Multi-Pollutant Evaluation Method
Technical Document

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Executive Summary

The Bay Area Air Quality Management District (District) is preparing the Bay Area 2009 Clean Air Plan (CAP) to update its current ozone plan (the 2005 Ozone Strategy), as required by the California Health & Safety Code. The District has elected to broaden the scope of this effort by developing an integrated, multi-pollutant air quality plan. 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 has developed to help analyze and compare potential emission control measures on a multi-pollutant basis for the 2009 CAP. Air pollution imposes a range of negative health impacts and economic costs on the Bay Area. This tool weights each pollutant based on its relative harm. For purposes of the MPEM, harm is defined in terms of the impacts that each pollutant imposes on human health and/or the earth's climate (i.e., anticipated impacts from global warming). We plan to use the MPEM to analyze how potential control measures would reduce these negative impacts and to estimate the associated cost savings of the avoided negative impacts.

Our objective is to develop a methodology 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.

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)

We propose to build upon established precedents embedded in these studies, but go beyond them by:

- Employing our method to help evaluate individual control measures for the CAP
- Using computer models to estimate how ambient concentrations of each pollutant are affected by changes in emissions of the pollutants or its precursors
- 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, we included ozone and PM, the two criteria pollutants for which the Bay Area does not yet attain all standards. The Bay Area does attain all standards for the other criteria pollutants (CO, SO2, NO2, and lead). It should be noted, however, that there may still be health effects at ambient concentrations even below the current standards for all the criteria pollutants.

Toxic air contaminants are a separate category of pollutants. Although there are several hundred 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 are the major culprits in global warming.

We 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 our methodology, 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 of a ton of greenhouse gas reduced (CO2-e) does not fully capture all potential costs related to climate change and global warming.

In developing the methodology, we 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 NOx, then the NOx emission reductions

would be geographically distribute across Bay Area grid cells in the same proportion as the overall NOx inventory is distributed across those grid cells. (Section 1.5)

- For purposes of estimating population exposure (Step 3 below), 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 straighforward. The methodology involves several stages of calculations for each proposed control measure[1]. The steps are:

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

Step 2. Concentrations: We estimate how a change in emissions of each pollutant would affect its ambient concentrations and other pollutants related to it, 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. (Section 4)

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

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 amount of reduction (or increase) in each pollutant.

[1] For ozone, PM2.5, 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.

Applications

The primary purpose of the multi-pollutant evaluation methodology is to estimate the aggregate value of the health and climate protection benefits of each potential control measure. The methodology should be especially valuable in helping us to evaluate potential trade-offs; i.e. a situation where a control measure may reduce one pollutant, but increase a different pollutant. In addition, the MPEM (or portions thereof) can potentially be applied for related purposes, such as:

- Estimating the total cost of health impacts and monetary costs associated with current emission levels and ambient concentrations
- Backcasting to estimate the health impacts and monetary costs associated with emission levels and ambient concentrations in years past
- Estimating the aggregate benefit of the overall emission reductions for the proposed 2009 CAP control strategy as a whole
- Evaluating the benefits of GHG reduction measures in reducing criteria pollutants

Probability Analysis

We consider the range of the uncertainty by means of a probability analysis which is described in Appendix XX. The probability analysis estimates the degree of uncertainty in the assumptions and computations related to each step in the method, and then calculates overall probability distribution for the results of the methodology as applied to each control measure. The probability analysis will help us compare the potential benefits of various control measures.

Conclusion

The multi-pollutant evaluation methodology summarized above, and described in detail in the body of this document, represents our best effort to analyze control measures on a multi-pollutant basis, *using the tools and technical data available at this time*. The results of the analysis are intended to inform, but not prescribe, policy by providing data for comparing and evaluating proposed control measures.

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 (see Section 6). Although the MPEM described in this document is far from perfect, we believe that it does provide a viable and valuable tool for use in crafting the 2009 CAP, as well as a solid foundation upon which we hope to build for future air quality planning efforts.

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Multi-Pollutant Evaluation Method

1. Introduction

The Bay Area Air Quality Management District (District) is developing a 2009 Clean Air Plan (CAP) that will provide 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. Our objective is to develop a methodology that will enable us 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 NOx
- $PM_{2.5}$ both - primary $PM_{2.5}$ as well as precursors of secondary $PM_{2.5}$ (NOx, SO_2, NH_3)
- Air toxics
- Greenhouse gases (GHGs)[2]

The goal of the MPEM is to assess a measure's impact on the air pollutants listed above and 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 and to compare a measure's benefits to its costs.

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, PM2.5, and air toxics, we employed steps 1 through 5 below; for green house 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.

[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.

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 times the estimated social cost per ton of GHGs expressed in terms of CO_2-equivalent.

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

1.1 Probability Analysis

Even though we choose our 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 Appendix XX.

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 that are used in the methodology include the following:

- Estimated emission reductions for each control measure[3]
- Data on ambient concentrations of pollutants derived from the District's ambient air quality monitoring network

[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.

- 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. 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

In this Section, we discuss the choices that we made.

1.3.1 Air Pollutants Included in the Methodology

There are literally 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 pollutants[4] 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 Area[5]. Two other pollutants, carbon tetrachloride and hexavalent chromium, which make up much of the remaining risk from air toxics, were not considered for various reasons[6].

[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 PM2.5), 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 is little or 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.

The pollutants chosen are not all emitted directly. Thus, we need to consider two lists – the list of 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 (ozone in the case of VOCs). Ozone is almost entirely a secondary compound, formed largely from reactions of NOx and VOCs. Thus, we will consider emissions of VOCs and NOx.

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

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

For this study, we will consider the emissions of NOx, 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 clearly part of overall $PM_{2.5}$. But it is 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. However, we add the endpoint of lung cancer cases not resulting in death so as to include the costs of both fatal and non-fatal lung cancer.

For greenhouse gases (GHGs), we include the Kyoto 6: CO_2, methane, nitrous oxide, sulfur hexafluoride, hydrofluorocarbons, and perfluorocarbons. Of these, the first three constitute 99% of the known GHG potential of the Bay Area (BAAQMD 2006).

The ground level ozone may be a GHG. Black carbon $PM_{2.5}$, or soot, also contributes to global warming. However, the global warming impacts of ground level ozone and soot 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.

[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.

Table 1 summarizes which pollutant emissions will be tracked and which pollutants will be evaluated for their health/social impact.

Table 1 – Pollutants Included in Multi-Pollutant Evaluation Methodology

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

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. In addition, there are risks that undoubtedly exist but have not been quantified. An example is the carciniginicity 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.

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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.

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.

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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 diesel[8], are measured at various District monitoring stations around the Bay Area. These observations can be 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.

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 $\mu g/m^3$, 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 specie is explained in detail in Appendix E.

Ozone: A number 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 NOx may actually cause increases in ozone in some areas during some episodes. (Appendix B shows an example.) Conversely, due to the NOx quenching or ozone titration, high NOx 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.

[8] Diesel PM cannot be directly measured.

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 toxic 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 (EPA 2006) eliciting opinions about the effect of $PM_{2.5}$ on mortality found that the experts were unanimous in rejecting the idea of a population threshold, although some thought it may exist at the individual level. In their evaluation of the effects of $PM_{2.5}$ on mortality, which used the EPA study as a basis, CARB staff recommended "that the cut-off level be expressed as a range of values from 2.5 to 7 $\mu g/m^3$." 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) investigated the literature and 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, but take the possibility of a threshold as low as 40 ppb into account in the uncertainty analysis.

1.3.5 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 $1/500^{th}$ reduction in NOx 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 model runs.

2. Estimating Concentrations from Emissions

This section explains the calculations of concentrations from emissions. Although the approaches are generally similar, the estimation methods for toxics, ozone and $PM_{2.5}$ are discussed separately.

The BAAQMD 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 grids 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 2000 base year inventory for ozone and PM and 2005 base year emissions for toxics.

The models were run both with a base case and with 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, 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, de_r (considered as a percentage of total annual District emissions of that pollutant). So for the concentrations of primary pollutants, the following relation exists:

$$\Delta c_{ri} = c_{0i} \, (dc_i/de) \, de_r \qquad (1)$$

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 between 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 *marginal* reduction when considered with other

precursors. This is analogous to the difference between a simple derivative and a partial derivative.

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 marginal reduction, as follows:

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

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

provides factors to convert from the individual effect to the marginal 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

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

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. de_{1r}, de_{2r} are the percent reductions in precursor 1, precursor 2 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

[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 $PM_{2.5}$ was run for a December-January period; the toxics model for a week in July and a week in December; the ozone model run for two summer episodes of about a week each.

concentration and the annual concentration are of interest. Thus, the target of the extrapolation of the concentrations may vary for the different pollutant categories.

Estimating initial concentrations – use of ambient concentrations: The models are used for estimating the *change* in concentrations, but the pollutant measurements made at BAAQMD monitoring sites may provide more accurate estimates of the initial concentrations. Also, ambient concentrations can provide information for extrapolating from the simulated periods to the rest of the year.

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 will be considered. But for $PM_{2.5}$, we consider 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.

Individual vs. Partial Effects: There are potential differences between how the concentration of a species is affected by the change in a precursor when considered in isolation vs. when considered with other precursors, analogous to the difference between a simple derivative and a partial derivative. Since we are concerned with all precursors, we attempt to estimate the partial effects. That is, we attempt to estimate the effect of changing a given precursor *with the others held constant*, rather than its effect considered in isolation. If the effect on concentration of various precursors is linear, then using these partial coefficients will correctly estimate the joint effect on concentration of a given set of precursor reductions.

The ozone and $PM_{2.5}$ models were run with reductions in individual precursors, but also with reductions of all precursors jointly. This allowed for a comparison: The joint reduction in concentrations could be regressed against the reductions from individual precursors. The fits were generally excellent, suggesting that most of the change in ozone and $PM_{2.5}$ concentrations could be explained from individual precursor reductions without interactions.

An individual precursor slope of 1 indicated that the effect of the precursor was the same whether considered individually or jointly (partial derivative = simple derivative). If the slope were < 1 then the precursor had a smaller effect when considered jointly.

The regression coefficients were applied as factors in the formulas used to estimate changes in ozone and $PM_{2.5}$ concentrations from changes in precursor emissions.

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.

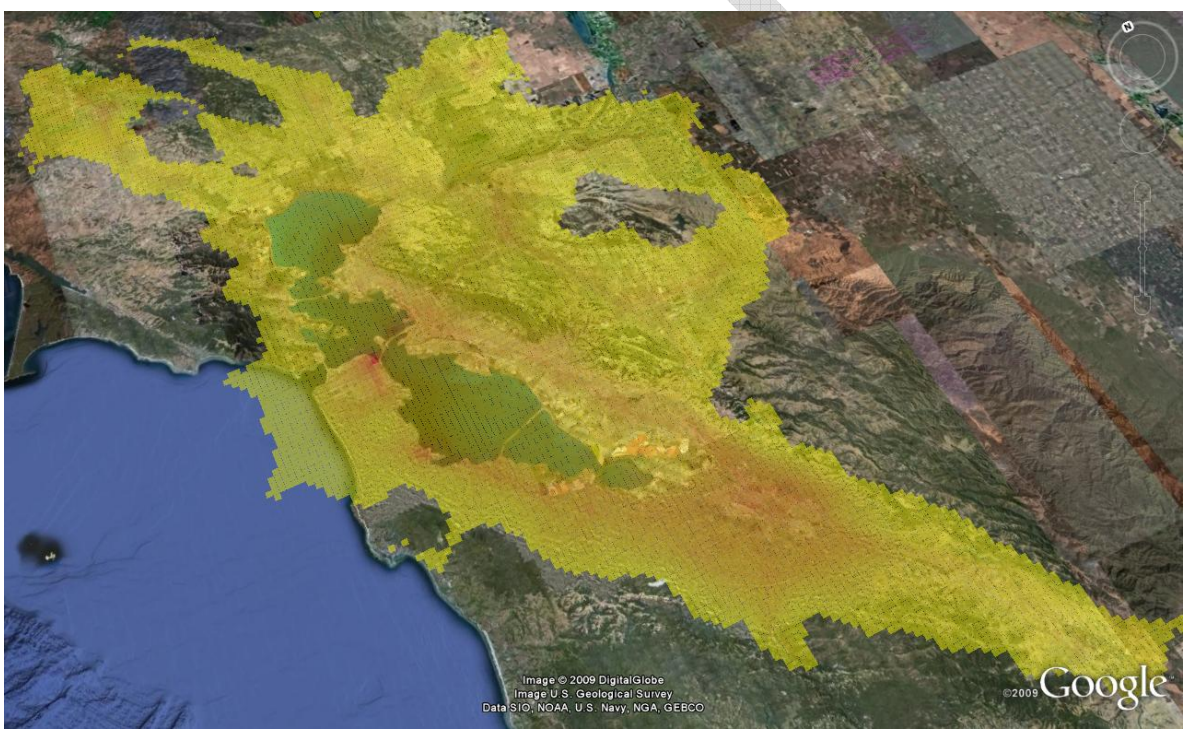

Figure 1. Grid of model estimated reductions in benzene concentrations for a 10% reduction in benzene emissions. Values range from 0.01 ppb (light yellow) to 0.08 ppb(red).

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.

For this analysis, it will be assumed that the model provides an unbiased estimate of the true relationship between % reductions in emissions and % reductions in concentrations. But the initial concentrations, c, are scaled relative to the average annual ambient concentrations with a ratio, $r_0 = \frac{\bar{c}_0}{\bar{c}_m}$ Specifically, for each grid square, the assumed initial concentration is

$$c_{0i} = c_{mi} * r_0$$

where c_{mi} = the initial modeled concentration in grid cell, i, $\bar{c}_0$ = the mean of the annual average ambient concentrations at Fremont, SF and SJ, and $\bar{c}_m$ is the mean of the July+December modeled concentrations for the grid squares containing the Fremont, SF and SJ sites.

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

[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.

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 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 NOx and VOCs. The goal was to estimate the change in ozone as a function, f(n,v), of reductions in NOx and VOCs, that included the possibility of non-linear interactions.

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

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

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

Details are provided in Appendix B.

2.3.3 Estimating initial ozone concentrations

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 apply the change functions derived from the model to observed daily 1-hour maximum ozone for 2004-06, 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, The function $f(r) = a + br + cr^2$ = estimated ppb-days above 50 ppb for a reduction of r% was fit for values of r = 0, 0.01, …, 0.20, where $f_i(r)$ was the summation of $z_i = (1-r)y_i - 50$ for all 1-hr max ozone values y_i in 2005-07 for which z_i 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 NOx emissions from a given control measure (as a percent of total NOx emissions) is Δe_n, and the reduction in VOC is Δe_v, and the

$\Delta f_i = [f_i(0)-f_i(r)]/(3*365)$,

where $r = a_i\Delta e_n + b_i\Delta e_v$ = estimated percent reduction in ozone concentration, and Δf_i = change in daily 1hr max ozone > 50 ppb, and 3*365 equals numbers days in 2005-07 period.

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 $\mu g/m^3$ 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, 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.

Thus, both 24-hour and annual-average $PM_{2.5}$ concentrations are of interest. 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. There is also a large secondary component composed primarily of ammonium nitrate and ammonium sulfate. Our analysis of $PM_{2.5}$ is limited to those components of $PM_{2.5}$ that we can reduce by means of control measures in the 2009 CAP: 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. Lowering of the national PM2.5 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. NOx, 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 are highly seasonal, 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.

The $PM_{2.5}$ model was run with a 20% reduction in all direct $PM_{2.5}$ emissions including those of carbonaceous particles. The model tracks concentrations of EC and directly emitted anthropogenic OC. We assumed that the modeled change in EC concentrations resulted from a change in EC emissions, and similarly for OC.

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A comparison of the model with 20% reductions in all precursors, run 6, showed almost the same changes in PM2.5 concentrations from the base case as run 5. Therefore, no adjustment was made to convert from individual to marginal 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. Thus, here we need to adjust winter modeled concentrations to annual average concentrations.

The District measured elemental carbon (EC) and organic carbon (OC) on PM_{10} 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 measurement and modeled value includes the carbon mass only, whereas the original $PM_{2.5}$ molecule 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 emissions is:

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

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 individual to joint 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. These measurements 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:

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

where c_i is the average sulfate concentration from the base-case model run.

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 NOx in ammonium nitrate concentrations. The effect of directly emitted nitrate was negligible.

Preliminary modeling showed a potential disbenefit to NOx 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, an analysis of ambient data found that reductions in NOx were correlated with annual reductions in nitrate. This included a comparison of weekend to weekday NOx and nitrate, where weekend ambient nitrate was found to be lower than

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

weekday. The modeled nitrate values are sensitive to the balance of NOx, VOC and ammonia emissions. Thus, for the effect of NOx emissions on ammonium nitrate concentrations, we use a positive conversion factor of 0.032 rather than the modeled results. See Appendix F for details.

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 individual to joint effects were 0.93 for ammonia, 1.00 for NOx 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 District site, San Jose. It is also measured at Point Reyes. The 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 the 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:

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

3. Estimating Population Exposure

This section explains how we estimate population and population exposure.

[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.

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

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 Δconcentration was then summed for each county and divided by the county population, yielding a population-weighted Δconcentration.

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

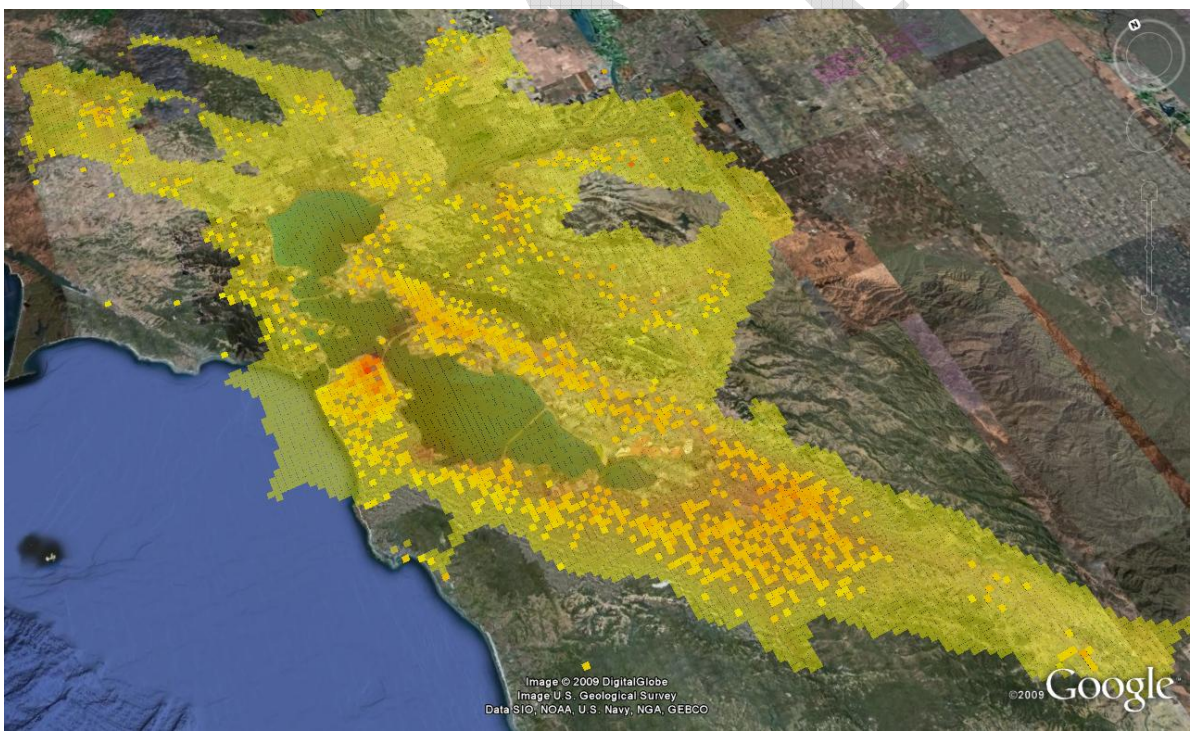

Figure 2. Reduction in benzene concentration overlaid with population. Grids with population shown with solid color.

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

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

It should be noted that if we were able to estimate true exposures (which we are not able to do at this time), the bias would be even stronger because people spend most of their time indoors. The amount of $PM_{2.5}$ that infiltrates is perhaps 70% of the ambient levels (See, e.g., Lurmann & Korc 1994), so this would impart a greater downward bias.

In layman's terms, the concentration-response function (C-R coefficient) is calculated by analyzing the relationship between known health outcomes for a given population compared 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 bias[14], 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 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.

Thus, this is a second reason why it's likely that the C-R coefficients from epidemiological studies underestimate the true C-R effect. 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 li3kely 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, but 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

[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.

assumed lung cancer risk for DPM is 300 in a million per $\mu g/m^3$ (OEHHA 2005) for an average lifetime exposure of 1 $\mu g/m^3$ (a concentration very near the annual average for the Bay Area). As discussed above, however, most people spend most of their time indoors, say 90%, so, assuming that the PM penetration rate is 70%, then their average exposure would be about 0.1(1) + 0.9(1 x 0.7) = 0.73 $\mu g/m^3$, for a true risk of 0.73 x 300 = 219 in a million.

3.4 Exposure Probability Analysis

The discussion in the previous two sections suggests that our estimates of exposure are not only uncertain, but likely to be biased, as are the C-R coefficients in relation to our estimated exposures. As explained above, these biases could lead to underestimation of health effects. In the Monte Carlo simulation we attempt to account for these biases, estimating exposure distributions with means lower than backyard exposure to account for the likely lower indoor concentrations of ambient pollution, but with C-R functions increased to approximately correct for the biases discussed in 3.2 and 3.3.

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:

$$\Delta\text{cases} = \text{baseline incidence} \times \Delta\text{risk} \qquad (4.0)$$

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 7.0 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….

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

Δcases = baseline incidence x $(e^{bc} - 1)$ formula (4.1)

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

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

Δcases = population x (baseline incidence rate) x $(e^{bc} - 1)$ formula (4.2)

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

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

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

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

4.3 C-R functions for cancer

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

Δcases = population x b/70 x c formula (4.4)

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 2007 county-by-county rates from the California Office of Statewide Planning and Development (OSHPD).

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 EPA uses 0.00365, based on western states & 1999 data.

For school absence rates, we used a recent San Francisco 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, EPA (2008). We also use the BENMAP coefficients, uncertainty estimates and C-R functions.

The one exception is for mortality, where we use the CARB estimate of 1% increase in mortality for a 1 $\mu g/m^3$ increase in $PM_{2.5}$.

For ozone, we use the effects, C-R functions, coefficients and uncertainties from Ostro *et al.* 2006.

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).

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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.

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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	CARB 2008	≥ 30	1.0	4.1	all non-accidental deaths by county of residence	California Department of Health Statistics
Chronic Bronchitis Onset	Abbey *et al.* 2005	≥ 27 (w/o bronchitis)	1.32	4.3	0.00378	Abbey *et al.*
COPD Hospital Admissions	Ito 2003 & Moolgavkar 2003	≥ 65	.116(.206) Ito 0.185 (.052) Moolgavkar	4.2	county-specific rates, 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 *et al.* 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 *et al.* 1999	< 18	1.653	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHPD**
Acute Bronchitis Episodes	Dockery *et al.* 1996	5-17	2.721	4.3	0.043 cases per child per year	American Lung Association 2002
Upper Respiratory Symptom Days	Pope *et al.* 1991	Asthmatic children 5-17	0.36	4.3	124.8	California Center for Health Statistics reported that in 2003, 14.8% of children and adolescents in California had been diagnosed with asthma
Lower Respiratory Symptom Days	Schwartz & Neas 2000	7-17	0.6	4.3	0.438	Schwartz *et al.* (1994,Table 2)

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Work Loss Days	Ostro 1987	18-64	0.46	4.2	2.17	Adams *et al.* 1999
Minor Restricted Activity Days	Ostro & Rothschild 1989	≥ 18	0.741	4.2	7.8	Ostro & Rothschild 1989
Ozone			**% per ppb 1-hr max ozone**			
Mortality	Ostro 2006	All ages	0.04	4.2	all non-accidental deaths by zip of residence	California Department of Health Statistics
Hospital Admissions for Respiratory Diseases	Thurston & Ito 1999	All ages	0.16	4.2	county-specific rates, 2007 Bay Area rate 0.0025	OSHPD**
Asthma Emergency Room Visits	Ostro 2006	< 18	0.24	4.2	county-specific rates, 2007 Bay Area rate 0.0056	OSHPD**
School Loss Days	Gilliland *et al.* 2001	5-17	2.12	4.2	SFUSD rates	SFUSD 2009
Minor Restricted Activity Days	Ostro & Rothschild 1989	≥ 18	0.22	4.2	7.8	Ostro & Rothschild 1989
Toxics			**lifetime risk / $\mu g/m^3$**			
Lung Cancer (DPM)	OEHHA 2005	all ages	.0003	4.4	NA	NA
Leukemia (1,3-butadiene)	OEHHA 2005	all ages	.00017	4.4	NA	NA
Leukemia (benzene)	OEHHA 2005	all ages	.000029	4.4	NA	NA
Cancer – various sites (acetaldehyde)	OEHHA 2005	all ages	.0000027	4.4	NA	NA
Cancer – various sites (formaldehyde)	OEHHA 2005	all ages	.000006	4.4	NA	NA

* See formulas in text above.
** OSHPD = California Office of Statewide Health Planning and Development.

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4.7 Calculation of change in incidence

Estimates of the incidence of various health effects is made for each grid square, then summed to get county and Bay Area totals. For example, consider acute bronchitis attacks. 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, 2.12, the incidence rate, 0.043, and the estimated 5-17 year-old population, p_i, to produce

$$p_i * 0.043 * (e^{2.12*\Delta ci} - 1)$$

that is, the estimated reduction in the annual number of acute bronchitis episodes among 5-17 year-olds with residences within grid square i. These values are then summed by county.[15]

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

Mortality. For mortality, we used county-level incidence rates. But rather than basing the C-R function on an average county concentration, we computed the C-R function for each grid square and, for each county, computed the population-weighted average of these functions.

Effects with county-level incidence rates. The hospital admissions and emergency room visit incidence rates were by county, I_c, say. Here the C-R function was computed for each grid square, and the population-weighted total was summed for each county, c, then multiplied by I_c to get the estimated incidence.

School absences. Schools are only open on weekdays, and they close during the summer months, i.e., the months where the Bay Area has its highest ozone.

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

[15] 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.

• Adverse effects on others (family, friends, caregivers, etc.) resulting from their own adverse health effects (Hall 2008).

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

5.1 Concepts and Measures of Value

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

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

5.1.1 Cost of Illness

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

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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: 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.

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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 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). 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). 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 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 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 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	EPA 2005 reports estimates of WTP based on preventing respiratory symptoms caused by acute bronchitis. 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 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 EPA 2005 for distribution derivation.

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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 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 *Occupational Employment Statistics Survey(1st 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 EPA 2005, the value of a school absence is the estimated daily lost wages for women over age 25. Using BLS data (Women in the Labor Force: A Databook 2008) we find that the weekly median age for women over 25 in 2007 was $614, thus the estimated daily median wage $123. The labor force participation rate for women over 25 with children under the age of 18 was .713. Thus, .713*$123=$88 (in 2007$) or $91 in 2008$ (Where $91 is the lost productivity at the female's parent's wage. This is with the assumption that if a child stays home from school, a working mother will have to stay home from work to take care of the child.)
Minor Restricted Activity Days	$61	WTP	Applying similar methods to 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 EPA 2005 and EPA 2008, we arrive at a triangular distribution by applying the following: by definition, MRAD odes 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 where estimated from a literature review completed by McCubbin et al (1996). The lower and upper bound include all costs of cancer, including medical costs, pain and suffering to both patients and friends, and the lost of production to society.

*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)

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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 2009 Clean Air Plan (CAP). (See the CAP for discussion re: greenhouse gases and climate change.) Therefore, in addition to the value of health benefits from reducing ozone, PM, and air toxics, we include an estimated value of the social benefit of GHG reductions in the MPEM. In order to do so, we need to assign a value to the benefit of reducing a ton of GHG (CO_2-e). This section describes key issues related to valuing GHG reductions, and explains how we went about selecting a reasonable GHG value for the MPEM.

After reviewing the literature of studies that have been performed to estimate the cost or value of GHG emissions, we conclude that $28 per ton of CO_2-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.[16] These gases vary significantly in terms of the volume (mass) of emissions as well as their specific global warming potential (GWP) expressed on a CO_2-equivalent (CO_2-e) basis. It should be noted that, to the extent that 2009 CAP control measures may reduce emissions of other (non-Kyoto Six) GHGs, or other pollutants such as black carbon[17] that are not included in our calculations, the MPEM may underestimate the benefit of control measures in protecting our climate.

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

[17] 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 change, 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 Appendix XX) 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,[18] 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.[19] 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.

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 emission. 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

[18] 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 (44/12). A value of $10 per ton of carbon equates to $28 per ton for CO2-e.
[19] See http://www.ecx.eu/

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 pay the price for 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 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 distribution[20] 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).

[20] 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 frame. 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 by season.

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

Population exposure: For the 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. At a minimum, a representative week from each season should 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

The simplest assumption about how emissions reductions affect concentrations is a proportional rollback. Naively, one would expect that if emissions of a pollutant were reduced 10%, say, then its concentrations would also be reduced by 10%.

Pollutants do not always behave so simply, however, 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.

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, 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 simple rollback.

[*] 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.

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

Ozone Trends at Los Gatos

Figure 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

* 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, previously, ozone had been recorded only to the nearest 10 ppb, which would have made comparing percentiles, which differ by only parts per billion, problematic.

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*.

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[**] 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 1. Trend in San Jose Benzene Distribution 2003-07 vs 1987-91

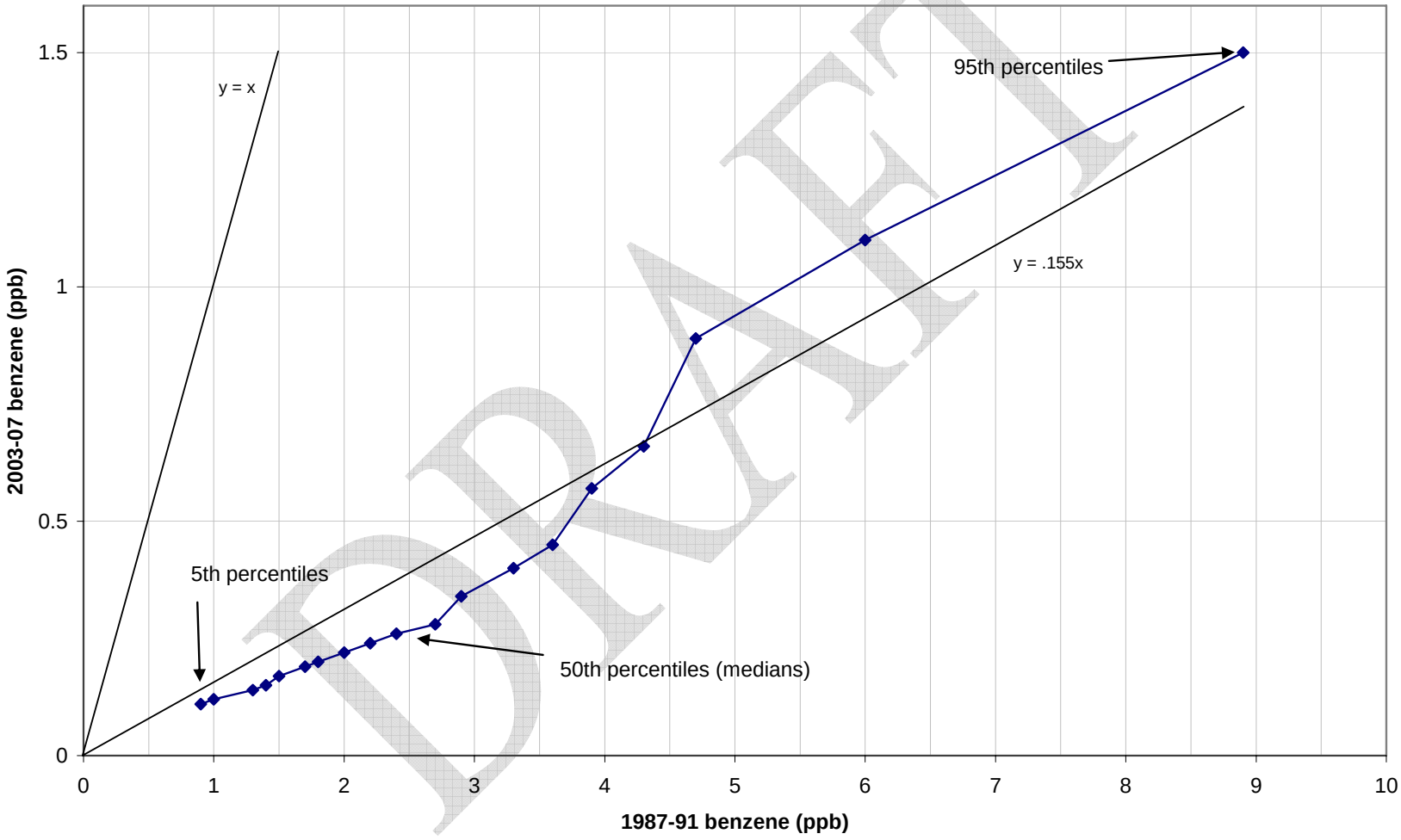

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Figure 2. Trend in San Jose 1,3-Butadiene Distribution 2003-07 vs. 1990-94

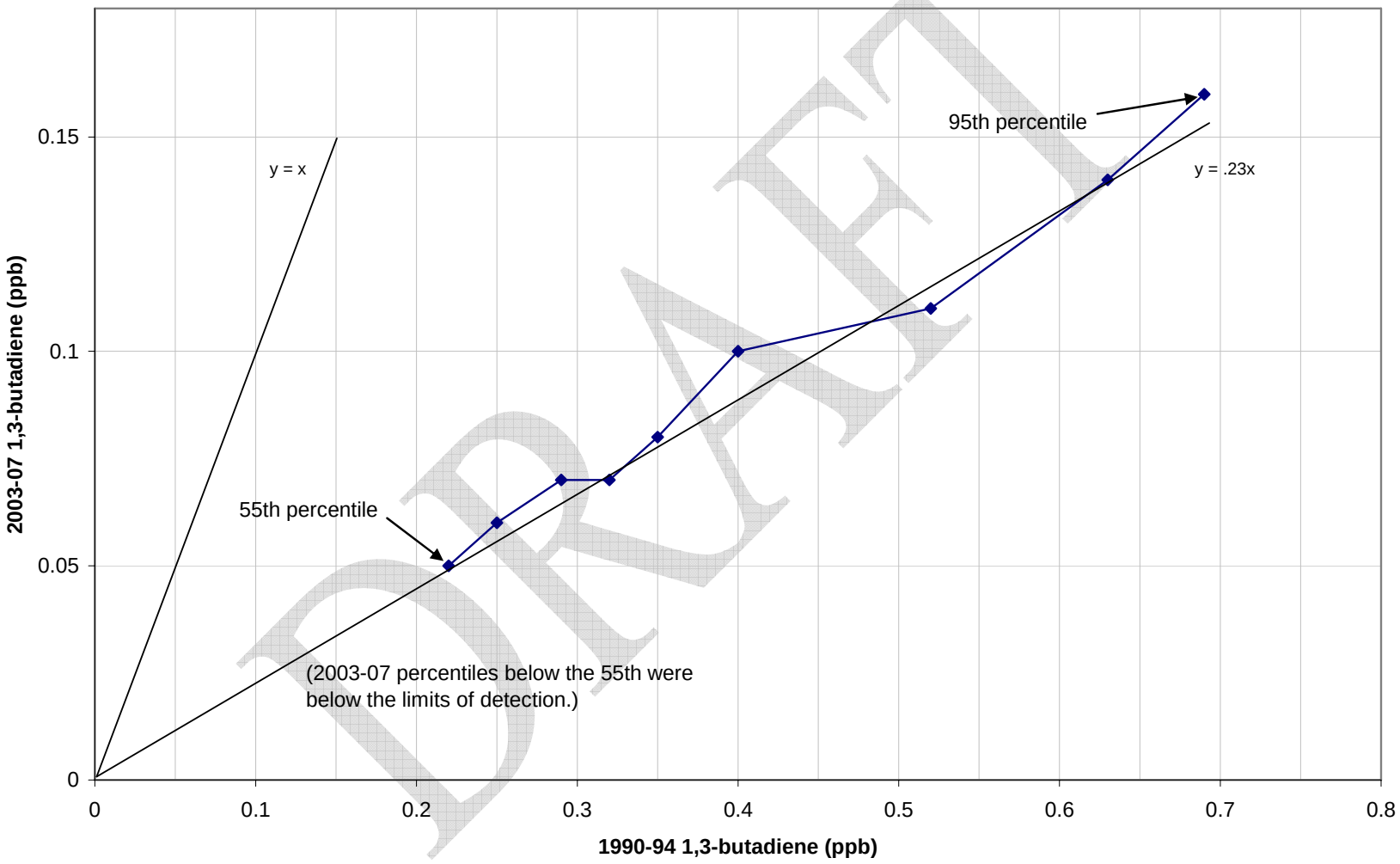

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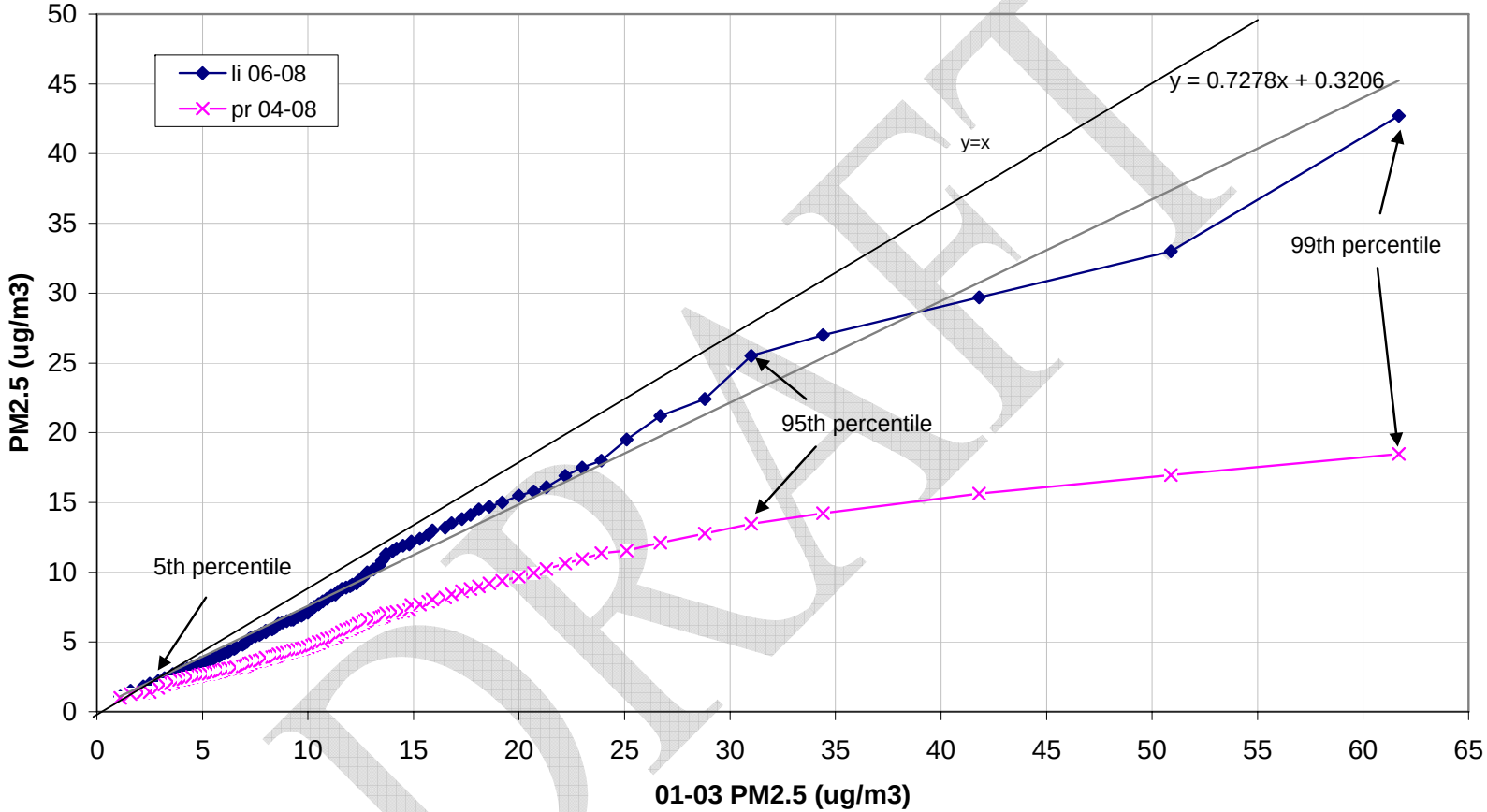
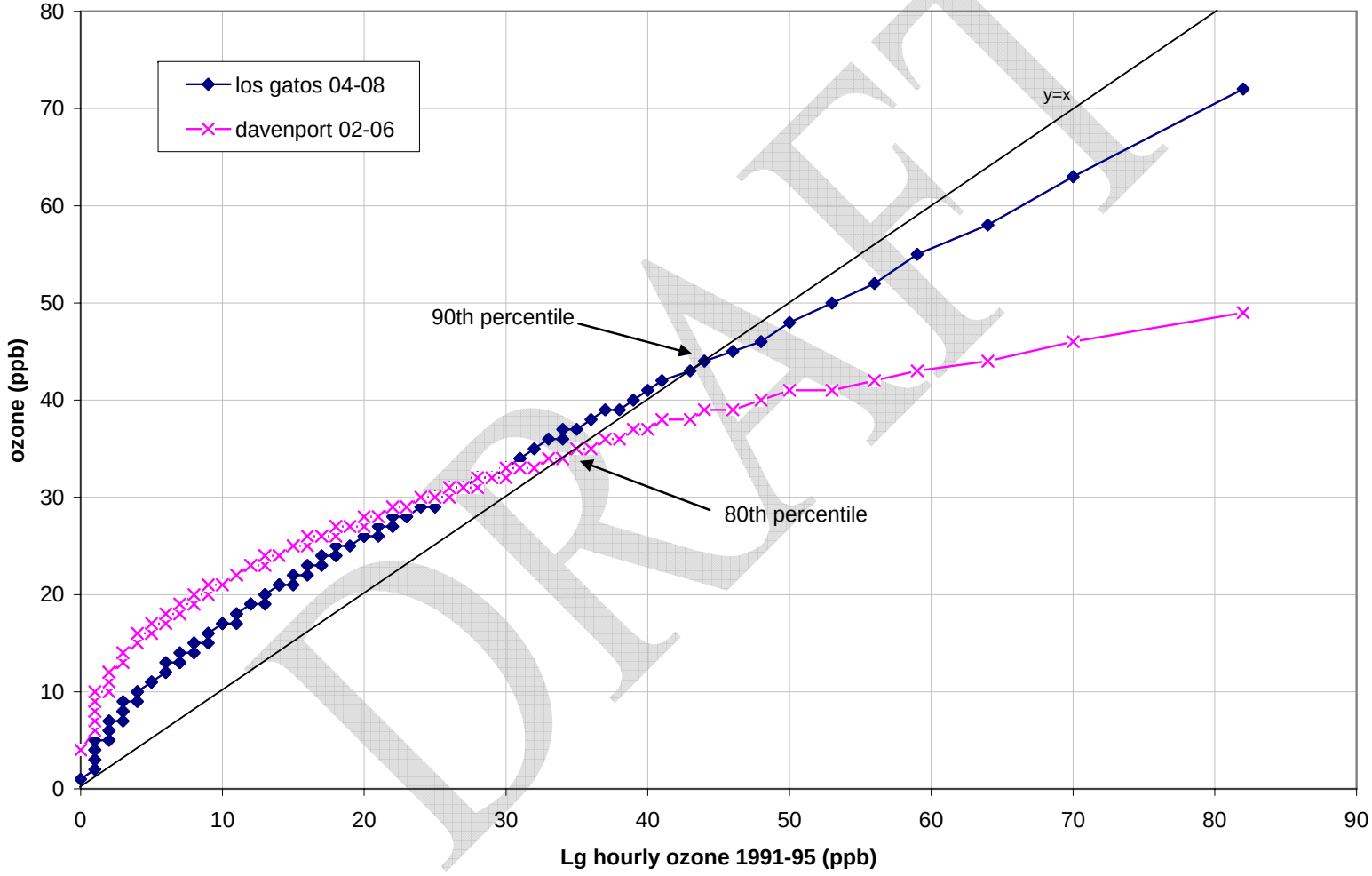
Figure 3. Trend in Livermore PM2.5 2006-08 vs 2001-03, and vs. Point Reyes "background"



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



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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 2009 Clean Air Plan, the District's plan to address multiple pollutants. 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 complicated, however. Reaction rates depend not just on the amounts of these precursors, but also their ratio; i.e., 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 seems valuable to take advantage of the District's photochemical model capabilities. The photochemical model provides information on how reductions in ozone precursors would affect ozone in different locations in and near the Bay Area. The picture it provides is complex and raises the question of whether the effects on ozone of reductions in NOx should be treated differently from the effects of reductions in VOC.

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 CAMX 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 2x2 km grid 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.

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Results

Figure 1 shows histograms of the distribution of the percent change in ozone resulting from different NOx 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 NOx 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 NOx had been reduced 10%, that is, a change from (0 VOC, -10% NOx) to (-10% VOC, -10% NOx); the two panels in the fourth row show the change if both NOx 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 NOx-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 NOx, leads to very similar reductions in ozone as the reduction of VOC with no reduction in NOx. (Row 3 vs. Row 2)

- The reduction of both VOC and NOx leads to changes in ozone that are close to additive. If these reductions (y) are regressed on the NOx only (n) and VOC only reductions (v), the resulting regression equation is: $y = .94n + .89v - .07$, with an R^2 of 99%.

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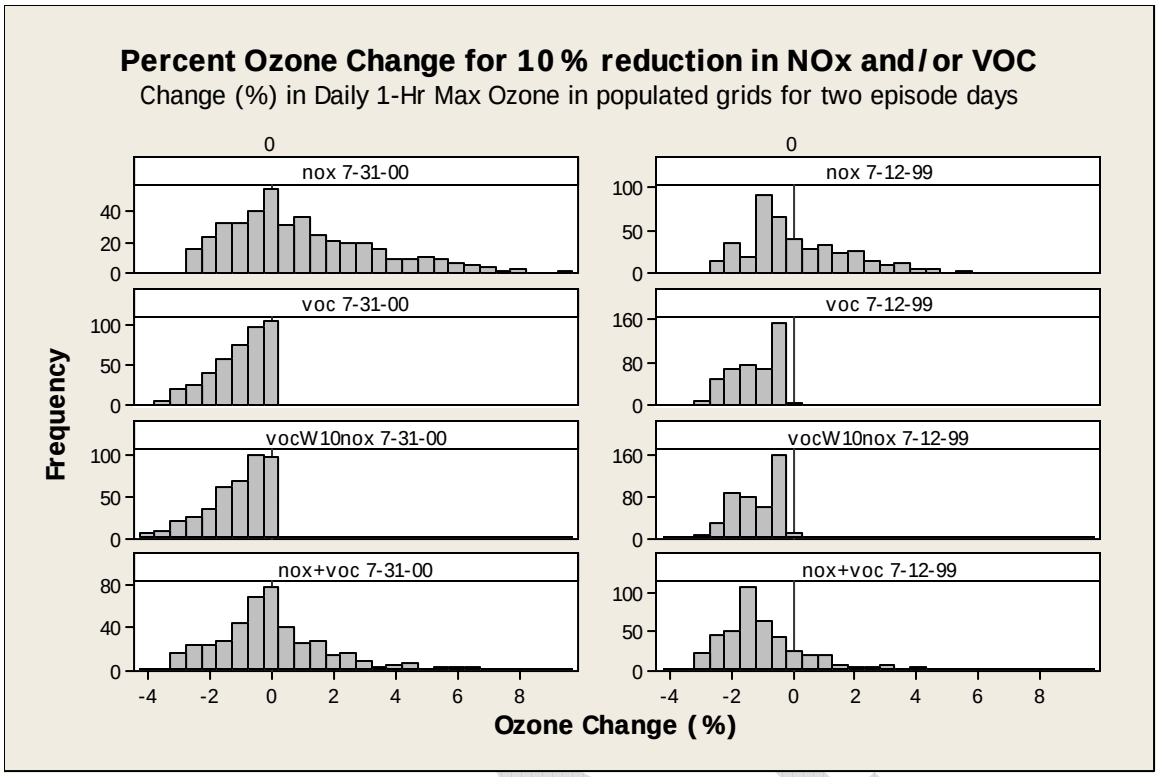

Figure 1. Percent change in 1-hour daily max ozone for various NOx and VOC 10% reductions: NOx only, VOC only, VOC reduction with 10% lower NOx, and reductions in both NOx and VOC.

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Relation of the effect of 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 4 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%).

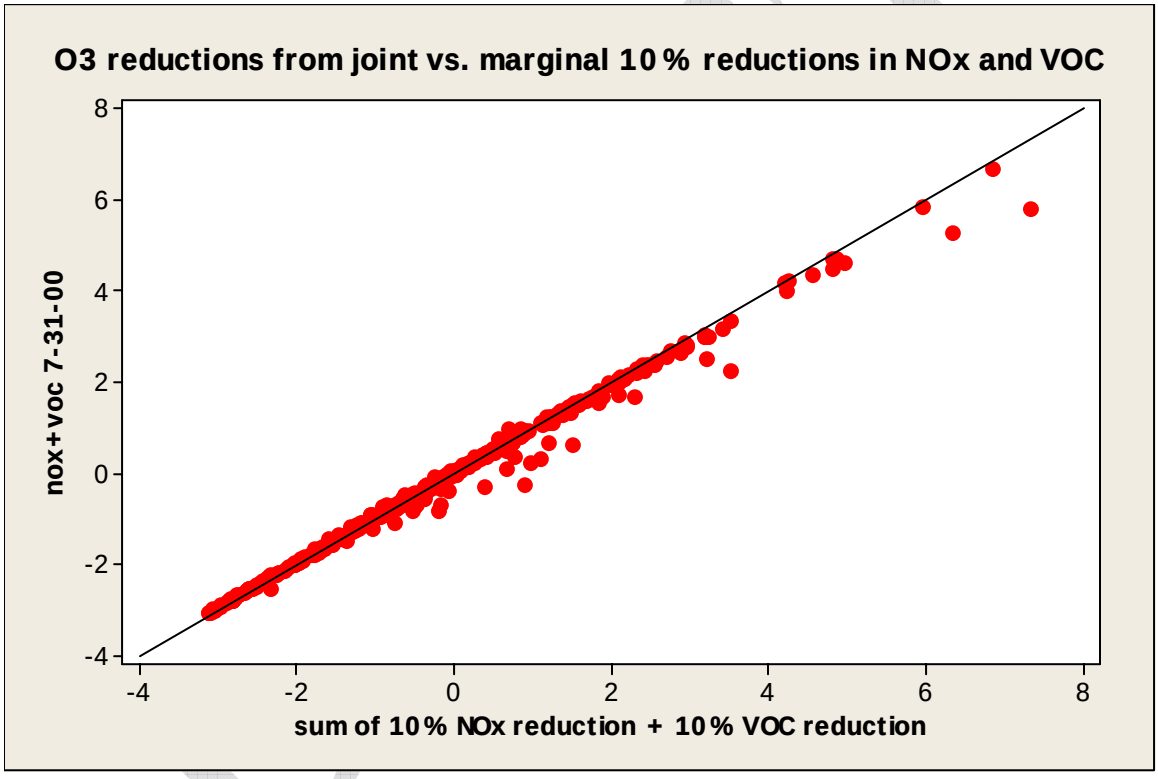

Figure 4.

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 eliminated 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 R^2 value was 0.61, and 90% of the R^2 values were at least 0.30.[21]

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.

[21] Technically, the values computed were not R^2, because the regressions were fit without an intercept. If intercepts were included, the R^2 values would be somewhat larger.

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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 evaluation of the impact of changes in $PM_{2.5}$ would be evaluated by summing over the changes in daily health effects.

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

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

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

As an example, consider the acute MI. Using Peters (2001), the C-R function is proportional to $e^{.02412x} - 1$. Using $PM_{2.5}$ measured at San Jose, the values for 2007 for the lh and rh sides of equation C1 were .3323 and .3023, respectively. For 2008, the values in equation C1 were .3825 and .3572 respectively. These represent underestimates of about 8% in 2007 and 6% in 2008.

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

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

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

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

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

Substituting D1 and taking expectations:

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

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 particle 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 NOx, 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 (PM2.5).

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 mostly formed from the combustion of carbonaceous compounds, and also from char-broiling meat.

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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 cm3/mol/sec	2
MEOH	Methanol	2
MGLY	Methyl glyoxal	2
NO	nitric oxide	1
NO2	nitrogren 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 cm3/mol/sec	2
PSO4	PM sulfate	4
RCHO	Lumped aldehyles with 3+ carbons	2
RCO_OH	Higher organic acides	2
SO2	Sulfur dioxide	5
SULF	Sulfates (SO3 or H2SO4)	5
TRP1	Terpenes	2

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

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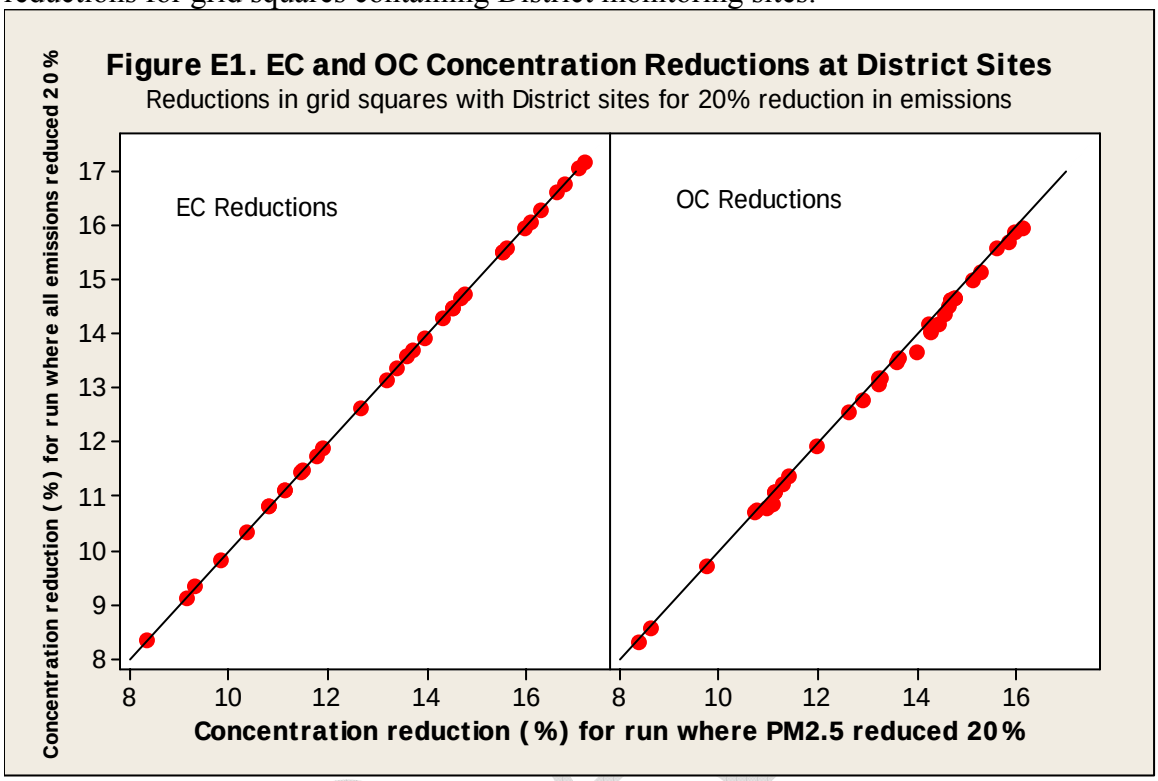
One sensitivity run (4 in Table E1), reduced all directly emitted $PM_{2.5}$. There was also a run where all emissions except for CO were reduced 20%. Figure E1 shows the percent reductions for grid squares containing District monitoring sites.



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 and the 20% run where all emissions were reduced, so that except for reductions in directly emitted EC and OC, 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 is 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.

Table E2. Comparison of ambient and modeled OC and EC

	OC ($\mu g/m^3$)		EC ($\mu g/m^3$)	
	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 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($\mu g/m^3$)	OC($\mu g/m^3$)	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 is considerably more emissions of organic carbon precursors, such as alpha pinene. 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*6.02 = 3.15 $\mu g/m^3$ of the 3.87 $\mu g/m^3$ 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 was 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, 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

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

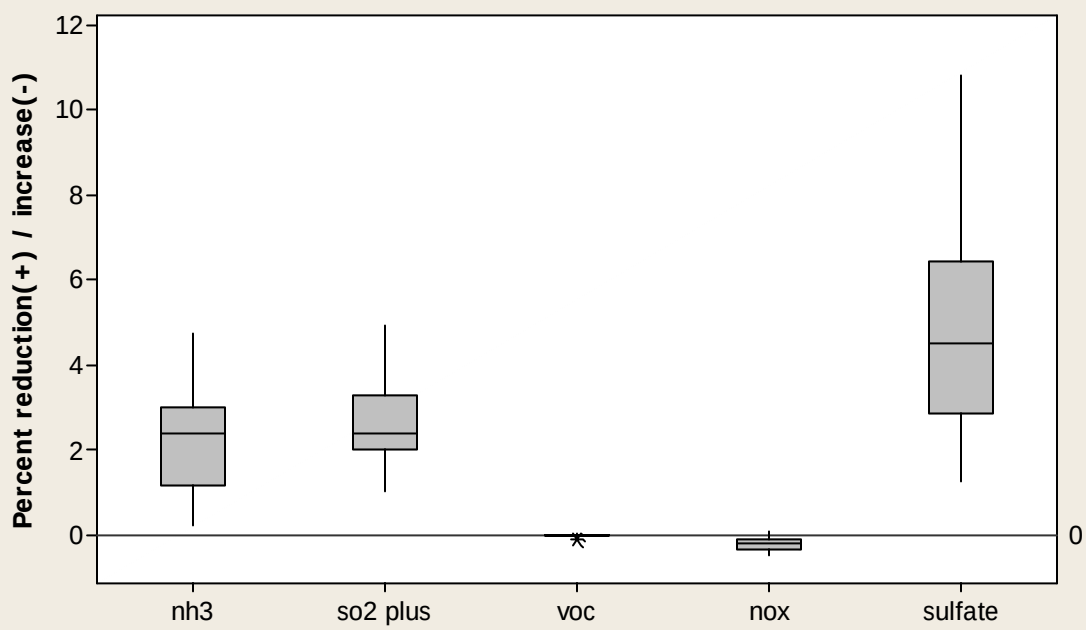
Where EC_{base} and OC_{base} are the EC and OC concentrations from the base case and EC_{20} and OC_{20} are the EC and OC concentrations from the run with 20% lower direct $PM_{2.5}$ emissions.

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, NOx, 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% to 3% for nh3. The "whiskers" represent the 5th and 95th percentiles. For nh3, the lower is slightly above 0% to almost 5%.

Figure E2. Percent Change in Ambient Sulfate Concentrations
for a 20% reduction in its precursors, for grid cells containing District sites



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 ammmonium 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 a reduction 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 R^2 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, so2 and directSO4 are the reductions in sulfate for the individual runs: nh3, sulfur gases, and directly emitted $PM_{2.5}$, with all reductions in $\mu g/m^3$.

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 PM_{10} 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.

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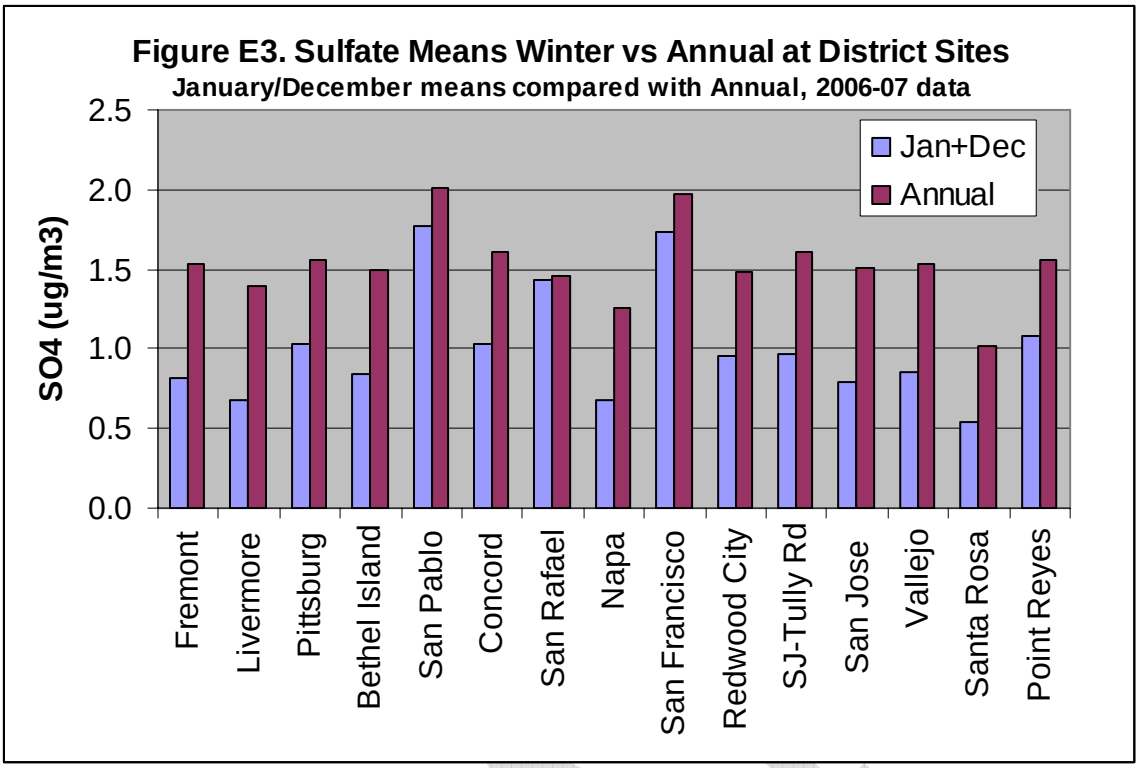


Figure E3 shows annual and January/December means for 2006-07 from the sites. Unlike many Bay Area $PM_{2.5}$ species, sulfate concentrations are lower in winter.

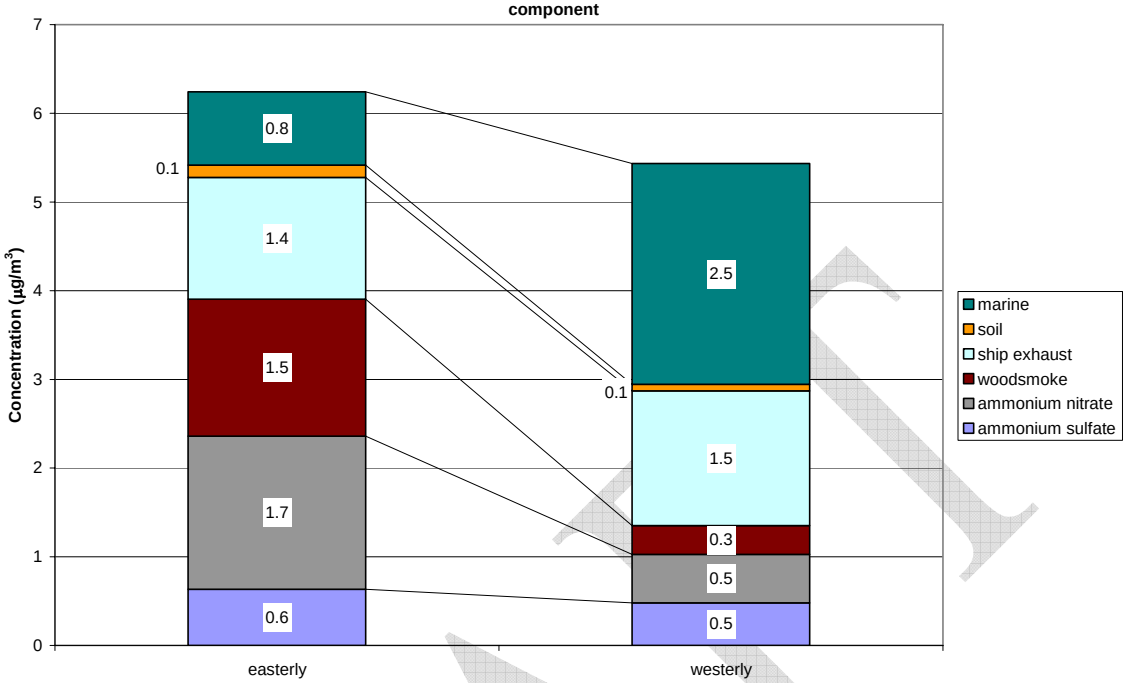
A simple way to extend from winter to annual would be to scale modeled wintertime concentrations by the annual/winter ratio. But the goal of the extension is to estimate how annual reductions in sulfate precursor emissions will affect annual average sulfate concentrations.

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 occuring 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. Thus, there is no basis for making an adjustment to the modeled concentrations.

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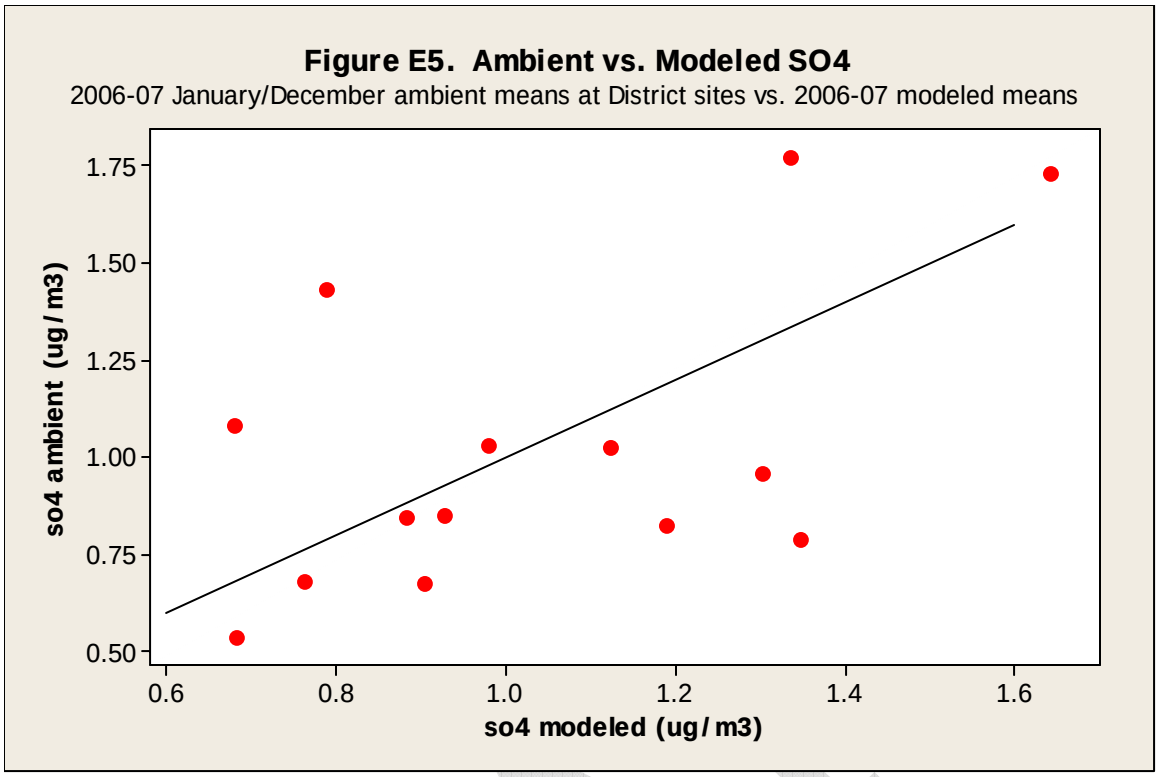


Thus, no additional adjustments were made either to extend to the full year or to correspond to ambient measurements.

Finally, to convert from sulfate to ammonium sulfate $(NH_4)_2SO_4$, we multiply by the ratio of atomic weights: 132/96.

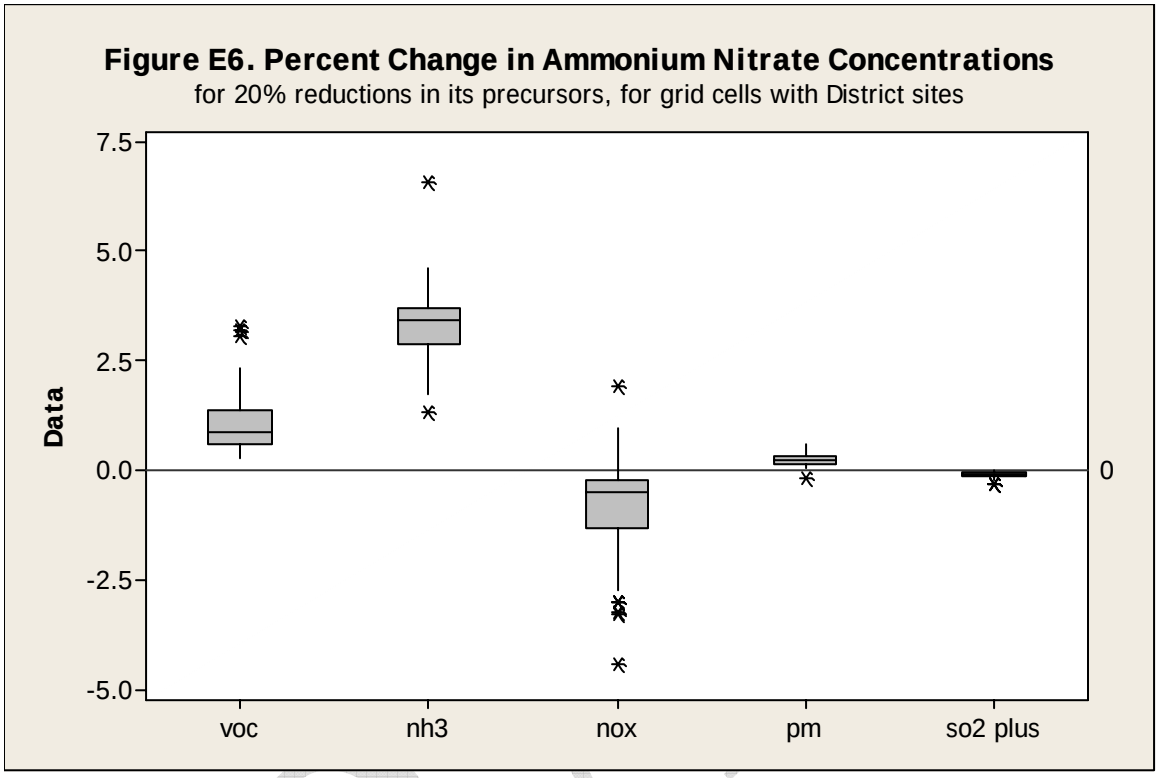
Thus, to estimate reductions in ammonium sulfate concentrations from a 20% percent emissions reduction of its precursors, we use:
where nh3, sulfur gases, and directSO4 are the modeled reductions in sulfate emissions (ug/m3) from the runs reducing ammonia, sulfur gases and PM individually by 20%.

E4. Ammonium Nitrate and its Precursors

Ammonium nitrate derives almost exclusively from secondary atmospheric reactions. The key precursors are ammonia, NOx 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, NOx, 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% to 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 NOx 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, a 20% reduction in NOx causes and *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 nh3 + 1.12 voc + 1.00 nox + 1.70 pm

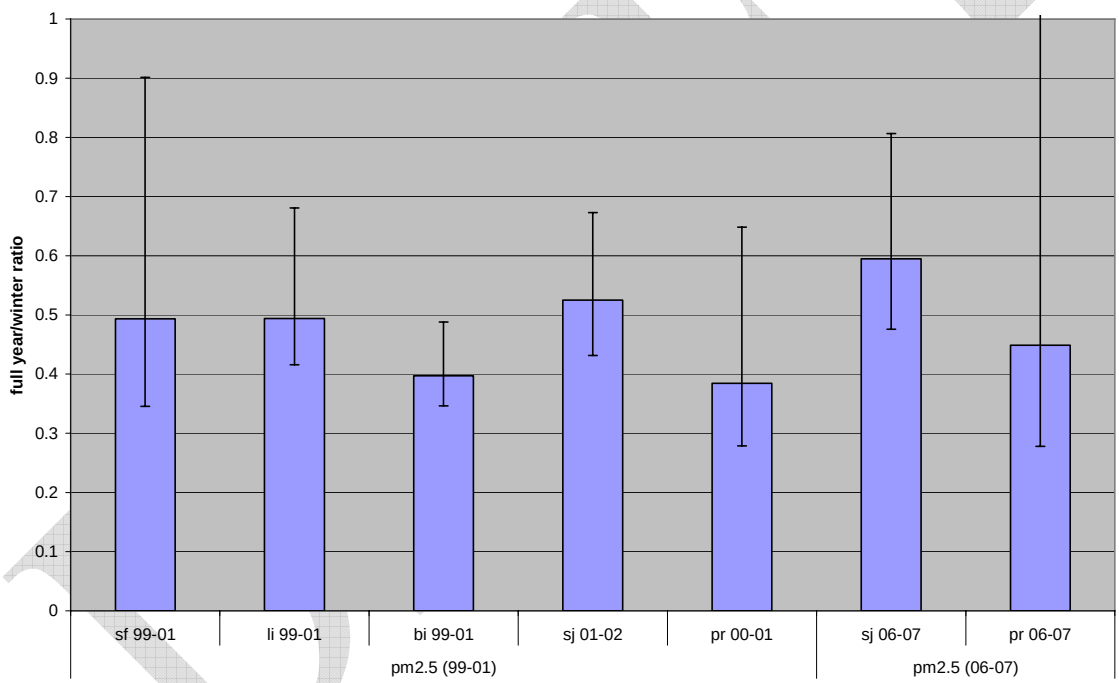
with an adjusted R^2 of 99.1%, where y is the reduction in ambient nitrate from run 6, and nh3, voc, nox and pm, the reductions from the curresponding runs, the units being ug/m^3. The high R^2 value indicates that nitrate formation is well-approximated by a linear function of its individual precursors. 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 represetative 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 $(NH_4)NO_3$, 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.

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

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

Based on a comparison of ambient San Jose NOx and nitrate (NO_3) 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 NOx concentration, where both are expressed in $\mu g/m^3$.

Background

The issue of how much a given reduction in NOx reduces ammonium nitrate has been important in several contexts, including the use of NOx 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 NOx.

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 NOx, ammonia and also ROG, and also depend on meteorological conditions. Ammonium nitrate formation dynamics have been estimated through comparisons of ambient NOx and nitrate (NO_3) 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 NOx 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 NOx 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 = NOx + reaction products of NOx) 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 NOx reductions could actually increase nitrate in many parts of the Bay Area. However, the modeling is still preliminary. As yet, 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 NOx 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 NOx concentrations to aid us in estimating the effects of NOx reductions.

Note: In what follows, NOx concentrations are compared with concentrations of NO_3 on a mass basis. In the Conclusion section of this appendix, the results are applied to the relation between NOx and ammonium nitrate (NH_4NO_3), 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.[22] 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 PM_{10} 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.[23]

NO and NO_2 data was available for all of the site-years just described. NOx was computed on a mass basis, namely converting from ppb to ug/m3 via: 1.065*NO + 1.633*NO_2.

[22] Similar monitoring began at Livermore and Vallejo in September, but the data are still insufficient to project an annual average.

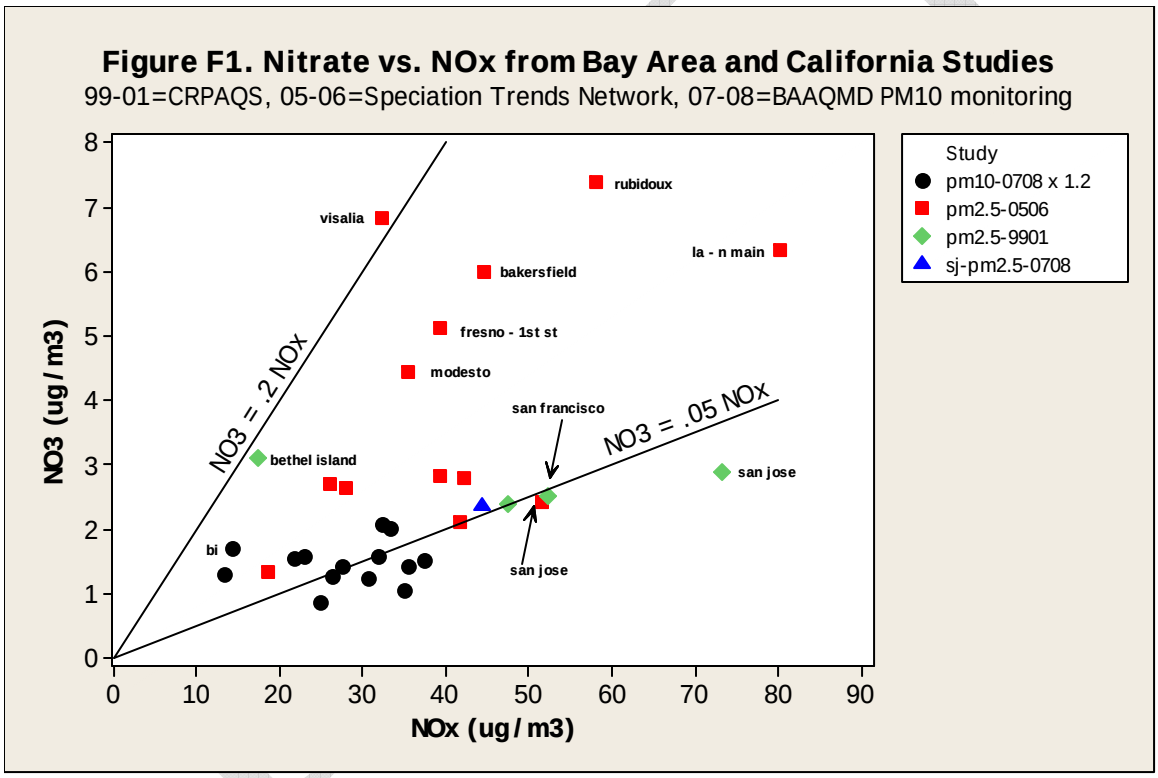
[23] Nitrate is volatile and evaporates off filters unless the filters are refrigerated. The STN and CRPAQS $PM_{2.5}$ were refrigerated, but the PM10 filters weren't. The result is that the PM10 nitrate can be considerably underestimated especially in the winter as the filters are taken from the cool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

Results

Figure F1 shows annual average nitrate (NO_3) vs. annual average NOx for the site-years indicated above. The District PM_{10} nitrate data is multiplied by 1.2 as a rough adjustment for measurement error.[24] The 2007-08 PM_{10} District sites, represented by black circles, fall near the line NO_3 = 0.05 NOx, as do the green triangles representing CRPAQS Livermore and SF values, and the blue triangle representing San Jose 2008.

The one exception is Bethel Island, both of whose values are closer to the line NO_3 = 0.20 NOx. Bethel Island lies in the Central Valley and its high NO_3/NOx 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 NO_3 = 0.13 NOx.



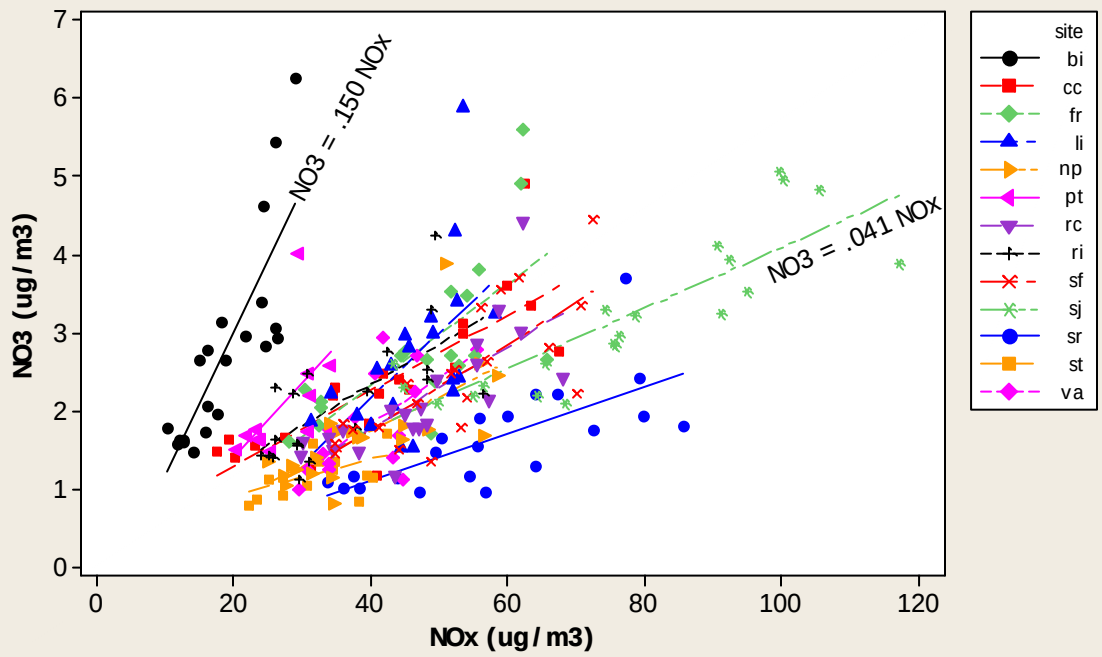
[24] The ratios of $PM_{2.5}$ nitrate to PM_{10} nitrate were 1.19 for San Jose 2008, 1.23 for Livermore 2000, 1.39 for SF 2000, and 1.50 for Bethel Island. BI is likely not representative of much of the rest of the Bay Area. The choice of 1.2 seemed roughly correct and, if anything, conservative.

NO_3 and NOx annual means

Figure F2 shows annual PM_{10} nitrate means[25] vs. annual NOx means at various Bay Area sites for 1988-2008. There is a positive relationship at every site, with higher NOx values associated with higher nitrate values. Also, both NOx and nitrate are trending downward so the plots show that the downtrend in NOx is matched by a downtrend in NO_3.

The figure also shows the regression lines for NO_3 on NOx. 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.

Figure F2. Nitrate (x1.2) vs. NOx annual means at Bay Area sites 1988-2008



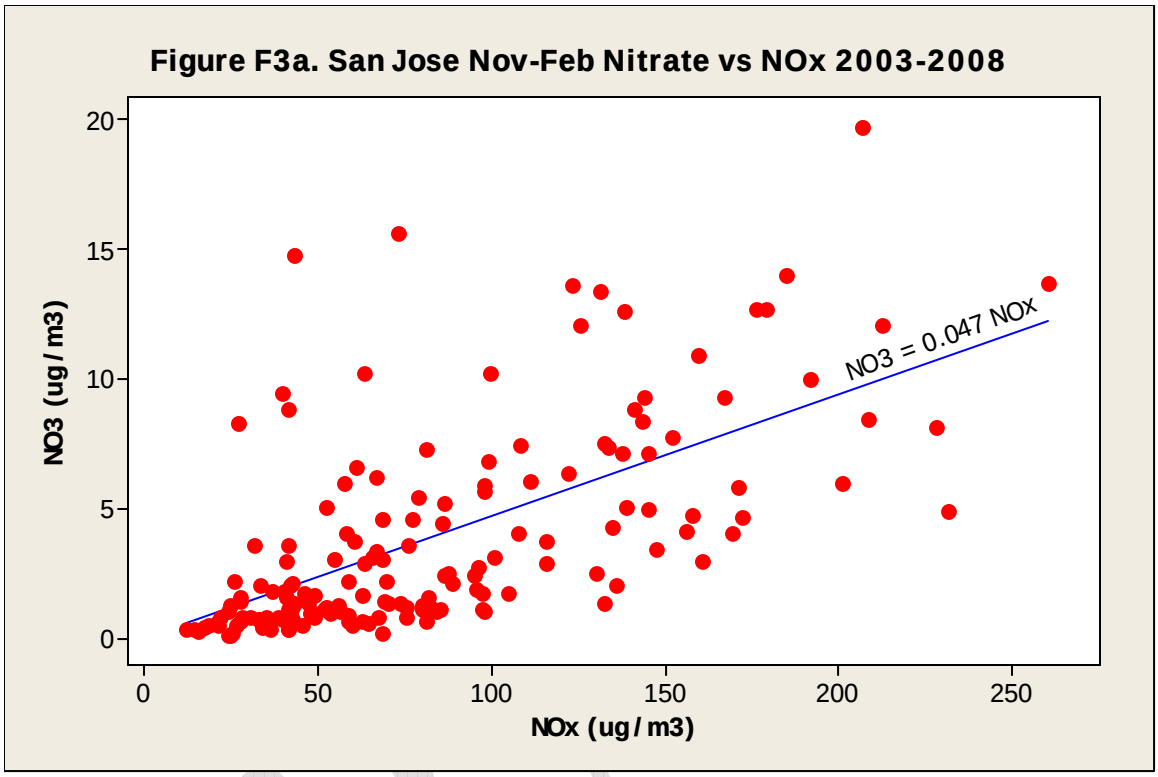
San Jose daily NO_3 vs. NOx

Another approach is to pair daily NO_3 values with daily NOx 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.

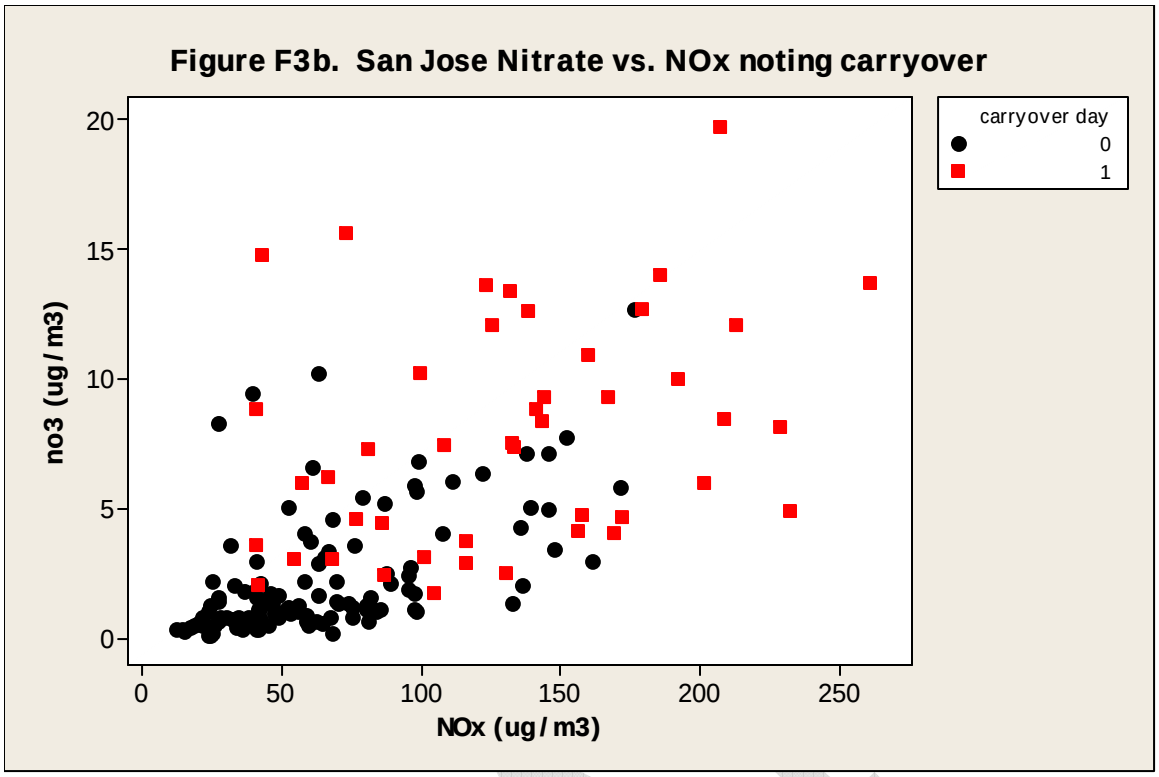
Figure F3a. shows the wintertime values. There is a significant relationship with a linear regression equation of NO_3 = 0.047 NOx.

[25] 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.



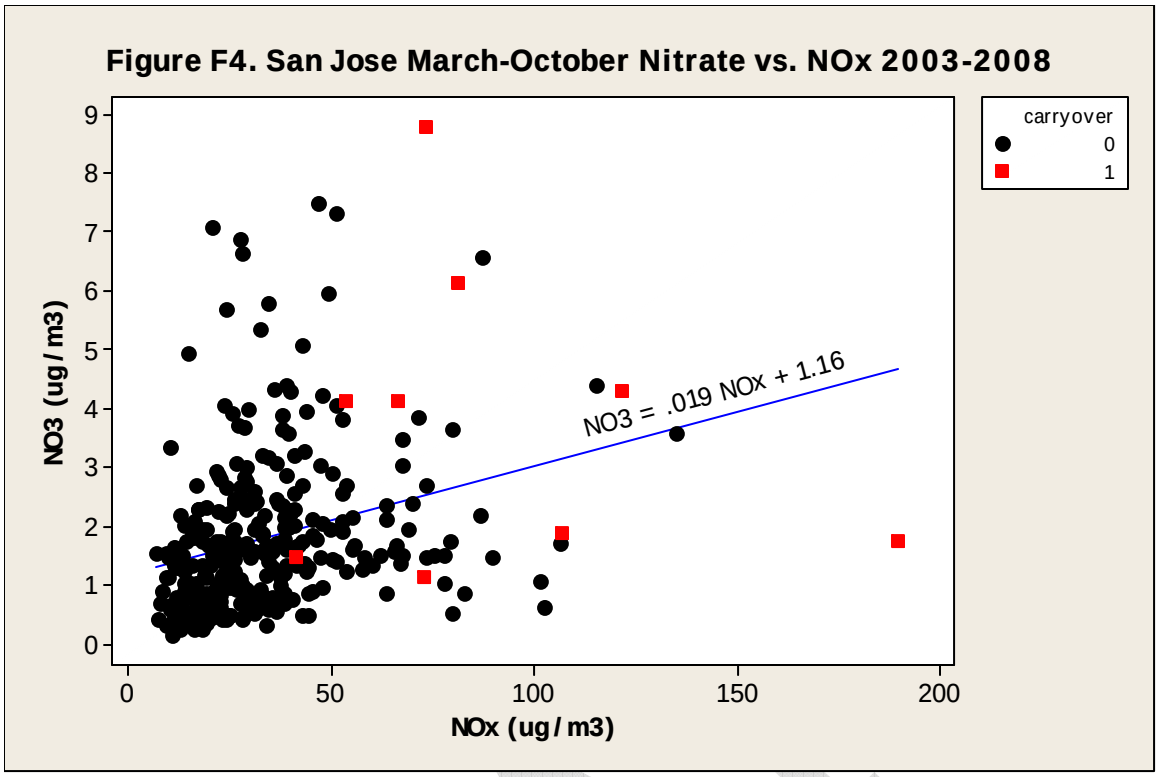
To address 1), I estimated carryover based on the number of days with light winds and little rain. Figure F3b. is the same as F3a except that "carryover days" are indicated, where carryover days are defined as having at least 3 successive days with winds < 5 mph and San Jose rain < 0.02 inches.



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 $x_3 = PM_{2.5}$ – 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.

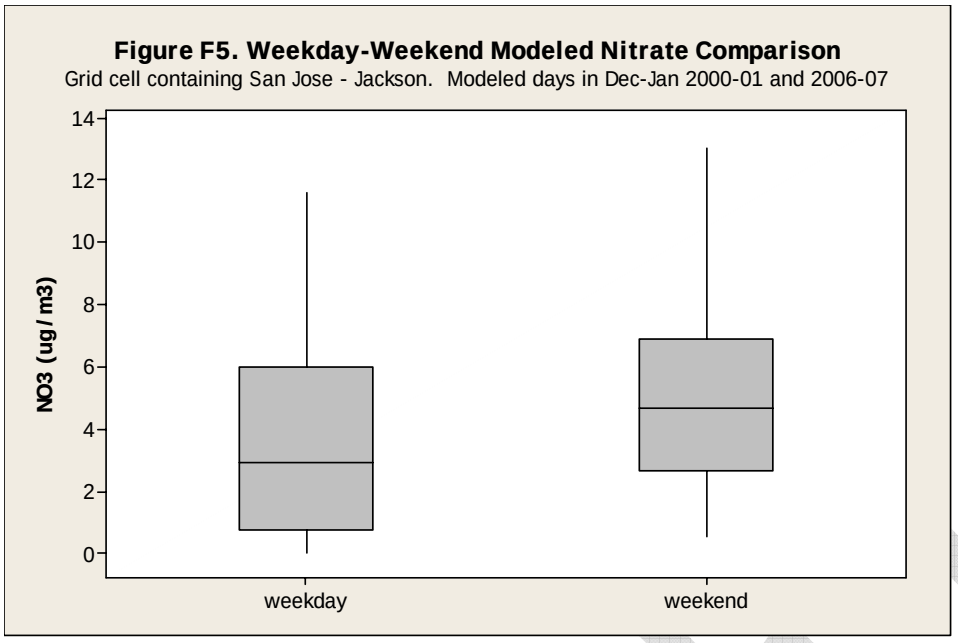


Figure F5 shows the nitrate distribution for the grid cell containing San Jose for weekdays and weekends. The figure shows that, if anything, modeled weekend nitrate is greater on weekends than weekdays, with a weekend mean of 5.2 $\mu g/m^3$ vs. 4.1 $\mu g/m^3$ for weekdays, and medians 4.7 and 2.9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

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

Table 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 $\mu g/m^3$ *increase* in ammonium nitrate District-wide, and an increase of 0.10 $\mu g/m^3$ 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.

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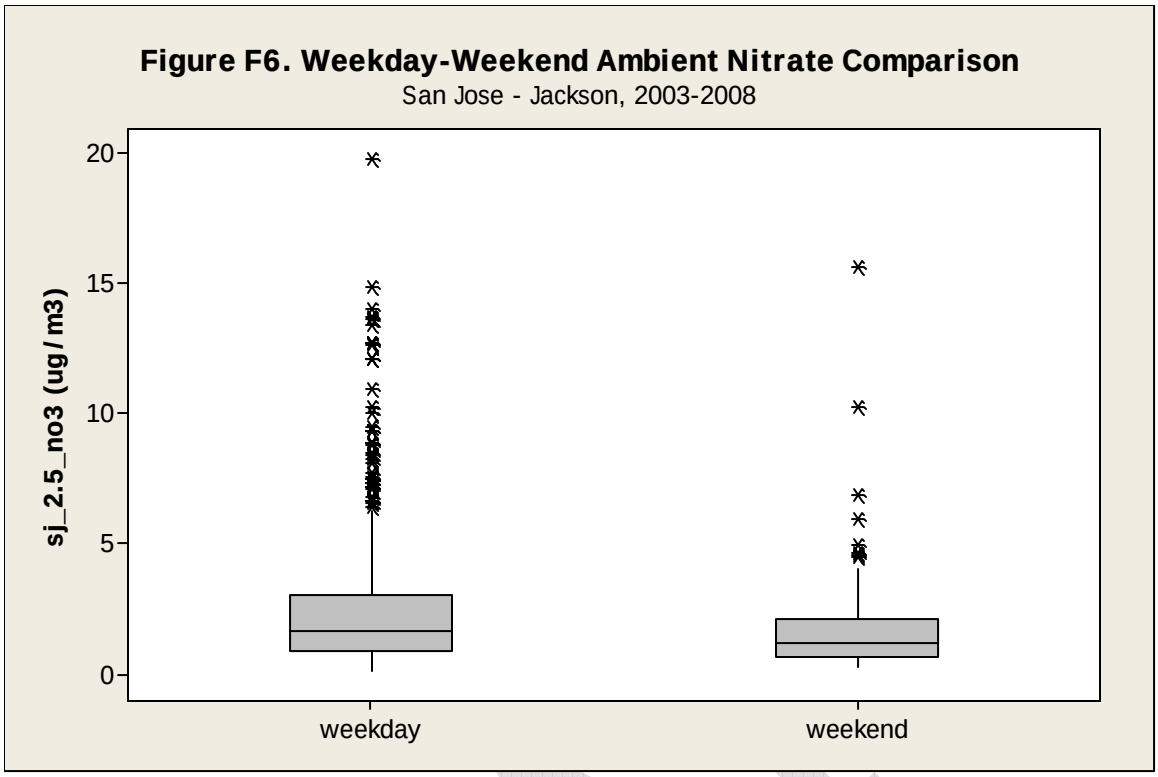

The figure shows that weekends generally have lower nitrate concentrations than weekdays. The mean weekday value was 2.6 μg/m^3 and the weekend mean was 1.9. This difference is statistically significant based on a two-sample t-test. The medians were 1.65 and 1.18 respectively and the difference is significant (p-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 μg/m^3 close, and statistically indistinguishable from, the ambient mean of 4.1 μg/m^3. But whereas the modeled weekend mean is 1.1 μg/m^3 *greater* than the weekday, the ambient weekend mean is 0.9 μg/m^3 *less* than the weekday.

Table 2. Mean ambient and modeled San Jose nitrate (μg/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.[26]

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

	nitrate($\mu g/m^3$)	NOx ($\mu g/m^3$)
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 $\mu g/m^3$ NOx corresponds to a drop of 0.029 $\mu g/m^3$ nitrate. This comports well with the previous memo, which concluded that a drop of 1 $\mu g/m^3$ NOx corresponds to a 0.025 $\mu g/m^3$ drop in nitrate.

Discussion

The above analysis is correlational; it can't 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 NO_3 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.

[26] 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 $PM_{2.5}$ - ammonium nitrate in the regression probably overcompensates for the effect of atmospheric inversions. $PM_{2.5}$ and NOx 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 NOx on ammonium nitrate. This suggests that 0 (the x_1 slope of the regression with x_1, x_2 and x_3 all in the regression) serves as a lower bound for the effect of NOx and NO_3.

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 NOx 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 NOx *caused* lower weekend nitrate levels, the relationship is based on the balance between NOx, ROG and ammonia that existed between 2003 and 2008. In the future, the balance might change and that change might change the effect of NOx reductions on nitrate concentrations.

Conclusion

The ambient data suggest a modest but positive effect of NOx on ammonium nitrate in the Bay Area. For the multi-pollutant method, we choose 0.025 as the factor estimating the concentration ($\mu g/m^3$) of nitrate from a given concentration ($\mu g/m^3$) of NOx. For converting from NOx to ammonium nitrate (on a mass basis), the factor is 0.025 x (80/62) = 0.032.

NOx emissions to ammonium nitrate concentrations

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

For now, we rely on a rough relation between the mean annual NOx among Bay Area sites, 33 $\mu g/m^3$ in 2005, compared with the annual tons/day from the 2005 annual emissions inventory, 521 tons/day. This suggests that one ton/day of NOx produces 33 / 521 $\mu g/m^3$ = .063 $\mu g/m^3$ NOx concentration.

Finally to convert from nitrate to ammonium nitrate, we need to multiply by 80/62, the ratio of the molecular weights. Thus, the formula to predict ammonium nitrate reductions for a given reduction in NOx emissions of z tons/day will be:

Δammonium nitrate = (80/62) * .025 * 33 * (z/n)

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

References

CARB 2003. "Airborne toxic control measure for stationary compression-ignition engines," Stationary Source Division, Emissions Assessment Branch. September, 2003.

CARB 2005. "Recommended conversion factors for secondary formation of PM-nitrate from NOx emissions", Air Resources Board Research Division, September 2005.

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.

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

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

Bivariate Normal Case

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

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

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

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

where $\beta > 0$, by assumption.

(See, e.g., Introduction to the Theory of Statistics, by A. M. Mood, F. A. Graybill, and D. C. Boes, 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^2_δ, in other words, we measure X with error, and the measurement is unbiased. Then $\mu_{x*} = E(X^*) = E(X) + E(\delta) = \mu_x$,
$\sigma^2_{x*} = Var(X^*) = Var(X) + Var(e) = \sigma^2_x + \sigma^2_\delta$, and

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

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

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

Simple linear regression

In the simple linear regression model, we have

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

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

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

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

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

The simple linear regression fit yields

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

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

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

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

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

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

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

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

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

The inequality doesn't always hold. For example, suppose $d_i = - x_i/2$. 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.

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

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

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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.

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