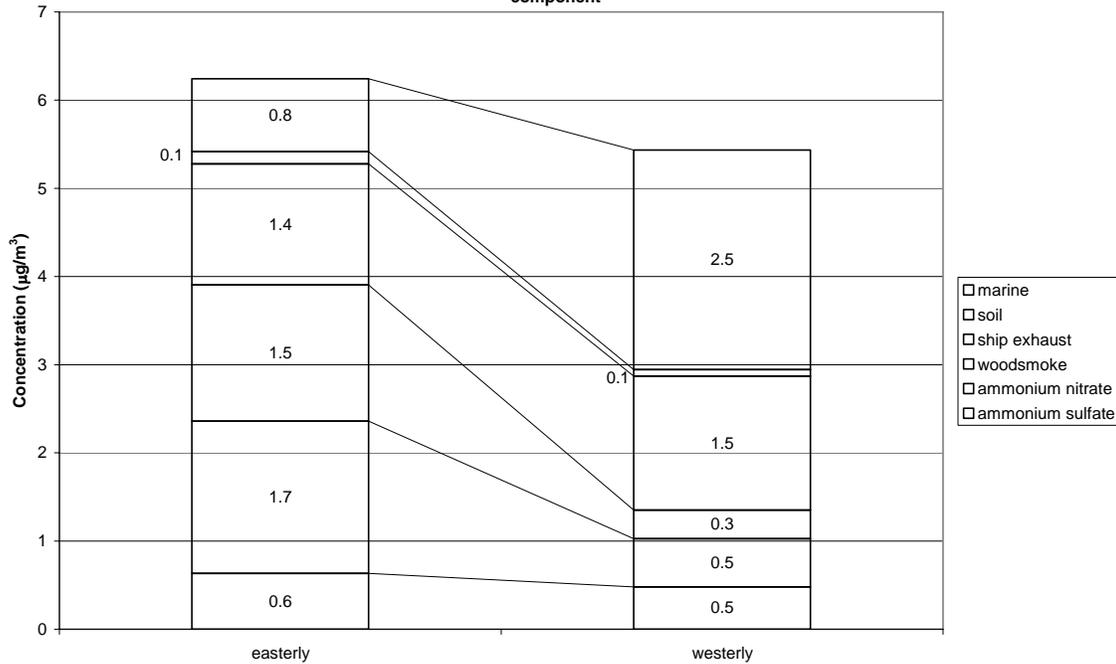
Figure E3 shows annual and January/December means for 2006-07 from the sites. Unlike many Bay Area PM_{2.5} species, sulfate concentrations are lower in winter.

A simple way to extend from winter to annual would be to scale modeled wintertime concentrations by the annual/winter ratio. But the goal of the extension is to estimate how annual reductions in sulfate precursor emissions will affect annual average sulfate concentrations.

The rationale behind scaling based on the annual/winter ratio would be if the fraction of locally produced sulfate was constant throughout the year. But this is unlikely to be the case. Winter wind patterns differ substantially from summer patterns. In winter there are frequent periods of relatively stagnant air and easterly drainage, whereas in summer, winds are most typically westerly. Thus, the Bay Area's summer air derives from the Pacific with substantial quantities of ship-produced sulfate and also naturally occurring marine sulfate (which constitutes 7.7% of sea salt).

A chemical mass balance (CMB) analysis was performed for PM_{2.5} samples collected at Point Reyes in 2005 and 2006 on a 1-in-3 day schedule. Figure E4 shows the estimated average source contributions broken down by whether the 3-day mean wind direction was easterly or westerly.

Figure E4. Point Reyes Estimated Mean PM_{2.5} Source Contributions by Wind Direction
Sources estimated by CMB, classified by the 3-day mean Point Reyes winds had an easterly or westerly component

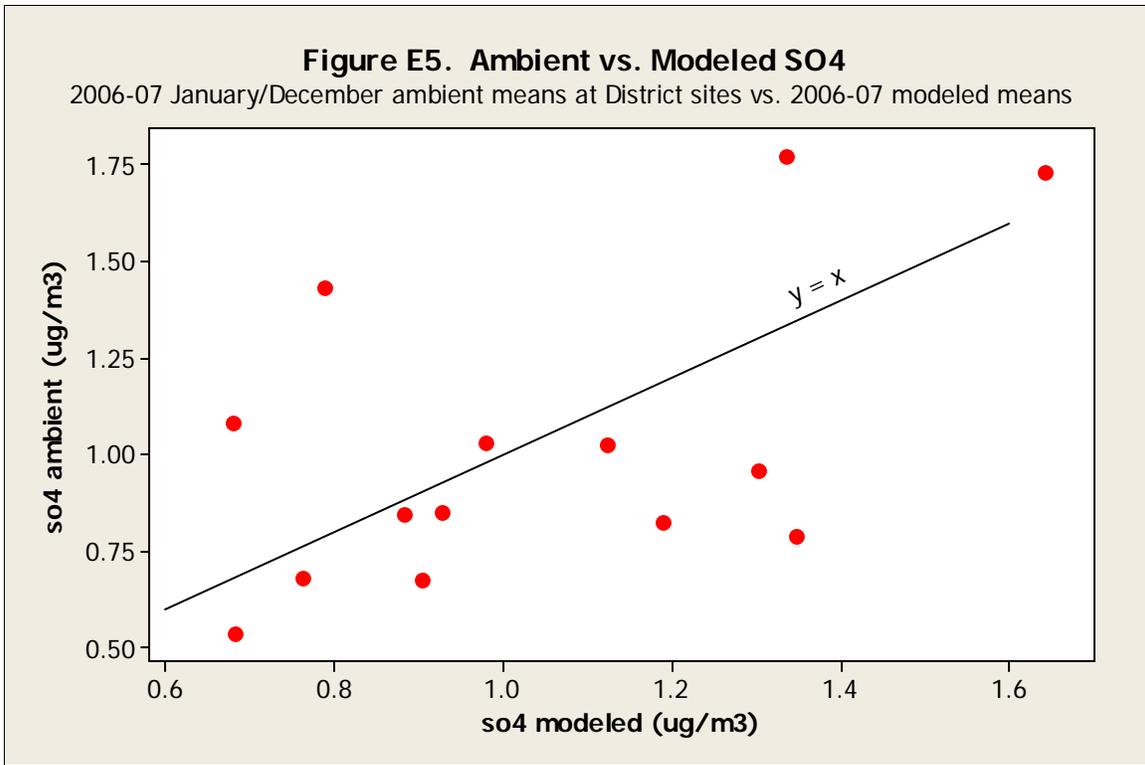


The figure shows that, overall Point Reyes PM_{2.5} concentrations were greater when the winds were easterly, that is, from further inland. But the marine component, not surprisingly, was larger, and the ship exhaust component about the same. The sulfate listed represents that left unaccounted for by the marine and ship exhaust categories.

What this suggests is that the increase in ammonium sulfate in the summer months is due to higher marine air concentrations, at least for Point Reyes. This suggests that the January/December ammonium sulfate mean may not be a bad surrogate for the annual mean of *anthropogenic* sulfate. So, in the absence of other information to the contrary, we will take the modeled concentrations as being representative of the full year.

E3.2 Ambient – Model Comparison

Figure E5 shows 2006-07 January/December sulfate means at the 15 Bay Area sites vs. modeled mean sulfate for the 2006-07 period in grid cells containing the sites. The differences are not statistically significant. Visually, the points are scattered more or less at random around the line $y = x$. Thus, there is no basis for making an adjustment to the modeled concentrations.



Thus, no additional adjustments were made either to extend to the full year or to correspond to ambient measurements.

Finally, to convert from sulfate to ammonium sulfate $(\text{NH}_4)_2\text{SO}_4$, we multiply by the ratio of atomic weights: 132/96.

Thus, to estimate reductions in ammonium sulfate concentrations from a 20% percent emissions reduction of its precursors, we use:

$$(132/96)[0.85\text{nh}_3 + 0.90\text{sulfur-gases} + 1.03\text{directSO}_4]$$

where nh_3 , sulfur gases, and directSO_4 are the modeled reductions in sulfate emissions (ug/m3) from the runs reducing ammonia, sulfur gases and PM individually by 20%.

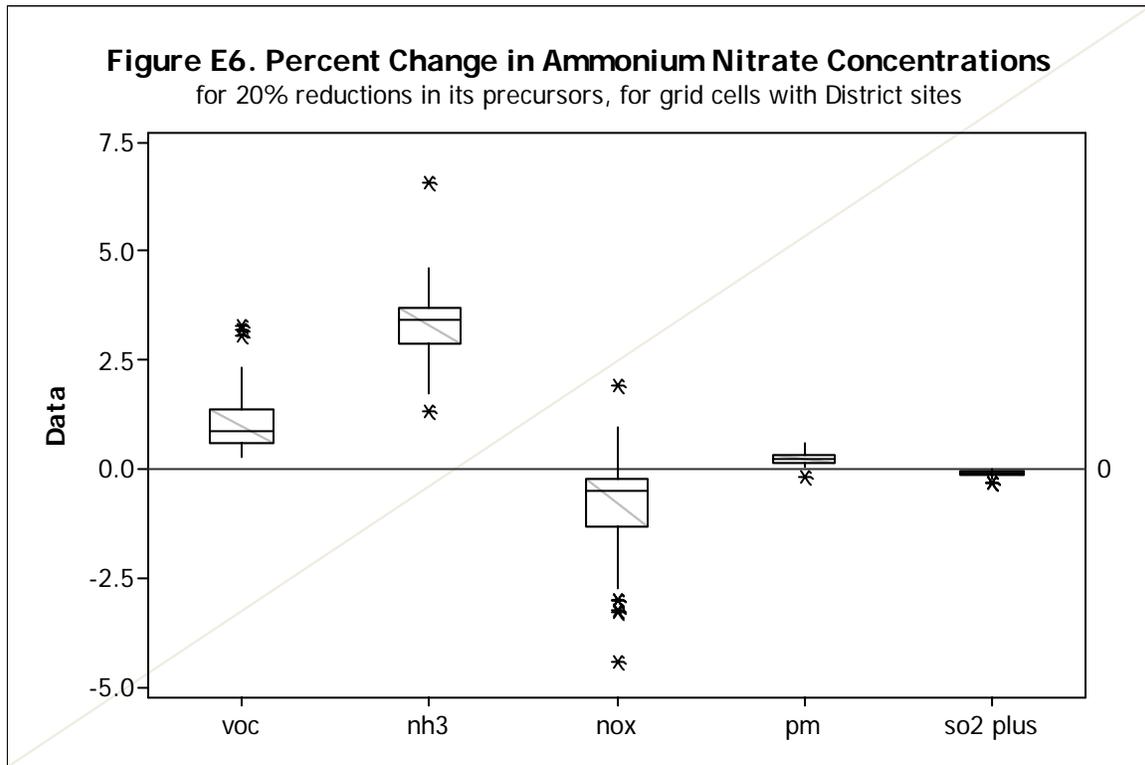
E4. Ammonium Nitrate and its Precursors

Ammonium nitrate derives almost exclusively from secondary atmospheric reactions. The key precursors are ammonia, NO_x and VOCs. The chemistry has similarities with how ozone is formed, and some of the same counter-intuitive behavior can occur.

Figure E6 shows model simulations of ambient nitrate for 20% reductions in VOC, ammonia, NO_x , and PM (directly emitted nitrate), the result limited to grid cells containing District sites. For a 20% reduction in VOC, there is a median reduction of about 1% in ammonium nitrate, with 25th and 75th percentile reductions of 0.5% and

1.5%. A 20% reduction in ammonia has a larger effect, with a median reduction of 3.4% with 25th and 75th percentiles of 2.9% and 3.7%.

But the effect of NO_x reductions is remarkable, with a median -0.5% reduction, in other words, an increase. The 25th and 75th percentiles are -1.3% and -0.2%, so that for over ¾ of the sites, modeling shows that a 20% reduction in NO_x causes an *increase* in ammonium nitrate.



Reductions of PM of 20% cause increases of a couple of tenths of a percent. This may be due to small amounts of particulate nitrate emissions. A 20% decrease in sulfur gases cause tiny increases in ammonium nitrate, about a tenth of a percent, indicating that the increase in ammonia available from being freed from the creation of ammonium sulfate is minor.

A regression of the reductions in run 6 on the reductions in the individual runs yields a regression equation:

$$y = 0.93 \text{ nh}_3 + 1.12 \text{ voc} + 1.00 \text{ nox} + 1.70 \text{ pm}$$

with an adjusted R² of 99.1%, where y is the reduction in ambient nitrate from run 6, and nh₃, voc, nox and pm, the reductions from the corresponding runs, the units being ug/m³. The high R² value indicates that nitrate formation is well-approximated by a linear function of its individual precursors. The regression equation shows that the effect of ammonia on nitrate formation is slightly less (than 1.00), the effect of voc slightly more

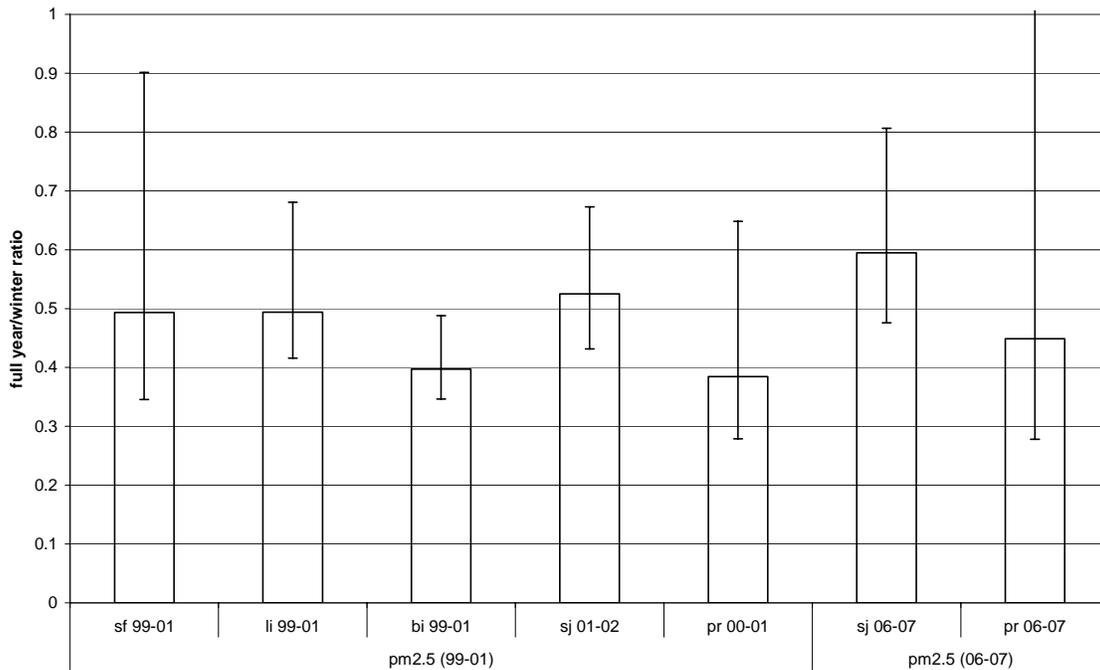
(than 1.00), and the effect of nox about the same (as 1.00), when considered jointly compared to the effects considered individually.

E4.1 Extension to full year

Unlike sulfate, nitrate values peak in wintertime. Ammonium nitrate is volatile, and converts from solid to gas at higher temperatures. Bay Area PM_{2.5} nitrate measurements are limited, being measured at only two sites, San Jose and Point Reyes until very recently. Additional measurements were made in the 1999-2001 CRPAQS study at San Francisco, Livermore and Bethel Island.

Figure E7 shows the ratios of the full-year mean to January/December means for the CRPAQS measurements and for San Jose and Point Reyes in both the CRPAQS period and also 2006-07. For the CRPAQS period, the three Bay Area sites in urbanized areas (sf, li and sj) have ratios of about 0.5. The Bethel Island ratio is lower, but may be more representative of the Central Valley. The Point Reyes ratio is lower, but its confidence interval includes 0.5. San Jose's 2006-07 ratio is almost 0.6, but its CI still includes 0.5. Point Reyes' 2006-07 is also higher than its 00-01 ratio, and again its confidence interval includes 0.5.

Figure E7. Ratios of full-year to winter mean NO₃ concentrations
ratios of means and 90% confidence bars based on bootstrap sampling



Based on this very limited information, the full-year to January/December ratio may have increased between 2000-01 and 2006-07, but the increase is not statistically significant. The value 0.5 is simple and is not contradicted by the available data, so it will be used to extend to the model results to the full year.

Finally, to convert from nitrate to ammonium nitrate (NH₄)NO₃, we multiply by the ratio of atomic weights: 80/62.

The table below shows a summary of how the different components of PM are calculated.

Table E4. Terms in the formula* for converting precursor emissions to concentrations of PM_{2.5} constituents

Ambient PM Component	Precursor ^a	Regression Factor ^b	Year Factor ^c	Conversion from species to particle ^d	20% Reduction run concentration ^e (ug/m3)	Emissions Reduction ^f (x%)
Carbonaceous PM _{2.5}	Elemental Carbon	1	0.52	1	Direct PM	x/20
	Organic Carbon	1	0.57	1/.833	Direct PM	x/20
Ammonium Sulfate	Ammonia	0.85	1	132/96	nh3	x/20
	Sulfur gases	0.90	1	132/96	sulfur gases	x/20
	Sulfate	1.03	1	132/96	Direct PM	x/20
Ammonium Nitrate	Ammonia	0.93	0.5	80/62	nh3	x/20
	VOC	1.12	0.5	80/62	VOC	x/20
	NOx	1.00	0.5	80/62	NOx	x/20

*The formula for a precursor is the product of the terms in the last 5 columns in its row of the table.

^a Species emitted as a precursor of the ambient PM compound.

^b Factor to estimate relative marginal effect of the precursor when considered jointly with other precursors.

^c Factor to estimate the change in annual concentration relative to the change in concentration for the modeled December-January period.

^d Factor to convert the modeled concentration of carbon, sulfate or nitrate into the concentration of the organic carbon, ammonium sulfate and ammonium nitrate particles.

^e Observed reductions (or increases) in OC, EC, sulfate or nitrate concentrations from the base case to the sensitivity run specified.

^f x = the ratio of the reduction in emissions of the precursor to total Bay Area emissions.

Appendix F. Estimation of the rate of conversion from NO_x to ammonium nitrate base on ambient data

The question of how NO_x emission reductions affect ammonium nitrate concentrations is a key issue. Both ARB guidance and District policies are predicated on the assumption that reducing NO_x reduces ammonium nitrate. But our preliminary PM modeling showed potential ammonium nitrate increases for decreases in NO_x in some parts of the Bay Area, as discussed in Section 2.4.10. However, analysis of ambient data suggests that NO_x reductions do reduce ammonium nitrate concentrations.

Based on a comparison of ambient San Jose NO_x and nitrate (NO₃) concentrations, both daily values and weekday and weekend averages, we concluded that a conversion factor of 0.025 was reasonable; that is, the change in nitrate concentration is predicted to be 0.025 times the change in NO_x concentration, where both are expressed in μg/m³.

This appendix presents an analysis of the evidence of ambient data on the NO_x-nitrate relationship. A factor is developed for use in the MPEM that, we believe, represents a conservative estimate of that relationship.

Background

The issue of how much a given reduction in NO_x reduces ammonium nitrate has been important in several contexts, including the use of NO_x reductions in lieu of PM_{2.5} reductions to gain PM_{2.5} offsets, and the estimation of the indirect benefits to health of reducing ammonium nitrate through reducing NO_x.

The formation of secondary ammonium nitrate is at least as complex as the formation of ozone. Rates of ammonium nitrate formation depend on the levels of NO_x, ammonia and also ROG, and also depend on meteorological conditions. Ammonium nitrate formation dynamics have been estimated through comparisons of ambient NO_x and nitrate (NO₃) levels and trends and, more recently, by computer models such as CMAQ.

ARB has used this combination of ambient comparisons and modeling to develop conversion factors. CARB (2003) used an average factor of 0.1 to convert from NO_x to ammonium nitrate for California urban areas, including 0.04 for some areas. More recently, for the San Joaquin Valley and the South Coast, CARB assumed that from 30% to 50% of the NO_x is converted to ammonium nitrate on a mass basis (CARB 2005). They cite modeling results by Kleeman (probably Kleeman 2004), who reports their model of a 1996 Central Valley episode predicted that "13% - 18% of the reactive nitrogen (NO_y = NO_x + reaction products of NO_x) emitted from local sources within the SJV was converted to nitrate..." ARB and Kleeman both stress, however, that there is a large variation in the conversion rate. Kleeman adds that "Urban areas with large amounts of fresh NO emissions converted little reactive nitrogen to nitrate..."

BAAQMD modeling suggests that NO_x reductions could actually increase nitrate in many parts of the Bay Area. However, the modeling is still preliminary. As yet, only

limited model runs have been made to test the sensitivity of the results to changes in the emissions inventory. The result is also sensitive to chemistry calculations. Moreover, we are concerned with annual average ammonium nitrate, whereas the PM model was only applied to winter periods; ammonium nitrate dynamics differ markedly at other times of the year.

Other model conclusions appear solid. The Bay Area has both more NO_x emissions and very likely less ammonia than the Central Valley. Kleeman found that reducing ammonia was effective in reducing ammonium nitrate for the Central Valley, so our modeling that shows reducing ammonia being effective in the Bay Area appears reasonable. Both Kleeman and our model also show ROG reductions reducing ammonium nitrate.

Thus, for this version of the multi-pollutant method, we will depend on the modeling results for the effects of ammonia and ROG reductions on ammonium nitrate, but we analyze ambient nitrate and NO_x concentrations to aid us in estimating the effects of NO_x reductions.

Note: In what follows, NO_x concentrations are compared with concentrations of NO₃ on a mass basis. In the Conclusion section of this appendix, the results are applied to the relation between NO_x and ammonium nitrate (NH₄NO₃), by multiplying by the ratio of ammonium nitrate mass to that of nitrate, that is, 80 to 62.

Data

San Jose – Jackson is the only District monitoring site with a significant current record of PM_{2.5} nitrate.²⁵ It is part of the national Speciation Trends Network (STN) that includes 13 other California sites such as Bakersfield, Fresno, Modesto, Los Angeles, and Sacramento. San Jose data was available through 2008, and data for the other California sites through 2006.

The 1999-2001 CRPAQS study also collected PM_{2.5} nitrate data. The Bay Area sites were Livermore, San Francisco and Bethel Island. San Jose data was also available for some of this period.

There are also District PM₁₀ nitrate measurements. These have the advantage of having been collected at a large number of Bay Area sites and having a record extending back to 1988, but the disadvantage that the measurements have large uncertainties with a tendency to underestimate nitrate, particularly in the winter.²⁶ For this reason, the District PM₁₀ nitrate measurements were adjusted upward by 20%.

²⁵ Similar monitoring began at Livermore and Vallejo in September 2008 and Oakland in Feb. 2009, so we have sufficient data to compute an annual average, but not annual trends.

²⁶ Nitrate is volatile and evaporates off filters unless the filters are refrigerated. The STN and CRPAQS PM_{2.5} were refrigerated, but the PM₁₀ filters weren't. The result is that the PM₁₀ nitrate can be considerably underestimated especially in the winter as the filters are taken from the cool outside air and then kept at room temperature before analysis.

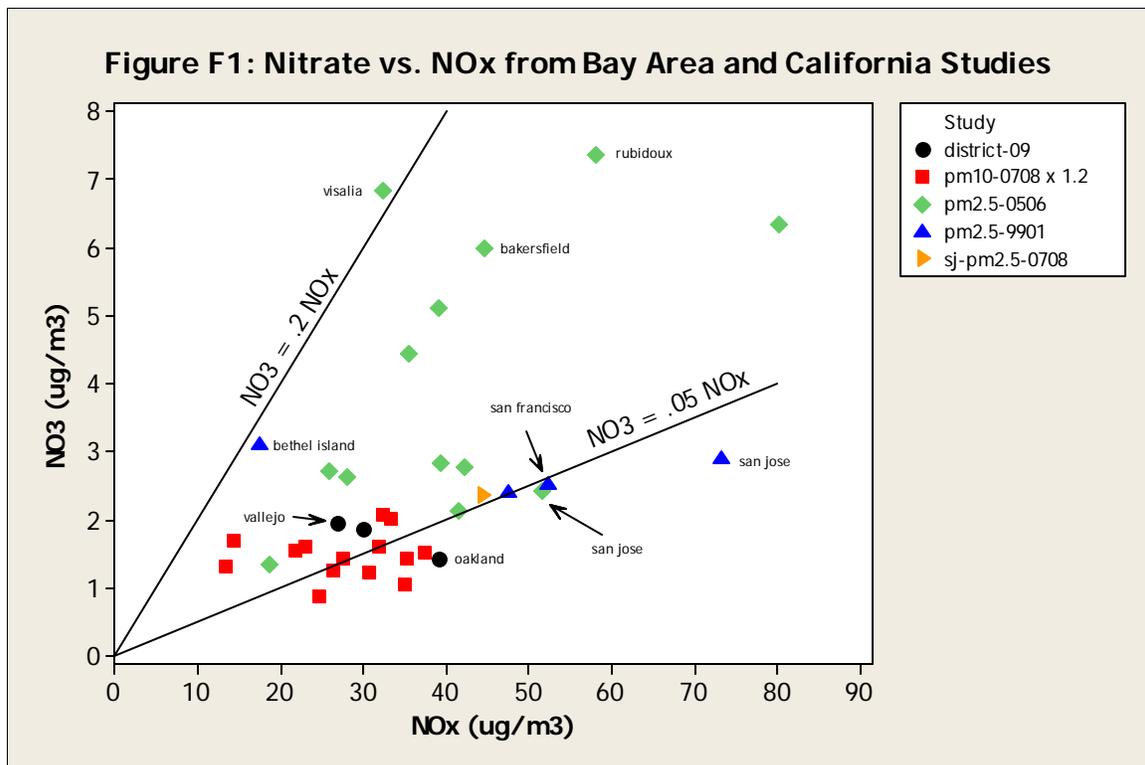
NO and NO₂ data was available for all of the site-years just described. NO_x was computed on a mass basis, namely converting from ppb to ug/m³ via: 1.065*NO + 1.633*NO₂.

Results

Figure F1 shows annual average nitrate (NO₃) vs. annual average NO_x for the site-years indicated above. The District PM₁₀ nitrate data is multiplied by 1.2 as a rough adjustment for measurement error.²⁷ Most District observations fall near the line NO₃ = 0.05 NO_x: the 2007-08 PM₁₀ District sites, represented by red squares, the blue triangles representing CRPAQS Livermore and SF values, the orange triangle representing San Jose 2008, and the 2009 SASS data represented by black circles..

The one exception is Bethel Island, both of whose values are closer to the line NO₃ = 0.20 NO_x. Bethel Island lies in the Central Valley and its high NO₃/NO_x values are similar to another rural valley site, Visalia.

The other STN sites fall between the two lines, with the SJV sites falling near the line NO₃ = 0.13 NO_x.



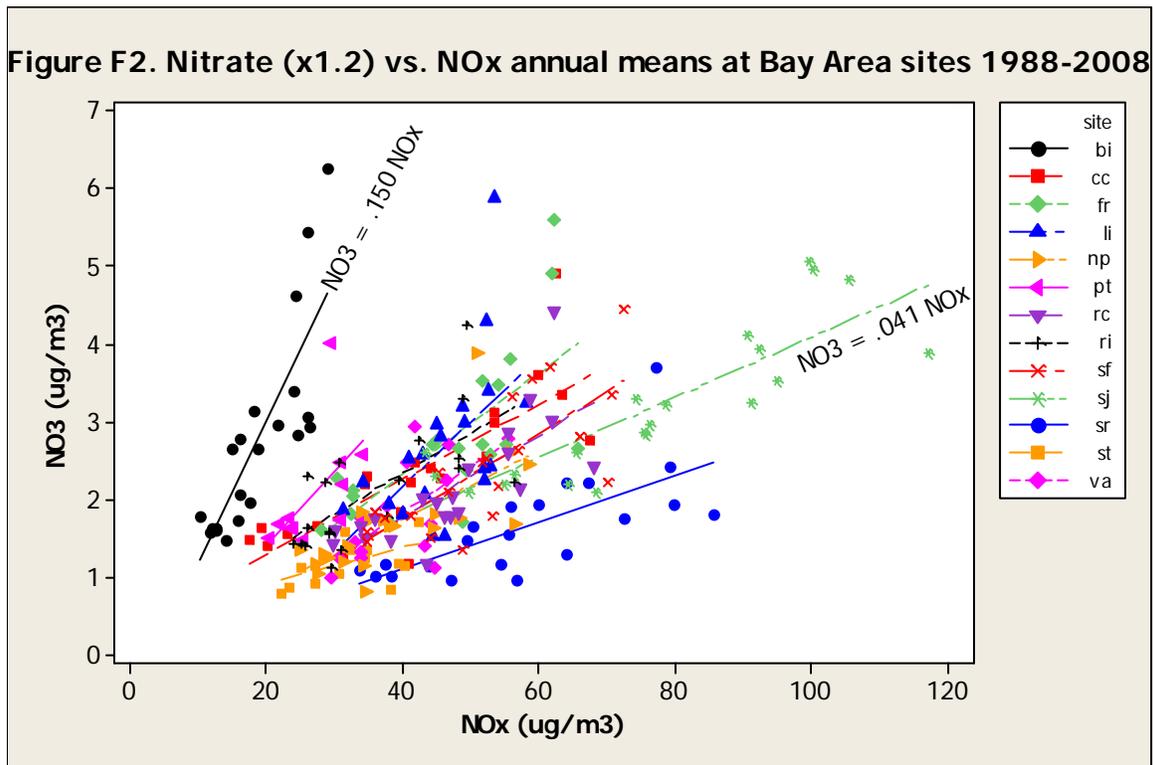
Study: district-09=District SASS monitoring, 2009; pm10-0708x1.2=District PM₁₀ nitrate times 1.2 for 2007-08; pm2.5-0506=California STN monitoring, 2005-06; pm2.5-9901=CRPAQS monitoring 1999-2001; sj-pm2.5-0708=San Jose STN monitoring for 2007-08

²⁷ The ratios of PM_{2.5} nitrate to PM₁₀ nitrate were 1.19 for San Jose 2008, 1.23 for Livermore 2000, 1.39 for SF 2000, and 1.50 for Bethel Island. BI is likely not representative of much of the rest of the Bay Area. The choice of 1.2 seemed roughly correct and, if anything, conservative.

NO₃ and NO_x annual means

Figure F2 shows annual PM₁₀ nitrate means²⁸ vs. annual NO_x means at various Bay Area sites for 1988-2008. There is a positive relationship at every site, with higher NO_x values associated with higher nitrate values. Also, both NO_x and nitrate are trending downward so the plots show that the downtrend in NO_x is matched by a downtrend in NO₃.

The figure also shows the regression lines for NO₃ on NO_x. The slope for San Jose is close to 0.04. Most other District sites are similar. Bethel Island is again an anomaly, with a slope of 0.15.



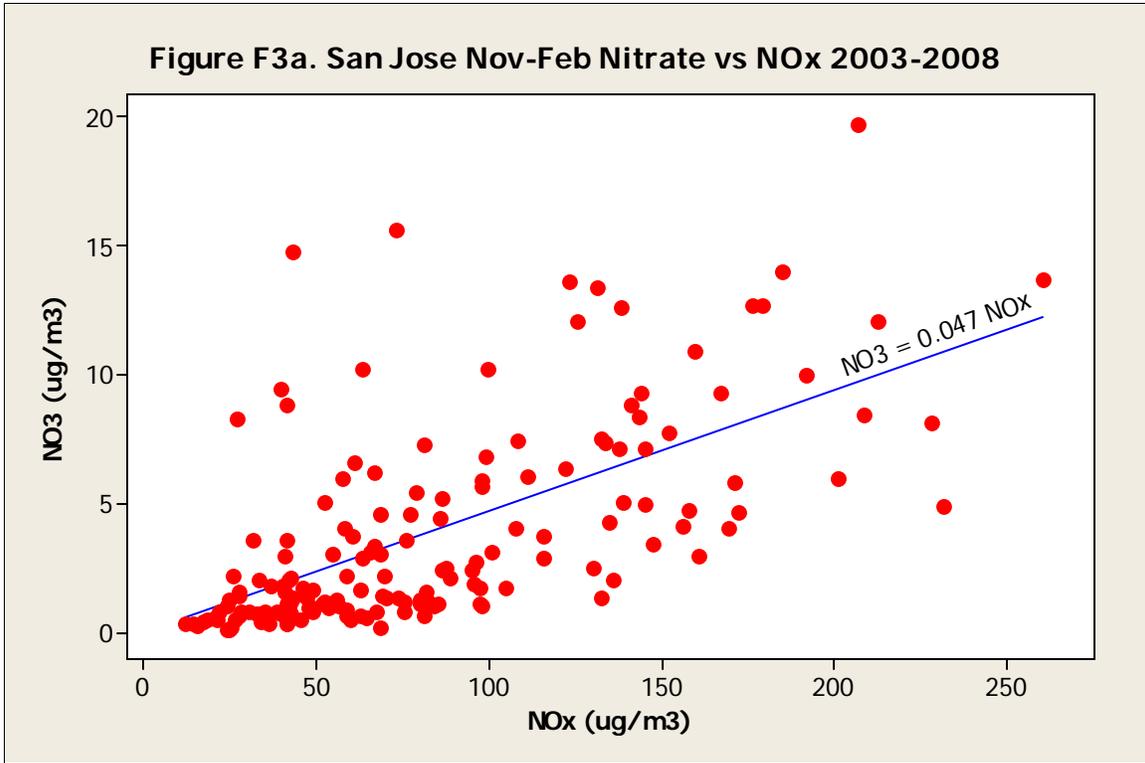
San Jose daily NO₃ vs. NO_x

Another approach is to pair daily NO₃ values with daily NO_x values. This was done for San Jose – Jackson, using all data from the site's inception in mid-2003 through 2008. Because the dynamics of nitrate formation differs substantially between winter and summer, the analysis was divided into November-February and March-October.

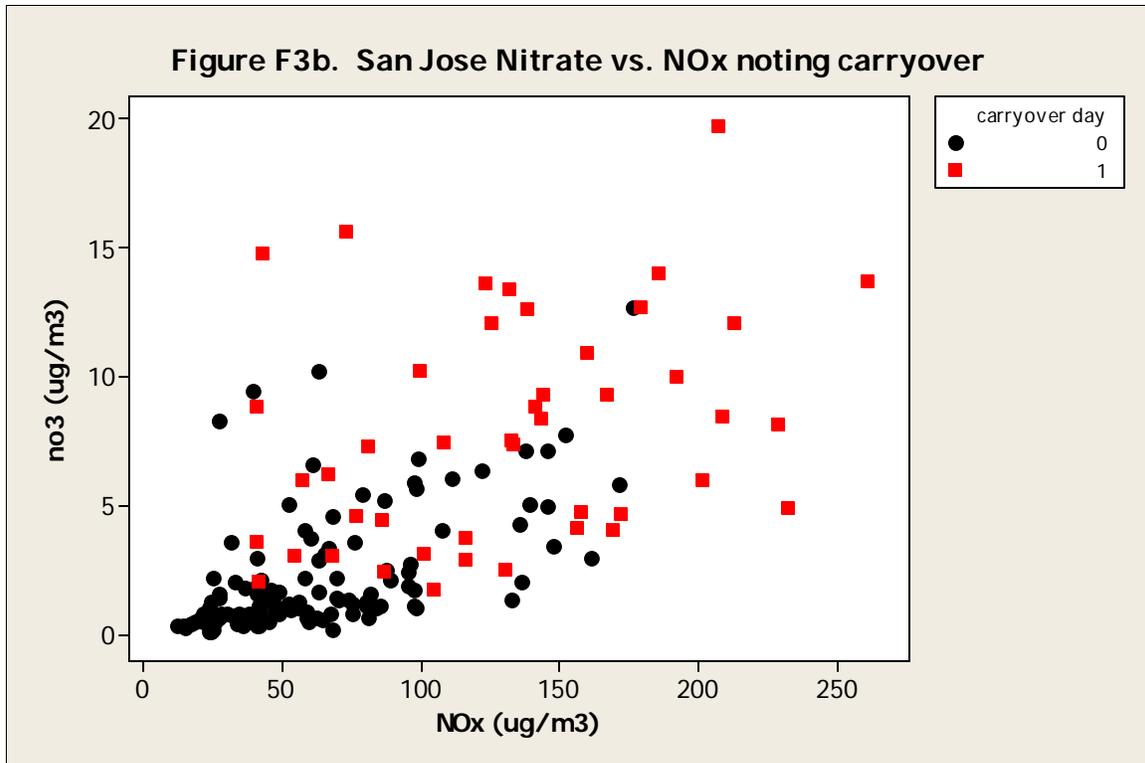
Figure F3a. shows the wintertime values. There is a significant relationship with a linear regression equation of $NO_3 = 0.047 NO_x$.

²⁸ The nitrate values are multiplied by 1.2 as in Figure 1.

But this relationship can't be totally causal, in that 1) not all the ambient NO_3 on a given day was created on that day; there can be significant carryover. And 2) atmospheric conditions such as the presence or absence of an inversion will influence all ambient concentrations similarly.



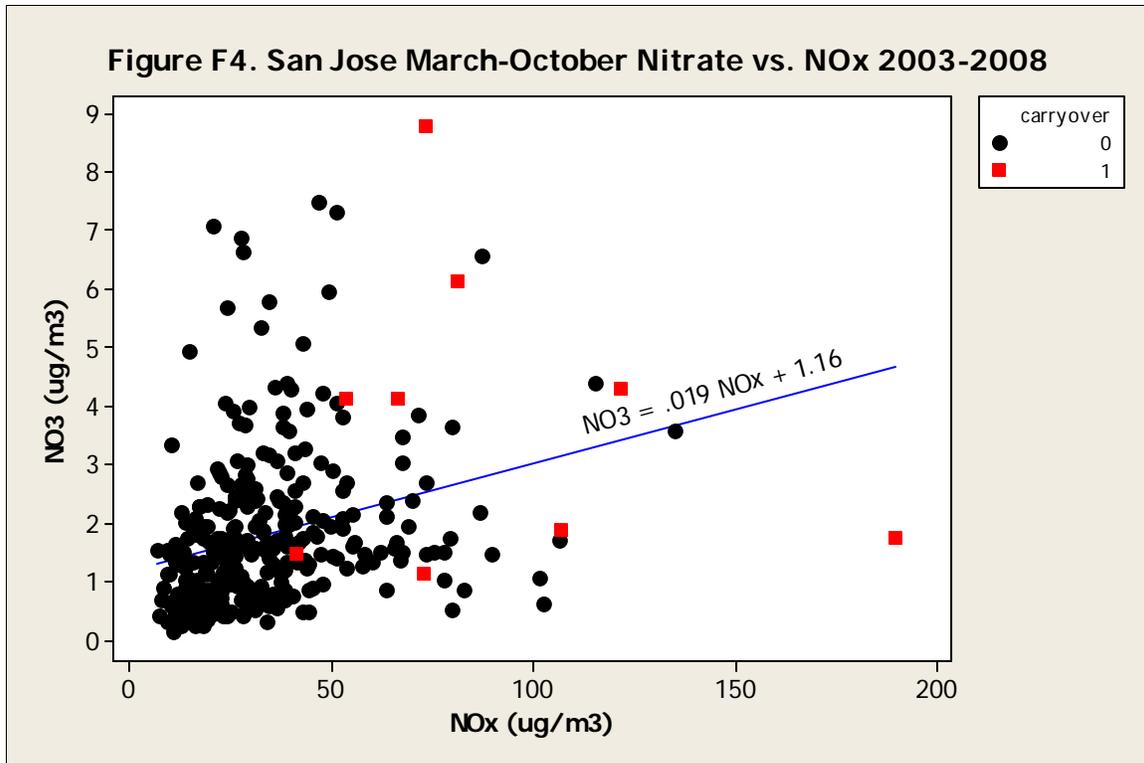
To address 1), I estimated carryover based on the number of days with light winds and little rain. Figure F3b. is the same as F3a except that "carryover days" are indicated, where carryover days are defined as having at least 3 successive days with winds < 5 mph and San Jose rain < 0.02 inches.



If a regression is fit with an indicator for carryover, the slope drops considerably. If instead of an indicator, x_2 = the number of successive days (≥ 0) with light winds and no rain along with predictor x_1 = NOx, the NOx slope becomes 0.027 for NOx rather than 0.047.

There is no perfect way to deal with issue 2), but we can use $PM_{2.5}$ measurements as a rough indicator of atmospheric stability. Since ammonium nitrate is part of $PM_{2.5}$, we subtracted it off, using $x_3 = PM_{2.5} - \text{ammonium nitrate}$ as another predictor along with x_1 (nitrate) and x_2 . With this model, the NOx slope becomes 0.001, statistically indistinguishable from 0.

Figure F4 shows nitrate vs. NOx for the rest of the year. Also shown are the carryover days as in Figure F3b. In Figure F4, however, there are virtually none, so carryover was ignored in fitting a regression. The slope of the line was 0.019.



Weekend-Weekday Comparisons

The idea for comparing weekends and weekdays is that weekend commercial activity is less than weekday. Emissions drop, with NOx emissions dropping more than ROG or ammonia emissions. Thus, we have a natural experiment to test the effect of NOx reductions on nitrate concentrations.

Specifically, we have results for San Jose both from the model and also from ambient data. The model predicts higher nitrate for the lower weekend emissions, but the ambient data show a statistically significant *decrease* in weekend nitrate. The magnitude of the decrease: a drop of 0.029 in nitrate for a unit drop in NOx, corresponds closely with the result above.

Weekend-Weekday Model Comparisons

There are two different approaches to use. One is based on comparing modeled weekdays with modeled weekends. The advantage is that it is directly what the model predicts, but the disadvantage is that there is considerable day-to-day variation from meteorology. The other approach is to use the modeled weekend vs. weekday inventory and plug the difference into the formulas in the MPEM template that convert emissions to concentrations.

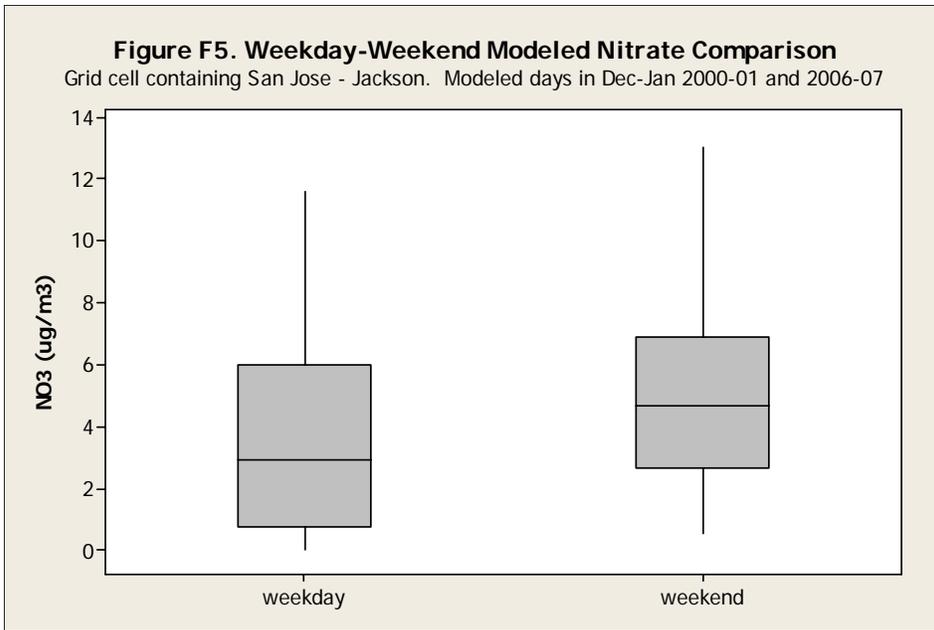


Figure F5 shows the nitrate distribution for the grid cell containing San Jose for weekdays and weekends. The figure shows that, if anything, modeled weekend nitrate is greater on weekends than weekdays, with a weekend mean of $5.2 \mu\text{g}/\text{m}^3$ vs. $4.1 \mu\text{g}/\text{m}^3$ for weekdays, and medians 4.7 and 2.9 respectively. But these differences are not statistically significant.

On a District-wide basis, the ratios of weekend to weekday emissions are:

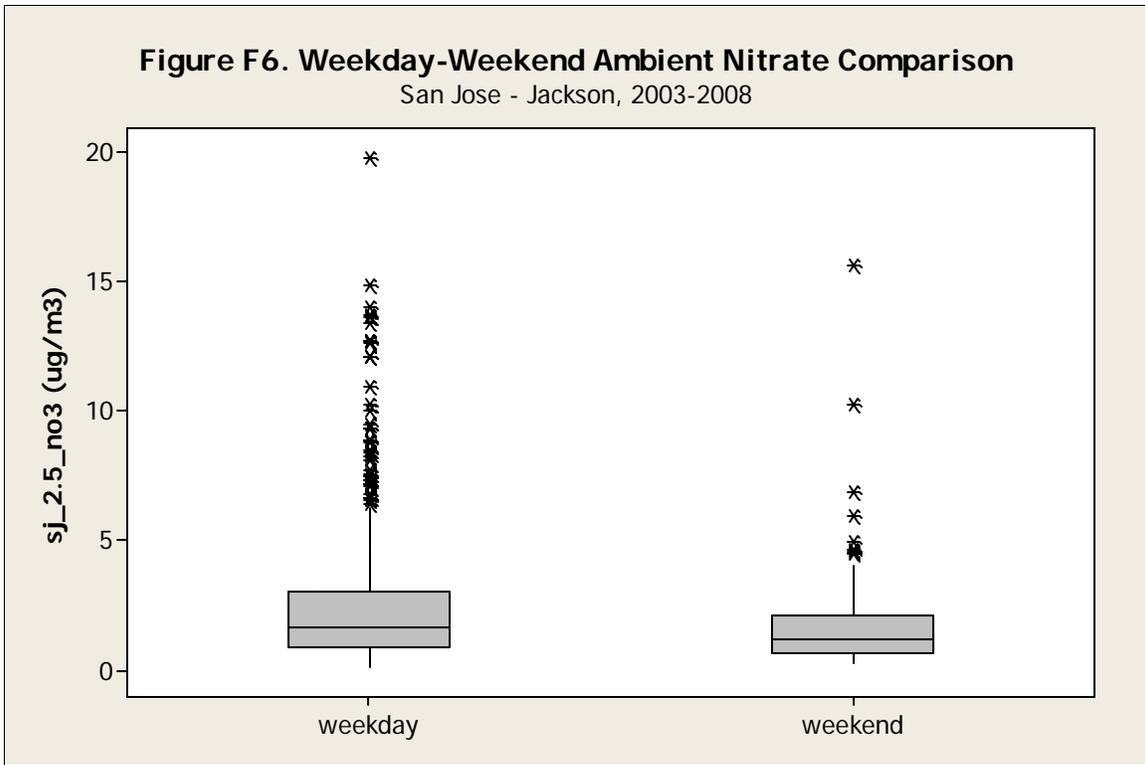
Table F-1. District-wide ratios of weekend to weekday emissions used in the CMAQ PM model

NOx	ROG	Ammonia
0.81	0.85	0.96

We applied these factors in the multi-pollutant template, that is, assuming a 19% reduction in NOx, a 15% reduction in ROG and a 4% reduction in ammonia. The template predicts a $0.08 \mu\text{g}/\text{m}^3$ increase in ammonium nitrate District-wide, and an increase of $0.10 \mu\text{g}/\text{m}^3$ for the San Jose-Jackson grid cell.

Ambient Results

We used PM_{2.5} nitrate data from San Jose-Jackson. Data were available for 2003-2008, a total of 487 observations, 408 weekday and 79 weekend. Figure F6 shows box-plots of these weekday and weekend values. Note that, in contrast to Figure F5, these are spaced throughout the year.



The figure shows that weekends generally have lower nitrate concentrations than weekdays. The mean weekday value was $2.6 \mu\text{g}/\text{m}^3$ and the weekend mean was 1.9 . This difference is statistically significant based on a two-sample t-test. The medians were 1.65 and 1.18 respectively and the difference is significant ($p\text{-value} = .01$) according to the Wilcoxon test.

Table 2 shows a comparison of ambient nitrate for December-January vs. the rest of the year. In both periods, mean weekend nitrate was lower than mean weekday nitrate. Also shown are the modeled December-January nitrate values. The modeled mean for all December-January days is $4.3 \mu\text{g}/\text{m}^3$ close, and statistically indistinguishable from, the ambient mean of $4.1 \mu\text{g}/\text{m}^3$. But whereas the modeled weekend mean is $1.1 \mu\text{g}/\text{m}^3$ greater than the weekday, the ambient weekend mean is $0.9 \mu\text{g}/\text{m}^3$ less than the weekday.

Table F-2. Mean ambient and modeled San Jose nitrate ($\mu\text{g}/\text{m}^3$)

	Ambient Feb-Nov	Ambient Dec-Jan	Modeled Dec-Jan
Weekday	2.26 (.13)	4.27 (.49)	4.1 (.63)
Weekend	1.66 (.17)	3.37 (1.28)	5.2 (1.11)
All Days	2.16	4.13	4.3

The December-January sample sizes are relatively small however, especially for weekends, where there are only 13 ambient measurements and 10 modeled measurements. Thus, although there is an apparent wide difference between modeled and

ambient, much of that could be due to random variation. The difference is statistically significant, but it may be considerably smaller than it first appears.²⁹

Table 3 compares San Jose weekend-weekday nitrate values with its NOx values measured on the same days. Weekend NOx concentrations were 59% of weekday, whereas weekend nitrate concentrations were 74% of weekday. The smaller drop in nitrate could be due to several factors, including the presence of background nitrate or transported nitrate, or non-linear chemistry. We note that ROG and ammonia both likely fall on weekends, but the effect of this on nitrate should, if anything, make the nitrate reduction greater.

Table F-3. Weekend/Weekday San Jose nitrate and NOx for same days

	nitrate($\mu\text{g}/\text{m}^3$)	NOx ($\mu\text{g}/\text{m}^3$)
weekday	2.61	54.4
weekend	1.94	31.9
we/wd ratio	0.74	0.59

We can estimate the effect of a unit mass drop in NOx concentrations on ambient nitrate by comparing the reductions in concentrations from weekday to weekend:

$$(2.61 - 1.94) / (54.4 - 31.9) = 0.029.$$

Thus, a drop of 1 $\mu\text{g}/\text{m}^3$ NOx corresponds to a drop of 0.029 $\mu\text{g}/\text{m}^3$ nitrate. This comports well with the results comparing annual trends in nitrate and NOx.

Discussion

The above analysis is correlational; it cannot prove causation. Nevertheless, the results are consistent with the hypothesis that NOx reductions have caused modest nitrate reductions in the Bay Area.

Figure F1 shows that the ratios of NO₃ to NOx are lower in most of the Bay Area than in most other populous areas of California so that CARB's conversion factor for the San Joaquin Valley and the South Coast serves as an upper bound. Figure F1 and the trends at most sites in Figure F2 show that the nitrate concentration is on the order of 0.04 to 0.05 times the NOx concentration, but Figures F3b and F4 suggest that accounting for carryover of ammonium nitrate, the relationship may be more like 0.02 to 0.025. The weekend-weekday factor was 0.029, very similar to the factor derived from daily values after adjusting for carryover.

²⁹ A t-test analogue comparing the ambient weekday-weekend difference to the modeled weekend-weekday difference yields a statistically insignificant result. However, the data are heavily skewed. If the same test is applied to the natural logs of the data, the result has a p-value of 0.04, borderline significant. Thus, the difference between modeled and ambient results is likely to be real but could be considerably smaller than first appears.

Including the (PM_{2.5} - ammonium nitrate) term in the regression probably overcompensates for the effect of atmospheric inversions. PM_{2.5} and NO_x are highly correlated, and the correlation is more than atmospheric: they share many of the same sources, especially diesel, but other fossil fuels also, and wood smoke. Thus, including this variable may attenuate the estimate of the real effect of NO_x on ammonium nitrate. This suggests that 0 (the x₁ slope of the regression with x₁, x₂ and x₃ all in the regression) serves as a lower bound for the effect of NO_x and NO₃.

Of course, this is a result at a single site, San Jose, but one that appears typical of Bay Area urban areas. The increase in nitrate that the PM model predicts for decreased NO_x emissions is greater for this site than for most other Bay Area sites. Thus, the 0.025 factor may, if anything, be conservative.

One caveat is that, although the weekend-weekday analysis strongly suggests that lower weekend NO_x *caused* lower weekend nitrate levels, the relationship is based on the balance between NO_x, ROG and ammonia that existed between 2003 and 2008. In the future, the balance might change and that change might change the effect of NO_x reductions on nitrate concentrations. However, analysis of the most recent SASS data from Livermore, Oakland and Vallejo also shows a drop in weekend average nitrate concentrations of a similar size to that found for San Jose, although the reduction is not statistically significant.

Conclusion

The ambient data suggest a modest but positive relationship between NO_x and ammonium nitrate in the Bay Area. For the multi-pollutant method, we choose 0.025 as the factor estimating the concentration (µg/m³) of nitrate from a given concentration (µg/m³) of NO_x. For converting from NO_x to ammonium nitrate (on a mass basis), the factor is 0.025 x (80/62) = 0.032.

NO_x emissions to ammonium nitrate concentrations

Another step is required for the multi-pollutant method, namely an estimate of the concentration of NO_x produced by a given amount of NO_x emissions. The PM and ozone models could be helpful in this regard.

For now, we rely on a rough relation between the mean annual NO_x among Bay Area sites, 33 µg/m³ in 2005, compared with the annual tons/day from the 2005 annual emissions inventory, 521 tons/day. This suggests that one ton/day of NO_x produces 33 / 521 µg/m³ = .063 µg/m³ NO_x concentration.

Thus, the formula to predict ammonium nitrate reductions for a given reduction in NO_x emissions of z tons/day will be:

$$\Delta \text{ammonium nitrate} = (80/62) * .025 * 33 * (z/n)$$

where n = annual 2005 District emissions of NO_x (tons/day).

References

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Kleeman M, Q Ying, and A Kaduwela 2004. "Control strategies for the reduction of airborne particulate nitrate in California's San Joaquin Valley," *Atmospheric Environment*, 39 (2005) 5325-5341.

Appendix G. 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 $\mu_x = E(X)$, $\mu_y = E(Y)$, $\sigma_x^2 = \text{variance of } X$, $\sigma_y^2 = \text{variance of } Y$, $\sigma_{xy} = E(X - \mu_x)(Y - \mu_y) = \text{covariance of } X \text{ and } Y$, and $\rho = \sigma_{xy}/(\sigma_x\sigma_y) = \text{correlation of } X \text{ and } Y$.

Suppose we measure $X^* = X + \delta$, where δ is a random variable independent of X and Y with mean 0 and variance σ_δ^2 . Then the mean of X^* is μ_x , the same as X . The covariance of X^* and $Y = E(X^* - \mu_x)(Y - \mu_y) = E(X - \mu_x + \delta)(Y - \mu_y) = E(X - \mu_x)(Y - \mu_y) + E[\delta(Y - \mu_y)] = \sigma_{xy} + 0 = \text{covariance of } X \text{ and } Y$. So the covariance of X^* and Y is the same as the covariance of X and Y . But the variance of X^* is $E(X - \mu_x + \delta)^2 = E(X - \mu_x)^2 + E[\delta(X - \mu_x)] + E(\delta^2) = \sigma_x^2 + \sigma_\delta^2$. 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 $\sigma_\delta^2 > 0$. 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_x^2, σ_y^2) , and covariance σ_{xy} . For simplicity, assume $\sigma_{xy} > 0$. The regression analogue is the expected value of Y given $X=x$:

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

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

$$\beta = \sigma_{xy}/\sigma_x^2 \tag{1}$$

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, pg 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_δ^2 , in other words, we measure X with error, and the measurement is unbiased. Then $\mu_{x^*} = E(X^*) = E(X) + E(\delta) = \mu_x$, $\sigma_{x^*}^2 = \text{Var}(X^*) = \text{Var}(X) + \text{Var}(\delta) = \sigma_x^2 + \sigma_\delta^2$, and

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

$$\text{So, } E(Y | X^* = x) = \mu_y + (\sigma_{x^*y} / \sigma_{x^*}^2)(x - \mu_{x^*}) = \mu_y + [\sigma_{xy} / (\sigma_x^2 + \sigma_\delta^2)](x - \mu_x).$$

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

Simple linear regression

In the simple linear regression model, we have

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

$i = 1, 2, \dots, n$, where we assume the x_i are fixed constants and the ε_i are independent with mean 0 and variance σ_ε^2 . 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_δ^2 , 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

$$\begin{aligned} E(\hat{\beta}^* | \delta_i) &= E \sum_i (x_i^* - \bar{x}^*)y_i / s_{x^*}^2 = \sum_i (x_i^* - \bar{x}^*)E(y_i) / s_{x^*}^2 = \sum_i (x_i^* - \bar{x}^*)(\alpha + \beta x_i) / s_{x^*}^2 \\ &= \beta \sum_i (x_i^* - \bar{x}^*)x_i / s_{x^*}^2 \end{aligned}$$

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

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

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

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

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 $\sigma_\delta^2 > 0$ for the right-hand side.

The inequality doesn't always hold. For example, suppose $\delta_i = -x_i/2$. Then the lhs 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 lhs represents the negative of the sample correlation, $-r$, between the x_i and the δ_i , and the rhs 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 the x_i and the 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 based on 1,000 simulated sets of x_i and δ_i .

Error Fraction, f	Sample Size	
	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. So the above results certainly don't 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 H. 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; and third, unlike other effects such as hospital admissions, the impact 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.³⁰ 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.

The figure 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 $0.63 * 100 + 9.376 = 72.376$.

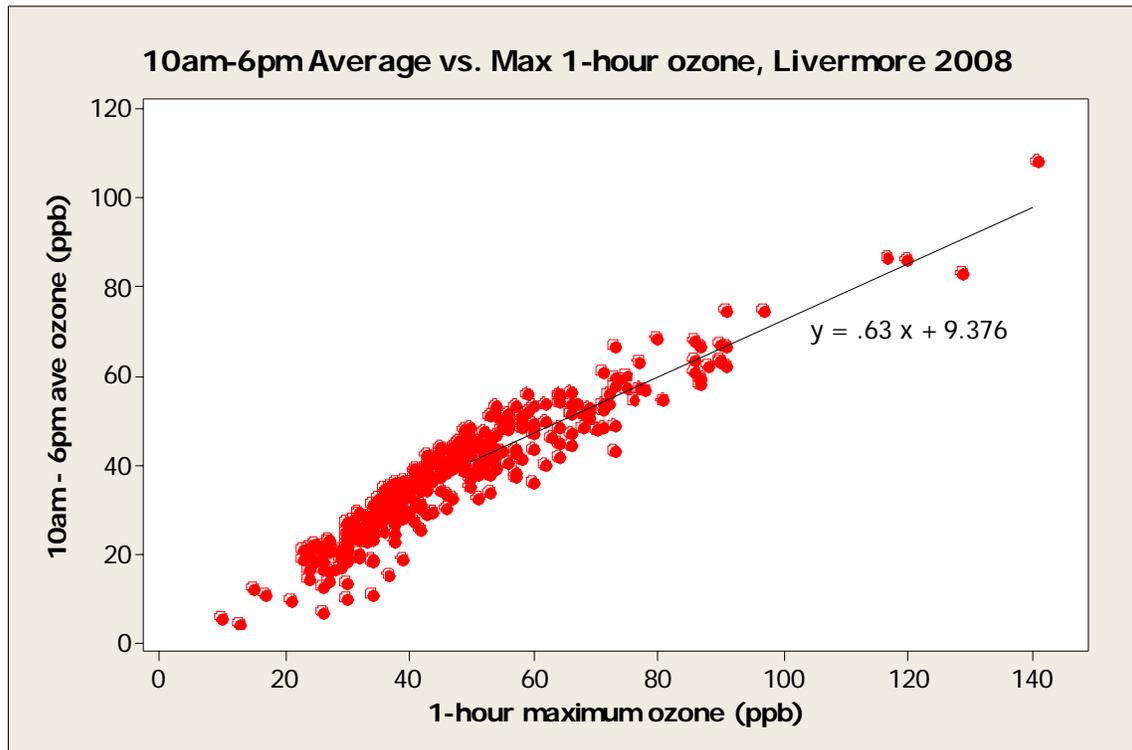


Figure F7.

³⁰ They found a 63% increase in illness-related absences, but our data on school absences is limited to the total.

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 don't 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 didn't obtain figures for the Bay Area, so we use the Hall estimates.

To account for intersection between 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:

$$y_0 \times 0.0047 \times 0.55 \times [\exp(0.0198z) - 1] \times (0.4 \times 365)$$

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

References

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