Attachment D

Method Used to Quantify Health Benefits of Draft Scoping Plan Co-Pollutant Emission Reductions

Introduction

The methodology described in Appendix A of ARB's 2006 Goods Movement Emission Reduction Plan (GMERP) was used to quantify the health benefits of the Draft Scoping Plan measures both statewide and in the South Coast Air Basin. This analysis used an emission inventory for criteria pollutants structured similarly to ARB's greenhouse gas inventory. This enables the benefits to be more readily compared with calculated benefits from existing California programs to reduce criteria pollutant emissions.

Summary of Methodology

The methodology ARB uses for quantifying premature death and other health impacts from PM exposure is similar to a methodology used by the U.S. Environmental Protection Agency for risk assessments (U.S. Environmental Protection Agency. Regulatory impact analysis for the final Clean Air Interstate Rule. Office of Air and Radiation. EPA-452/R-05-002, 2005).

This methodology was peer-reviewed during the development of the GMERP. ARB augmented EPA's methodolgy by incorporating the results of new epidemiological studies relevant to California's population as they became available.

This assessment of co-benefits of the Draft Scoping Plan focuses on health outcomes associated with PM2.5 air pollution. The key pollutants contributing to PM2.5 air pollution are NOx and directly emitted PM2.5. Assessing the ozone impacts without complex photochemical modeling would be difficult. In addition, PM2.5 exposures account for most of the estimated premature mortality associated with air pollution in California.

The following health endpoints were included in the analysis:

- **Premature death:** A death which occurs at a younger age than would be expected. Air pollution is not implicated as the *cause* of death, but rather a contributing factor in someone whose health is typically already compromised, thereby accelerating the time of death.
- **Hospital admissions for respiratory and cardiovascular causes:** Hospitalization admissions for causes including pneumonia, chronic obstructive pulmonary disease (COPD), asthma, heart attack, stroke, congestive heart failure and cardiac arrhythmia.
- **Asthma and lower respiratory symptoms:** Symptoms include cough, phlegm production, chest pain, or wheeze, associated with the lower respiratory tract (windpipe, lungs, and airways leading to/associated with the lungs).
- **Acute bronchitis:** Inflammation of the main airways to the lungs, resulting in symptoms such as hacking cough and phlegm production.
- **Work loss days:** Days of missed work for members of the population age 18-65.
- **Minor restricted activity days:** Days when a person is not able to engage in their usual range of activities due to minor health conditions (does not include work loss or bed confinement).

For the Scoping Plan co-benefits analysis, some refinements to the methodology used for the GMERP were necessary. They are as follows:

- **Updated emission inventories**. Since the adoption of the GMERP, the baseline emissions used to develop the above "tons per death" factors were revised to be consistent with the inventories used during the development of the statewide 2007 State Implementation Plan (SIP). The major categories which were updated included but are not limited to: on-road vehicles, ocean-going vessels, construction equipment, and commercial harbor craft.
- **Updated "tons-per-case" factors for health endpoints.** Based on the updated emission inventories, the "tons-per-case" factors were updated since the release of the GMERP.

APPENDIX A

QUANTIFICATION OF THE HEALTH IMPACTS AND ECONOMIC VALUATION OF AIR POLLUTION FROM PORTS AND GOODS MOVEMENT IN CALIFORNIA

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Appendix A Table of Contents

Executive Summary **5**
I. Introduction **9**
A. Overview of the Environmental Challenge 9
B. Community Concerns 9
II. Background **11**
A. Sources of Concern 11
B. Emissions 11
C. Previous Risk Assessments 15
D. Air Pollutants of Concern 16
1. Health Effects Associated with PM and/or Ozone 18
2. Selection Concentration-Response Functions for Quantified Analysis 19
3. Unquantified Adverse Effects 40
4. Community Health Impacts 40
III. Methodology **43**
A. Air Pollutant Emissions from Goods Movement-Related Sources 43
1. Ocean-going Ships 43
2. Commercial Harbor Craft 45
3. Cargo Handling Equipment 46
4. Trucks 47
5. Locomotives 51
B. Adjustment Factor for Ship Emissions 52
C. Exposure Estimates 54
1. Diesel PM 54
2. Particle Nitrate and Particle Sulfate 54
3. Secondary Organic Aerosols 59
4. Ozone 60
D. Health Impacts Methodology 60
1. Particulate Matter 61
2. Ozone 62
3. Port-Specific Modeling 63
E. Economic Valuation of Health Effects 64
F. Uncertainty Calculations 67
IV. Results **69**
A. Emissions Estimates 69
B. Exposure Estimates 69
C. Health Impacts Assessment 70
1. Statewide Impacts 71
2. Air Basin-Specific Impacts 71
3. Source-Specific Impacts 74
4. Pollutant-Specific Impacts 74
5. Cancer Risk 75
6. Port-Specific Impacts 76
D. Economic Valuation of Health Effects 76

Appendix A Table of Contents (continued)

V. Discussion 78
A. Health Impacts Assessment 78
1. Statewide Impacts 78
2. Sensitivity Discussion 79
3. Port-Specific Impacts 80
B. Uncertainties and Limitations 82
1. Uncertainty Associated with Emissions Estimation 82
2. Exposure Estimates and Populations 83
3. Concentration-Response Functions 85
4. Baseline Rates of Mortality and Morbidity 87
5. Health Effects from Sulfate Exposure 87
6. Unquantified Adverse Effects 88
7. Uncertainty Associated with Economic Valuation 89
C. Ongoing Studies to Reduce Uncertainties 89
1. Emissions 89
2. Exposure 90
3. Health and Environmental Justice 91
4. Economics 94
VI. References 96

Executive Summary

The California Air Resources Board (CARB) staff assessed the potential health effects associated with exposure to air pollutants arising from ports and goods movement in the State. This health impacts assessment focused on particulate matter (PM) and ozone as they represent the majority of known risk associated with exposure to outdoor air pollution, and there have been sufficient studies performed to allow quantification of the health effects associated with emission sources. This assessment quantifies the premature deaths and increased cases of disease linked to exposure to PM and ozone from ports and goods movement, and provides an economic valuation of these health effects. Because of the uncertain nature of several key inputs and methodologies, these results will be refined over time.

Background

Port and goods movement-related emission sources, which are mostly diesel engines, emit PM directly (i.e., diesel PM) and form additional PM (i.e., particle nitrate, particle sulfate, secondary organic aerosols) through chemical reactions and physical processes in the atmosphere involving emitted nitrogen oxides (NO_X), sulfur oxides (SO_X), and reactive organic gases (ROG). Emissions of NO_X and ROG also contribute to ozone formation through atmospheric reactions.

Population-based studies in hundreds of cities in the U.S. and around the world demonstrate a strong link between elevated PM levels and premature deaths, increased hospitalizations for respiratory and cardiovascular causes, asthma and other lower respiratory symptoms, acute bronchitis, work loss days, and minor restricted activity days. Ozone is linked to premature death, hospital admissions for respiratory diseases, minor restricted activity days, and school absence days in other scientific studies. Attaining the California PM and ozone standards statewide air quality would annually prevent about 9,000 premature deaths[1] (4% of all deaths)[2] with an uncertainty range of 3,000 to 15,000 based on 1999-2000 PM and 2001-2003 ozone monitoring data. This is greater than the number of deaths (4,200 to 7,400) linked to second-hand smoke in the year 2000. In comparison, motor vehicle crashes caused 3,200 deaths and there were 2,000 homicides.

Air pollution has a serious impact on the State's economy. An annual value of about $2.3 ($1.5 to 2.8 uncertainty range) billion is associated with hospitalizations and the treatment of major and minor illnesses related to air pollution exposure in California. In addition, the value of preventing premature deaths resulting from exposure to air pollution in excess of the State's PM and ozone standards is estimated to be $70 ($22 to 131 uncertainty range) billion.

Methodology

The methodology used to quantify the adverse health effects of PM and ozone is based on concentration-response functions – relationships between adverse health outcomes (for a population group) and air pollution levels. The fraction of PM and ozone pollution attributable to ports and goods movement was estimated from scaling factors (based on measurements and air quality modeling) linking air basin-wide emission inventories of diesel PM, other PM2.5 sources (e.g., ship boilers, truck brake

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[1] Calculated using concentration-response function for PM2.5 and premature death from Pope et al. 2002, which resulted in a 25% increase over estimates based on Krewski et al. 2000. The U.S. EPA also uses this study (e.g., see http://www.epa.gov/interstateairquality/pdfs/finaltech08.pdf).

[2] According to the Department of Health Services, there are about 235,000 annual deaths due to all causes in California (based on 2001-2003 data)

and tire wear), NO_X, and ROG to outdoor levels of PM components (diesel exhaust, particle nitrate, secondary organic aerosols) and ozone. A similar analysis for particle sulfate formed from SO_X emissions was also attempted, as described below.

Results

Table A-1 displays the estimated premature deaths and other health outcomes that can be associated with PM and ozone exposure from port-related goods movement and other port activities for the current year (2005). The estimated economic value of eliminating these adverse health effects, due mostly to avoided premature deaths but also to savings in health care expenditures, is also shown. Primary diesel PM accounts for 50% of the risk, followed by nitrate particles. Since it takes several hours to form nitrate particles from NO_X emission sources, risks are more uniformly distributed over an air basin than from diesel PM, which is highest for those living closest to the sources. The South Coast Air Basin dominates the risk (50% of goods movement-related deaths statewide), followed by other coastal air basins – San Francisco Bay Area, San Diego County, and South Central Coast. Not one source type dominates the risk and all contribute at least 5 to 10% to the total. Valuations are in year 2005 dollars and should be interpreted as the economic value of avoiding these adverse health impacts. They are not the costs of implementing the reduction strategies, which are presented in the main report.

Table A-1 Annual (2005) Statewide PM and Ozone Health Effects Associated with Ports and Goods Movement in California[1] .

Health Outcome	Cases per Year	Uncertainty Range[2] (Cases per Year)	Valuation (million)	Uncertainty Range[3] (Valuation - million)
Premature Death	2,400	720 to 4,100	$19,000	$5,900 to $36,000
Hospital Admissions (respiratory causes)	2,000	1,200 to 2,800	$67	$40 to $94
Hospital Admissions (cardiovascular causes)	830	530 to 1,300	$34	$22 to $53
Asthma and Other Lower Respiratory Symptoms	62,000	24,000 to 99,000	$1.1	$0.44 to $1.8
Acute Bronchitis	5,100	-1,200 to 11,000	$2.2	$-0.52 to $4.7
Work Loss Days	360,000	310,000 to 420,000	$65	$55 to $75
Minor Restricted Activity Days	3,900,000	2,200,000 to 5,800,000	$230	$130 to $350
School Absence Days	1,100,000	460,000 to 1,800,000	$100	$41 to $160
TOTAL VALUATION	NA	NA	$19,000	$6,000 to $36,000

[1]Does not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies.

[2]Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates. A negative value as a lower bound of the uncertainty range is not meant to imply that exposure to pollutants is beneficial; rather, it is a reflection of the adequacy of the data used to develop these uncertainty range estimates. Additional details on the methodology and the studies used in this analysis are given in later sections of this Appendix.

[3]Range reflects uncertainty in health concentration-response functions for morbidity endpoints and combined uncertainty in concentration-response functions and economic values for premature death, but not in emissions or exposure estimates.

Projecting future population and ports and goods movement emissions growth and control (for already adopted measures outside of the proposed plan) to the year 2020

results in 1,700 (500 to 2,800 uncertainty range) remaining deaths per year (Table A-15), with an estimated annual value (in 2005 dollars) of $5.5 to 9.4 ($2 to 18) billion. The contribution of PM outweighs that of ozone by tenfold. Primary diesel PM is presently the major contributor to the total estimated premature deaths attributable to ports and goods movement, but, in 2020, secondary diesel PM (i.e., particle nitrate) becomes the most significant contributor as measures are already in place to be effective in controlling primary diesel PM emissions in the long run.

The proposed year 2020 mitigation strategies presented in the main report are expected to result in a reduction of 820 (240 to 1400) deaths annually, with an estimated value of $2.7 to 4.7 ($0.9 to 8.8) billion. Without the implementation of the proposed mitigation strategies, cumulative deaths due to ports and goods movement emissions from 2005 to 2020 are estimated to be 7,200 (2,100 to 12,000 uncertainty range) with an economic value of $33 to 46 ($10 to 87) billion. Further discussions of the benefits and costs of the proposed mitigation strategies can be found in Chapter IV in the main body of the report.

Peer and Public Review

All the concentration-response functions originate from peer-reviewed scientific journals, and several key components of this assessment (i.e., diesel PM exposure estimates, PM and ozone health benefit methodology, economic valuation) have previously undergone peer reviews conducted by the California EPA's Scientific Review Panel, the University of California Office of the President, or the U.S. EPA's Scientific Advisory Board. Several university and government agency scientists commented on the calculation methodology proposed for the assessment in November 2005. Ten scientists reviewed the December 1, 2005 draft report in parallel with the public review. Their comments are presented in Section F of the Technical Supplement and, to the extent possible, incorporated into the revised assessment. Public comments are summarized in Section G of the Technical Supplement and were also incorporated into the revised assessment to the extent possible.

Recent Studies of Premature Death

A recent study (Jerrett et al. 2005) which analyzed PM exposure for Los Angeles found a 2.5 times higher estimate for premature death than the 51-city national study by Pope et al. (2002), but greater uncertainty. The 2.5-times higher result appears to be due to better exposure characterization techniques rather than higher toxicity of the PM mixture in Los Angeles. Several additional studies that have either just been published or will be in the next few months will help resolve this issue. CARB staff intends to review all of these studies and will solicit the advice of the study authors and other experts in the field and U.S. EPA to determine how to best incorporate these new results into future versions of health assessments for ports and goods movement.

Particle Sulfate

The December 2005 draft of this report did not include a quantitative health assessment of particle sulfate formed from goods movement-related emissions of SO_X. Any analysis is complicated by the fact that, in addition to sulfate formed from fossil fuel use in California, there are three other sources of atmospheric sulfate in California – natural "background" sulfate formed over the ocean by biologic activity, global "background" sulfate that is distributed throughout the Northern Hemisphere by the upper air westerly winds, and sulfate blown into Southern California from combustion in Mexico. New analyses of air quality and emissions data conducted in the intervening period indicate that uncontrolled SO_X emissions from ships increase the estimates of total goods movement-related health effects by about one quarter.

However, this preliminary estimate contains several uncertainties and a fully quantitative analysis must await the completion (by end of 2006) of research being jointly conducted by CARB staff, five university groups, the U.S. EPA and its contractors, and Environment Canada as part of a feasibility study for establishing a SO_X Emission Control Area (SECA) to reduce sulfur emissions from West Coast shipping. The research includes a refined inventory of ship activity and ship emissions, analysis of historical PM data from sites along the West Coast to look for evidence of ship emissions, development of new monitoring methods that can distinguish fossil fuel sulfate from that due to biologic activity in the ocean, and model development to allow simulation of sulfate formation and transport over the ocean and land areas of coastal California.

Other Uncertainties

There are significant uncertainties involved in quantitatively estimating the health effects of exposure to outdoor air pollution. Uncertain elements include emission and exposure estimates, concentration-response functions, baseline rates of death and disease, occurrence of additional unquantified adverse health effects, and economic values. Many of these elements have a factor-of-two uncertainty, but, over time, some of these will be reduced as new research is completed. However, significant uncertainty will remain in any estimate made over the foreseeable future.

It was not possible to quantify all possible health benefits that could be associated with reducing port-related goods movement emissions. Unquantified health effects due to PM exposures include myocardial infarction (heart attack), chronic bronchitis, onset of asthma, and asthma attacks, as there is some overlap between these and the quantified effects such as lower respiratory symptoms and all respiratory and all cardiovascular hospitalizations. In addition, estimates of the effects of PM on premature birth, low birth weight, and reduced lung function growth in children are not presented. While these outcomes can be significant in any assessment of the public health impacts of air pollution, there are currently few published investigations on these topics, or baseline disease rates for California air basins are not available for some endpoints. In other cases, the results of the studies that are available are not entirely consistent. Nevertheless, there are some data supporting a relationship between PM exposure and these effects, and there is ongoing research in these areas that should help to clarify the role of PM on these health outcomes.

Ongoing Studies

CARB and others fund and conduct studies that will improve our understanding of the emissions, exposure, and health and economic risks of port-related goods movement, especially in the communities closest to the port and associated rail and truck traffic. For example, emission testing of ships, trucks, and trains being conducted now and over the next two years will provide improved activity estimates and chemical speciation profiles. Beginning in fall 2006, the Wilmington Exposure Study will measure air pollution hotspots downwind of the ports, refineries, rail yards, freeways, and local roads. Air quality measurement and modeling to support the State Implementation Plan and a possible SECA designation for North America will improve estimates for particle nitrate, particle sulfate, and ozone during 2006. Over the next 30 months, CARB staff will conduct risk assessments for the 16 largest rail yards in California. As each project is completed, results will be made available to the public.

I. Introduction

The Goods Movement Action Plan: Phase I (BTH and Cal/EPA 2005) identified several elements that will guide efforts to develop a strategic plan for goods movement. One of these elements: "(to) acknowledge the environmental impacts and identify needed resources and strategies to help mitigate those impacts", was the genesis for this current effort.

A. Overview of the Environmental Challenge

The Phase I Report provided a general discussion of the extent of environmental and community impacts of goods movement based on preliminary reports and CARB estimates of port emissions in the South Coast Air Basin (SoCAB). One goal of this report is to provide a more detailed assessment of these environmental impacts, including health impacts, to properly identify potential mitigation strategies. This health impact assessment focuses on the health and attendant economic impacts of air pollution resulting from port-related goods movement throughout the state. Other environmental impacts discussed in Phase I, such as noise and light pollution, traffic-safety concerns, or blight are not within the scope of this analysis.

Emissions from goods movement activities, especially port-related goods movement, have been found to be a significant and growing contributor to regional and community air pollution. Unless further mitigation actions are taken, these emissions will increase with the rapid increase in trade. For instance, according to Phase I and other preliminary environmental assessments, it was estimated that without new pollution prevention interventions, a tripling in trade at the Ports of Los Angeles and Long Beach between the years 2005 and 2020 would result in a 50% increase in nitrogen oxide (NO_X) emissions and a 60% increase in diesel particulate matter (PM) from trade-related activities, during a time when overall air pollution will decrease (CARB 2005a).

A number of air pollutants are associated with goods movement related emissions; however, PM components (diesel exhaust, particle nitrate, particle sulfate, secondary organic aerosol) and ozone are considered to have the greatest impacts on human health. The most severe consequence of increasing emissions of these pollutants would be an increase in the prevalence of diseases such as asthma and heart disease and an increase in the number of premature deaths from cardiopulmonary disease or lung cancer. Increased health care costs, lost work days, and school absenteeism are some of the economic impacts that could result from an increase in disease rates.

B. Community Concerns

This health impact analysis uses air-basin-level emission inventories to evaluate port-related goods movement health impacts for the entire state, but it does not focus on near-source emissions and their potential impacts. Residents in neighborhoods in the vicinity of ports, rail yards or inter-modal transfer facilities, or those along major transportation corridors, are more likely to face greater health risks related to goods movement. Wilmington, City of Commerce, San Francisco's East Bay, and Roseville are examples of communities that may be more affected by port-related activities in comparison to those living elsewhere within an air basin. Many of these communities are made up of people from economically disadvantaged groups who would be the least able to sustain the personal and financial impacts related to increased disease burden. Several community-based air pollution studies and risk assessments have been performed by CARB, the South Coast Air Quality Management District (SCAQMD), and others to evaluate the impact of increased emissions on these populations (i.e., SCAQMD 2000). Many CARB research projects, aimed at increasing

our understanding of these impacts are also currently underway. A brief summary of these studies is provided in Section V-C.

Vulnerable populations in impacted communities throughout the state, including the elderly and children or those with existing health problems, are also likely to suffer more from an increase in air pollutants. Additional CARB projects are being conducted to understand these impacts and descriptions of these studies are also provided in section V-C.

II. Background

The Goods Movement Action Plan: Phase I (BTH and Cal/EPA 2005) provided an example of the environmental impacts associated with goods movement emissions in the SoCAB by examining the potential impacts of two major pollutants: diesel PM and NO_X. In that analysis, emissions from on-road heavy-duty trucks (diesel-fueled), gasoline vehicles, off-road equipment and industrial sources were viewed in comparison to port-related goods movement emissions. Port-related emissions for NO_X were significant in relation to the other emission categories in 2005 and the increase due to growth in the industry by the year 2020 makes them the most important source category by that time. Port-related emissions are expected to account for 20% of the SoCAB's NO_X emissions in 2020. Port emissions of diesel PM, which are now nearly equal to those of off-road equipment, will be over three times higher than off-road equipment in 2020 and at least 14 times that of on-road trucks. The Phase I Report concluded that "extensive actions" would be needed to bring port emissions under control to prevent them from becoming the single largest source of air pollution in the SoCAB.

A. Sources of Concern

Ships, railroads, diesel trucks, and cargo handling equipment are the most important port and goods movement-related emission categories. At the ports, ship emissions dominate and will continue to dominate in terms of the tonnage of emissions for diesel PM and NO_X. This is largely due to the cleaner diesel engines that will be required over time for the other source categories. However, in terms of risk resulting from diesel PM, the near-source emissions – those from sources operating from within the ports and by neighborhoods – will have a greater health impact than emissions further off-shore.

B. Emissions

Vehicles and equipment which move international and domestic goods through California are an important source of emissions. Table A-2 presents estimated statewide emissions related to goods movement in 2001, the base year for this study. On a typical day, we estimate more than 1000 tons per day of NO_X are emitted from statewide goods movement activities in California. NO_X emissions from statewide goods movement lead directly to formation of ozone and secondary particulate, and represent about 30% of the total statewide NO_X emissions inventory. More than seventy tons per day of SO_X were generated by goods movement related activities in 2001.

Emissions of diesel particulate, a known carcinogen, are particularly important; in 2001 diesel particulate emissions generated by ports and international goods movement were estimated to be about 57 tons per day of PM and represented about 75% of the statewide diesel particulate inventory.

Table A-2 2001 Statewide Pollutant Emissions by Goods Movement Source Type
(Tons per Day)

Pollutant	Ships	Harbor Craft	Cargo Handling Equipment	Trucks	TRU	Trains	Total
Diesel PM	7.8	3.8	0.8	37.7	2.5	4.7	**57.3**
NO_X	95	75	21	655	22	203	**1070**
ROG	2	8	3	56	13	12	**93**
SO_X	60	0.4	<0.1	5	0.2	8	**73**

Predicting growth in goods movement activities is a key element of the emissions inventory development process. Based on recent data, it is clear that California is experiencing a major increase in the amount of goods imported to our ports. Between 2000 and 2004, the number of containers measured as twenty-foot equivalent units (TEU) increased by 40% at the Ports of Los Angeles and Long Beach.[3] Between 1990 and 2004 traffic doubled from one to two million TEU per year at the Port of Oakland.[2.] The Southern California Association of Governments (SCAG) believes freight volumes will double or triple in the Los Angeles region over the next two decades[4]. The Bay Area Metropolitan Transportation Commission believes total cargo tonnage will double at the Port of Oakland between 2002 and 2020.[5]

The draft goods movement emission inventory released in December 2005 included growth estimates for international goods movement. With the inclusion of domestic goods movement, we needed to develop estimates of growth for domestic goods separate from the international goods. We also took this opportunity to refine our growth estimates for international goods movement activities. Below we briefly describe our refinements to the international goods movement growth estimates and our approach for determining the expected growth in domestic goods movement activities.

Staff has revised international goods movement growth estimates by making the growth rates of trucks and trains that transport goods to and from ports, consistent with the growth rates applied to ships. These growth estimates are based upon the change in number and capacity of container ships that occurred in the years 1997-2003. Specifically, the change in total installed power of container ships was used to estimate growth. Total installed power is a function of the number and the total size of container ships visiting California between 1997 and 2003. These growth rates agree well with container forecasts projected for the Ports of Los Angeles for the No Net Increase Report[6], Long Beach, and Oakland[5]. This plan assumes the numbers of containers processed by ports in California will nearly double by 2010 and nearly quadruple by 2020 relative to the number of containers processed in 2001.

Trucks and trains not involved in port-related goods movement are expected to grow at slower rates that those transporting goods to and from ports. The fraction of trucks and trains involved in goods movement was estimated, and this fraction was grown using the container ship growth rate described above. The remaining fraction of trucks and trains were grown at slower rates specific for these categories. VMT growth for trucks is largely provided by local planning organizations, and locomotive growth was based on national trend data. Domestic growth rates are projected to be much lower than international growth rates. For example, we expect total truck VMT in South Coast will increase about 80% between 2001 and 2025. At the same time, this plan assumes international truck VMT in South Coast will increase by 170%.

Figure A-1 provides all goods movement and Figure A-2 provides ports and international goods movement emission estimates by pollutant and by year for 2001-2025. While the SO_X emissions for all goods movement are projected to triple, the emissions for other pollutants are projected to decrease by 30 to 45% by 2025, The

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[3] American Association of Port Authorities (2005). US / Canada Container Traffic in TEUs. Available at: http://www.aapa-ports.org/industryinfo/statistics.htm.

[4] Southern California Association of Government (2004), Southern California Regional Strategy for Goods Movement, A Plan for Action. Available at: http://www.scag.ca.gov/goodsmove/pdf/GoodsmovePaper0305.pdf.

[5] San Francisco Bay Conservation and Development Commission and Metropolitan Transportation Commission (2003), San Francisco Bay Area Seaport Plan.

[6] Report to Mayor Hahn and Councilwoman Hahn by the No Net Increase Task Force: June 24, 2005. Available at: http://www.portoflosangeles.org/DOC/NNI_Final_Report.pdf.

emissions from ports and international goods movement increase with the dramatic growth in imported goods. By 2025 diesel particulate emissions are projected to more than double and SO_X emissions are projected to quadruple. NO_X emissions are projected to increase more than 70% by 2025, primarily in areas that are currently not in attainment with air quality standards.

Figure A-1 Statewide Goods Movement Emissions

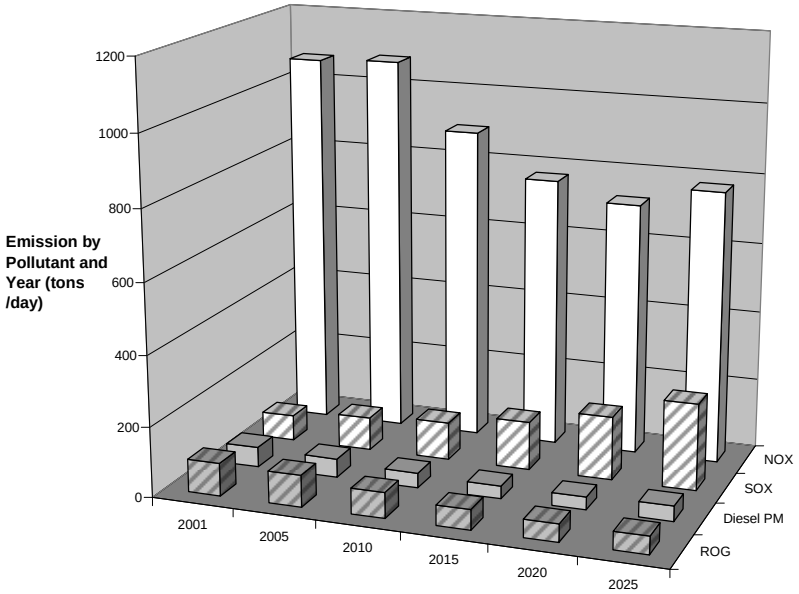
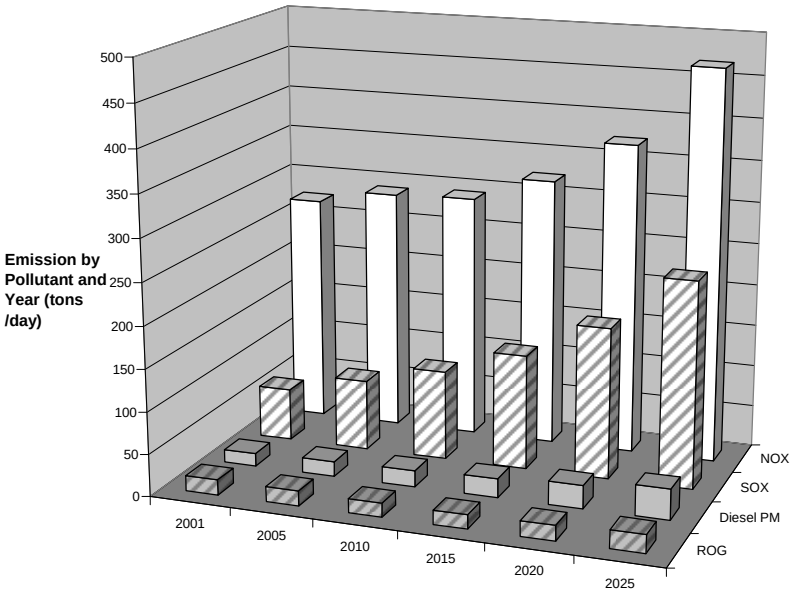


Figure A-2 Port and International Goods Movement Emissions



California has four major goods movement corridors: (1) the South Coast Region, (2) the San Francisco Bay Area Region, (3) the San Diego Region, (4), the San Joaquin Valley Region, and (5) the Sacramento Valley Region. Regions like the South Coast and the San Francisco Bay Area are major centers of goods movement because they contain the largest ports in California. In particular, the South Coast region contains the largest container cargo ports in the U.S. and southern California's economy and transportation infrastructure has developed around these ports. The San Joaquin Valley and Sacramento Valley are major corridors for transport of goods by truck and rail, and also contains the Ports of Stockton and Sacramento. Table A-3 provides 2001 emissions estimates for each of these five regions.

Table A-3 2001 Goods Movement Emissions by Region (tons/day)

Region	ROG	Diesel particulate	NO_X	SO_X
South Coast	23	14	256	23
San Francisco	12	6	120	11
San Diego	5	3	48	5
San Joaquin Valley	18	11	218	2
Sacramento Valley	5	2	51	1

C. Previous Risk Assessments

In October 2005, CARB staff released a draft risk assessment for the Ports of Los Angeles and Long Beach (CARB 2005a). These ports are located adjacent to each other on San Pedro Bay about 20 miles south of downtown Los Angeles. The purpose of the study was to increase understanding of the port-related diesel PM emissions impacts and how emissions from different source types affect cancer risk and other health outcomes. This study focused on the on-port emissions from ships, locomotives, on-road heavy-duty trucks, and cargo handling equipment. Cargo handling equipment is used to move containerized and bulk cargo, and includes forklifts, yard trucks, rubber tire gantry cranes, and many other equipment types.

Diesel PM emissions from the two ports were estimated to be 1,760 tons per year in 2002. This represents about 20% of the total diesel PM emissions in the SoCAB. About 73% of the emissions were related to ship activities in the California Coastal Waters (CCW), which is the region extending 14 to 100 miles offshore, depending on location. Commercial harbor craft vessel emissions were estimated at 14% of the total, followed by cargo handling equipment (10%), in-port heavy duty trucks (2%), and in-port locomotives (1%).

Locomotives are another source of goods movement related pollutants. In October 2004, CARB staff published the Roseville Rail Yard Study; a health risk assessment of particulate emissions from diesel-powered locomotives at the Union Pacific J.R. Davis Yard in Roseville, California. Diesel PM emissions from the rail yard were estimated to be about 25 tons per year, with moving locomotives accounting for about 50% of the emissions total, idling locomotives 45%, and engine testing 5% (CARB 2004).

The Roseville Rail Yard Study and the SoCAB port risk assessment both used an emission inventory and air dispersion modeling program to estimate the ambient concentrations to which nearby residents would be exposed, and both quantified cancer and non-cancer risk related to diesel PM. Risk assessment is a process with four inter-related steps: identifying the hazard, or in this case, the air pollutant of concern; determining how human health would be affected by the pollutant; determining the air pollution concentration to which an average person in the affected area would be exposed; and finally, assessing the rate of increased illness or premature death that would result from the exposure. These types of risk assessments are generally performed to determine the magnitude of health impacts from the sources and guide the design of activities to reduce the health hazard. Risk assessments are used routinely to guide development of regulations that focus on reducing (mitigating) pollutants from the most important sources. In risk assessments performed to help design control measures, the estimate of the inhaled concentration of the pollutant (dose) is multiplied by the OEHHA cancer potency factor (response rate) and multiplied by one million to arrive at the number of additional cancer cases estimated per one million population. In the case of non-cancer health effects, CARB and OEHHA use concentration-response functions derived from published epidemiologic studies to relate the changes in predicted concentrations to various health endpoints, the population affected, and the baseline incidence rates (CARB 1998c, Lloyd and Cackette 2000).

Based on the modeling analysis for the communities surrounding the ports in the SoCAB, potential cancer risk associated with on-port and vessel emissions was estimated to exceed 500 in a million. A 50 per million cancer risk still existed more than 15 miles from the ports. CARB staff's assessment of diesel PM health impacts of the Ports of Los Angeles and Long Beach characterized the increased risk of cancer and non-cancer health effects to nearby neighborhoods. The study determined these non-cancer health effects in the study area in year 2005 as follows: 67 premature

deaths, 41 hospital admissions for respiratory or cardiovascular causes, 2,100 cases of lower respiratory symptoms, 170 cases of acute bronchitis, 12,000 days of work loss, and 71,000 restricted activity days. In the health assessment for this plan, CARB staff updated the analysis of the non-cancer health effects in three ways. First, the impact of the two ports was calculated for the entire area surrounding the ports (40 mile by 50 mile), not the smaller study area near the ports. Second, the updated methodology, using Pope et al. (2002) for calculating premature death associated with particulate pollution was used. Third, the emissions inventory was updated from 2002 to 2005. In the Roseville Rail Yard Study, the risk assessment showed elevated concentrations of diesel PM contributing to cancer risks of 500 per million population on the rail yard property (an area between 10 to 40 acres). Elevated cancer risks between 100 and 500 million cases per million were estimated for the 700 to 1,600 acres surrounding the rail yard where 14,000 to 26,000 people live. And risk levels between 10 and 100 cases per million were estimated for a 46,000 to 56,000 acre area with a population of 140,000 to 155,000.

Movement of goods to and from port facilities, rail yards, distribution centers, and inter-modal transfer facilities will also result in increased exposure to nearby residents. Residents living in near major transportation corridors for goods movement will also experience elevated exposure and health risk in comparison to the average resident in the region. CARB staff have determined that living very near a large distribution center where hundreds of trucks operate could increase the cancer risk by as much as 750 cases per million (CARB 2004). A number of monitoring studies have concluded that PM and other traffic-related exposures are elevated in the vicinity of freeways (Zhu et al. 2002). Recently published epidemiologic studies estimate an increased risk for respiratory symptoms and asthma for those living near roads with heavy traffic (Kim et al. 2004, Gauderman et al. 2005).

The increasing on-road diesel truck traffic from expanding port cargo handling volumes is not only a concern due to its effect on community exposure and ambient air quality, but also adds to in-vehicle exposures. CARB studies indicate that non-smoking Los Angeles residents receive from 30% to 50% of their total diesel PM exposures during their 90 minute-per-day average drive time (Rodes et al. 1998, Fruin et al. 2004a). Some pollutants (e.g., ultrafine particles) show even higher in-vehicle percentages (Fruin et al. 2004b). Analyses of in-vehicle monitoring measurements have found that the high concentrations of black carbon (indicating diesel PM), NO, ultrafine particles, and particle-bound polycyclic aromatic hydrocarbons (PAHs) are primarily driven by diesel truck traffic volumes (Fruin et al. 2005, Westerdahl et al. 2005). Quantifying the increased in-vehicle exposures due to increased goods movement traffic emissions is beyond the scope of this report, but needs to be taken into account before total exposure impacts can be considered fully quantified. Nonetheless, in our exposure estimation for secondary PM, interpolations were first performed at the census tract level, which addresses some of the concerns regarding exposures at a smaller scale. The census-tract interpolated values were then weighted by census populations to arrive at population-weighted exposures for each county or air basin, which is consistent with how concentration-response functions are typically derived in epidemiological studies.

D. Air Pollutants of Concern

The air pollutants of concern related to goods movement are largely those associated with diesel-fueled engines, which cover nearly all of the trucks, locomotives, off-road equipment, and ships that move international goods. Diesel engine emissions are highly complex mixtures consisting of a wide range of organic and inorganic compounds including directly emitted organic (or elemental) and black carbon (EC

and BC), toxic metals, nitrogen oxides (NO_X), particulate matter (PM), volatile organic compounds, gases such as formaldehyde and acrolein, and PAHs. Diesel exhaust includes over 40 substances that are listed as hazardous air pollutants by the U.S. EPA and by the CARB as hazardous air pollutants (HAPs). In 1998, CARB (CARB 1998b, 1998c) identified diesel PM as a toxic air contaminant (TAC). Increases in lung cancer have been identified in most studies of groups occupationally exposed to diesel exhaust. Population-based case control studies identified statistically significant increases in lung cancer risk for truck drivers, railroad workers, heavy equipment operators, and others. On average, these studies found that long-term occupational exposures to diesel exhaust were associated with a 40% increase in the relative risk of lung cancer (OEHHA 1998). These results were largely confirmed in a recent analysis of lung cancer in a cohort of railroad workers (Garschick et al. 2004). Based on these studies and an estimated ambient concentration of diesel PM for which most Californians are exposed (1.54 $\mu g/m^3$), OEHHA estimated a annual range of additional cancer cases of 200 to 3600 for every one million residents over a 70-year lifetime (OEHHA 1998).

In addition to the long term cancer effects of diesel exhaust, short term effects have been observed. There are a number of indications in the occupational epidemiology literature (Delfino et al. 2002) and animal studies that some air toxics are associated with induction and exacerbation of asthma. These include chemicals that are products of fuel combustion, such as formaldehyde and acrolein. It has been shown in numerous studies that diesel exhaust particulate matter can enhance allergic asthma (Nel et al. 1998, Diaz-Sanchez et al. 1999, 2000, Saxon and Diaz-Sanchez 2000). Similar results have been obtained in animal models (Maejima et al. 2001). In addition, immune suppression (Burchiel et al. 2004) has been observed in experimental animals exposed to diesel exhaust resulting in increased susceptibility to respiratory infection (Castranova et al. 2001).

A major pollutant of concern is PM which can be either directly emitted into the atmosphere (primary particles) or formed there by chemical reactions of gases (secondary particles) from natural or man-made sources such as sulfur oxides (SO_X) and NO_X, and certain organic compounds. Ambient ozone pollution is formed from primary emissions of NO_X and other precursor compounds. We've focused primarily on PM and ozone, because these are the two pollutants for which there is sufficient evidence of adverse health effects.

The great majority of epidemiological studies reporting associations between PM and adverse health effects have used as their measure of PM either $PM_{2.5}$ (particles less than 2.5 µm in diameter) or PM_{10} (particles less than 10 µm in diameter). The particles in diesel emissions are very small (90% are less than 1 µm by mass). However, because there are very few studies that used $PM_{1.0}$ as the measure of particulate matter, we've primarily relied upon studies that used ambient $PM_{2.5}$ concentrations as the measure of particulate matter exposure. We did, however, include some studies that used ambient PM_{10} concentrations, because of other advantages these studies offered.

Ozone is regulated in California as a criteria air pollutant. In April of 2005, through collaboration with OEHHA, the CARB approved the nation's most health protective ozone standard with special consideration toward children's health. A new 8-hour-average standard for ozone was established as 0.070 parts per million (ppm), and a 1-hour-average ozone standard was set at 0.09 ppm. Ozone is a powerful oxidant that can damage the respiratory tract, causing inflammation and irritation.

1. Health Effects Associated with PM and/or Ozone

Many studies have investigated the relationship between PM and/or ozone and a variety of adverse health effects. For some health effects, concentration-response functions have been estimated in the epidemiological literature, and the "weight of evidence" argues in favor of their inclusion in a quantitative analysis. For other health effects, there is as yet an insufficient basis for inclusion in a quantitative analysis. The health effects that have been identified to be associated with PM and/or ozone, including those that are included in the quantitative analysis and those that are not, are given in Exhibit 1.

Exhibit 1. Summary of the Health Effects Associated with PM and Ozone

Health Effect	Identified		Included in Quantitative Analysis	
	PM	***Ozone***	***PM***	***Ozone***
Mortality				
All-cause mortality in adults	X	X	X	X
Cardiopulmonary mortality in adults	X	X	*	*
Lung cancer mortality in adults[1]	X	--	*	--
Infant mortality	X	--	†	--
Respiratory Hospital Admissions				
Hospital admissions for all pulmonary illnesses	X	X	X	X
Hospital admissions for chronic obstructive pulmonary disease	X	X	**	**
Hospital admissions for pneumonia	X	X	**	**
Hospital admissions for asthma	X	X	**	**
Cardiovascular Hospital Admissions				
Hospital admissions for all cardiovascular illnesses	X	--	X	--
Emergency Room Visits				
Emergency room visits for asthma	X	X	†	†
Other Morbidity Effects				
Myocardial infarction (heart attack)	X	--	†	--
Chronic bronchitis	X	--	†	--
Acute bronchitis	X	--	X	--
Asthma and lower respiratory symptoms	X	--	X	--
Minor restricted activity days	X	X	X	X
Work loss days	X	--	X	--
School absences	--	X	--	X
Asthma onset	--	X	--	†
Low birth weight, pre-term birth	X	--	†	--
Respiratory Symptoms in Asthmatics				
Exacerbation of asthma	X	X	†	†
Respiratory symptoms (e.g., bronchitis, phlegm, cough)	X	X	X	†
Asthma attacks	X	X	†	†

[1] Lung cancer mortality associated with exposure to ambient PM, and lung cancer risk associated with diesel particulates.

X These endpoints have been identified and, if sufficient data available, were quantified.

***** These endpoints were not included in the quantitative analysis because they are subsets of all-cause mortality, which is included.

****** These endpoints are a subset of all-respiratory hospital admissions.

† These endpoints were not quantified due to insufficient information to perform a quantitative analysis. Please see Appendix A for more detail.

-- These pollutants have not been identified as associated with these health endpoints in this document.

2. Selection Concentration-Response Functions for Quantified Analysis

There are many C-R functions available for estimating the reduced health risks associated with reductions in the levels of ozone and $PM_{2.5}$, as well as a variety of sources of uncertainty surrounding any such risk reduction estimates. When we conduct benefits analyses, we have to decide which health endpoints to include in the analysis and which epidemiological studies (reporting estimated C-R functions for those health endpoints) to use.

In its recent particulate matter risk assessment, U.S. EPA's Office of Air Quality Planning and Standards (OAQPS) included only those health endpoints "for which the overall weight of the evidence from the collective body of studies supports the CD [Criteria Document] conclusion that there is likely to be a causal relationship or that the scientific evidence is sufficiently suggestive of a causal relationship that OAQPS staff judges the effects to be likely causal between PM and the effects category" (Abt Associates Inc., 2005). In addition, EPA considered only those health endpoint categories for which there are C-R functions based on either directly measured $PM_{2.5}$ or $PM_{2.1}$, or concentrations of fine particles estimated using nepholometry data.

U.S. EPA is using this same "weight of the evidence" approach in selecting appropriate health endpoints in its current ozone risk assessment, and we used a similar approach in selecting health endpoints to include in this analysis.

In selecting C-R functions to use from among the many that are available in the epidemiological literature, we were guided by the following considerations:

- The geographic specificity of the study. A common study selection criterion for a benefits analysis that is specific to a given location (e.g., Los Angeles or California) is that the study was conducted at or near that location. The relationship between a pollutant and the population health response to that pollutant is likely to vary to some extent from one location to another, because of (1) differences among populations (for example, if the population in one location has a higher percentage of older and more vulnerable people than in another location) and, (2) for a pollutant such as $PM_{2.5}$, which is itself a mix of other "pollutant species," differences in the pollutant.
- Single-city versus multi-city C-R functions. All else being equal, a C-R function estimated in the assessment location is preferable to a function estimated elsewhere since it avoids uncertainties related to potential differences due to geographic location. There are several advantages, however, to using estimates from multi-city studies versus studies carried out in single cities. Multi-city studies are applicable to a variety of settings, since they estimate a central tendency across multiple locations. When they are estimating a single C-R function based on several cities, multi-city studies also tend to have more statistical power and provide effect estimates with relatively greater precision than single city studies due to larger sample sizes, reducing the uncertainty around the estimated coefficient. In addition, there is less likelihood of publication bias or exclusion of reporting of negative findings or findings that are not statistically significant with multi-city studies. Because single-city and multi-city studies have different advantages, if a single-city C-R function has been estimated in an assessment location and a multi-city study that includes that location is also available for the same health endpoint, one approach is to use the results from both. We have used that approach in this benefits analysis.

- Studies of the relationship between mortality and short-term vs. long-term exposure to $PM_{2.5}$. There is evidence suggesting that there are effects of long-term exposure to $PM_{2.5}$ that are not captured in the short-term studies. Several well-regarded studies of the relationship between mortality and long-term exposure to $PM_{2.5}$ are available, and have been used in recent EPA risk assessments and benefits analyses. Because using both studies of long-term exposure and studies of short-term exposure would result in double counting of mortality impacts, long-term studies are considered preferable to short-term mortality studies.[7]
- The year of publication of the study. If more than one study for a health endpoint is available, more recent studies are preferable to older studies because the statistical techniques for estimating concentration-response functions have become substantially more sophisticated over time. There are several ways in which techniques have improved, among which are improved methods for taking weather variables into account and better specification of lag structures (for example, several of the more recent studies of short-term effects have specified distributed lag models which may be superior to single-lag models). The exact publication date before which to exclude studies from consideration is obviously somewhat arbitrary. We considered 1990 a reasonable choice, however, since some of the more sophisticated techniques were first applied in the 1990s, and many studies were published after that date.
- $PM_{2.5}$ as the measure of particulate matter vs. PM_{10}. While it is still unclear exactly what components of particulate matter have adverse effects on health, most recent research suggests that adverse health effects are most associated with the fine portion of particulate matter, $PM_{2.5}$. In addition, as noted above, 90% of the particles in diesel emissions are less than 1 µm by mass.
- C-R functions estimated using GAMs in the software package S-Plus that have not been re-estimated. Many time-series studies, especially those carried out in recent years, involved use of generalized additive models (GAMs). In late May 2002, EPA was informed by the Health Effects Institute (HEI) of a generally unappreciated aspect in the use of S-Plus statistical software often employed to fit these models. Using appropriate modifications of the default convergence criteria code in the S-Plus software and a correct approach to estimating the variance of estimators will change the estimated C-R functions and could change the results of tests of significance of estimates, although it is not possible to predict a priori how estimates and significance tests will change. Many but not all of the C-R functions that were originally estimated using the S-Plus software for fitting GAMs have since been re-estimated using revised methods. In May 2003, HEI published a special peer-reviewed panel report describing the issues involved and presenting the results of the re-analyzed studies (Health Effects Institute, 2003). In its particulate matter risk assessment, EPA used as one of its selection criteria that a C-R function that had been estimated using GAMs S-Plus and had not been re-estimating using revised methods was excluded from consideration.
- Multi-pollutant C-R functions vs. single-pollutant C-R functions. Some epidemiological studies focusing on a given pollutant estimate C-R functions in

[7] For C-R functions of ozone and mortality, only short-term exposure studies are available.

which only that pollutant is entered into the health effects model (single pollutant models), while other studies include one or more co-pollutants in their models (multi-pollutant models). To the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to the targeted pollutant (i.e., the pollutant of interest) in single pollutant models, risks attributed to that pollutant might be overestimated where C-R functions are based on single pollutant models. On the other hand, inclusion of pollutants that are highly correlated with one another in a multi-pollutant model can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, inclusion of multiple pollutants in models often produces unstable and statistically insignificant effect estimates for the targeted pollutant and the co-pollutants. Neither single-pollutant nor multi-pollutant models is clearly preferable.

There is a stated or implied "all else equal" in most criteria, but in practice all else is often not equal. While any set of C-R function selection criteria can be used as a guide, they generally cannot by themselves determine which C-R functions to select, because the criteria may conflict with each other in the selection process. For example, one C-R function may have been estimated in the assessment location (e.g., Los Angeles) but used PM_{10} as the measure of particulate matter, while another C-R function may have been estimated in a different location but used $PM_{2.5}$ as the measure. By one selection criterion, we would select the first C-R function, but by another we would select the second. We therefore sometimes had to make "judgment calls," in which we weighed the particular strengths of one C-R function against those of another for the same health endpoint. In some cases, we used two different C-R functions for the same health endpoint, each of which offered specific advantages and disadvantages, and presented two alternative sets of results.

In its PM health risk assessment, staff at EPA's Office of Air Quality Planning and Standards (OAQPS) reviewed the evidence evaluated in the 2004 PM Criteria Document (CD) (see Chapter 3 of the 2005 PM Staff Paper) in selecting what it considered appropriate health endpoints to include. Given the large number of endpoints and studies addressing PM effects, OAQPS included in the quantitative PM risk assessment only:

- More severe and better understood (in terms of health consequences) health endpoint categories.
- Health endpoints for which the overall weight of the evidence from the collective body of studies supports the CD conclusion that there is likely to be a causal relationship or that the scientific evidence is sufficiently suggestive of a causal relationship that the effects would be judged to be likely causal between PM and the effects category.
- Health endpoint categories for which there were studies that satisfied their study selection criteria.

For the primary analysis, we used the same broad health endpoint categories for $PM_{2.5}$ that were selected by OAQPS. This includes:

- Non-accidental premature mortality associated with long-term exposures;
- Respiratory hospital admissions associated with short-term exposures;
- Cardiovascular hospital admissions associated with short-term exposures; and
- Respiratory symptoms not requiring hospitalization associated with short-term exposures.

Non-accidental, cardiovascular, and respiratory mortality due to short-term exposure, as well as cardiopulmonary and lung cancer mortality due to long-term exposure were also included in EPA's $PM_{2.5}$ risk assessment, because health risk reductions were not monetized, and so overlapping categories of health effects could be shown separately. For a benefits analysis, however, in which there is a final monetized benefit, this would not be appropriate.

Some health endpoints, such as chronic bronchitis, were *not* included in the EPA's $PM_{2.5}$ risk assessment because it was judged that there is as yet insufficient weight of evidence for them. However, EPA set fairly stringent criteria for inclusion in the risk assessment. For example, the PM Criteria Document notes that there is a reasonably significant relationship between long-term PM exposure and non-mortal respiratory effects.[8] As a result, we included some additional endpoints, such as acute bronchitis, minor restricted activity days (MRADs), and work loss days (WLDs).

In the primary analysis for ozone, we used those health endpoint categories that OAQPS staff selected for the ozone health risk assessment. This includes:

- Premature mortality associated with short-term exposures;
- Respiratory hospital admissions associated with short-term exposures.

In addition, we included two health endpoints, school loss days and MRADs, within the category of "minor effects."

Exhibits 2 and 3 below list the studies that were considered for use in the analysis for PM and ozone, respectively. Most of these studies were either conducted in California or are multi-city studies contained in U.S. EPA's Final Particulate Matter Criteria Document (2004) or its Second External Review Draft of the Ozone Criteria Document (2005). A few additional studies that are not included in the CDs because they were published too late to be included are also included in these Exhibits. Those studies that we used in the primary analysis are noted in the Exhibits.

[8] "For respiratory effects, notable new evidence from epidemiological studies substantiates positive associations between ambient PM concentrations and not only respiratory mortality, but (a) increased respiratory-related hospital admissions, emergency department, and other medical visits; (b) increased incidence of asthma and other respiratory symptoms; and (c) decrements in pulmonary functions" (EPA 2004, p. 9-79).

Exhibit 2. Studies Reviewed for Health Effects Related to Particulate Matter

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
Mortality associated with long-term exposures	Mortality; ages 30+	61 U.S. cities	Pope et al. (2002)		*X*
	Mortality; ages 30+	61 U.S. cities	Pope et al. (1995), reanalyzed by Krewski et al. (2000)		
	Mortality; ages 25+	6 U.S. cities	Dockery et al. (1993)		
	Mortality; ages 25+	6 U.S. cities	Laden et al. (2006), reanalysis of the Six Cities data		
	Mortality; ages 30+	Los Angeles	Jerrett et al. (2005)	*Extremely large effect estimate. Use in sensitivity discussion.*	
	Mortality; ages 65+	California	Enstrom (2005)	*Size of cohort about one tenth that of Pope* et al. *(2002), but all in California*	
	Infant mortality	86 U.S. Cities	Woodruff et al. (1997)		
	Infant mortality	California	Woodruff et al. (2006)		
Respiratory hospital admissions	HA, COPD, age 20-64	Los Angeles	Moolgavkar (2000a)		*X*
	HA, COPD, age 65+	Los Angeles	Moolgavkar (2000a), reanalyzed in Moolgavkar (2003a)		*X*
	HA, COPD; ages 65+	14 U.S. Cities (not including L.A.)	Samet et al. (2000), reanalyzed by Zanobetti and Schwartz (2003)	PM_{10} based.	X
	HA, pneumonia; ages 65+	14 U.S. Cities (not including L.A.)	Samet et al. (2000), reanalyzed by Zanobetti and Schwartz (2003)	PM_{10} based.	X
	HA, pulmonary, ages 30+	Los Angeles	Linn et al. (2000)	PM_{10} based.	
	HA, emergency and urgent asthma-related	Los Angeles	Nauenberg and Basu (1999)	PM_{10} based. Wet season only (Nov. 15 – March 1)	
Cardiovascular hospital admissions	HA, Cardiovascular, age 20-64	*Los Angeles*	Moolgavkar (2000b)		*X*

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
	HA, Cardiovascular, age 65+	*Los Angeles*	Moolgavkar (2000b), reanalyzed in Moolgavkar (2003a)		*X*
	HA, Cardiovascular, age 65+	14 U.S. Cities (not including L.A.)	Samet et al. (2000), reanalyzed by Zanobetti and Schwartz (2003)	PM_{10} based.	X
Emergency room visits for asthma	ER visits for asthma	Santa Clara Co., CA	Lipsett et al. (1997)	Based on winter-time observations with strong contribution from residential wood smoke.	
	ER visits for asthma	Seattle, WA	Norris et al. (1999)		
Other effects	Myocardial infarction		Peters et al. (2001)		
	Chronic bronchitis		Abbey et al. (1995)		
	Acute bronchitis	24 communities in the U.S. and Canada	Dockery et al. (1996)		X
	Lower respiratory symptoms	6 U.S. cities	Schwartz and Neas (2000)		X
	MRADs	Nationwide; workers aged 18-65.	Ostro and Rothschild (1989)	Fine particulate concentrations were estimated by regression from airport visibility data.	X
	WLDs		Ostro (1987)	Fine particulate concentrations were estimated by regression from airport visibility data.	X
Respiratory symptoms among asthmatics	Asthma symptom scores for asthmatics	So. CA community	Delfino et al. (1998b)		
	Exacerbation of asthma in African-American children	Los Angeles	Ostro et al. (2001)	Both PM_{10} and ozone in the model	
	Chronic airway disease determined by pulmonary function tests	CA communities	Berglund et al. (1999)		
	Asthma, bronchitis, cough, wheeze	12 southern CA communities	Peters et al. (1999)	Acid aerosols and NO2 linked to respiratory morbidity in children,	

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
	Bronchitis, phlegm, cough among asthmatic children	12 southern CA communities	McConnell et al. (1999; 2003)	Looked at a variety of measures of particulate matter, including and organic carbon, $PM_{2.5}$, and $PM_{10.}$	
	Asthma attacks	Santa Monica, Anaheim, Glendora, Garden Grove, Thousand Oaks, and Covina, CA	Whittemore and Korn (1980)	This study was published in 1980, and uses data from the early 1970s. It measured TSP instead of PM_{10} or $PM_{2.5}$.	
Birth outcomes	Low birth weight	California	Parker et al. (2005)		
	Preterm births	California	Ritz et al. (2000)		

Exhibit 3. Studies Reviewed for Health Effects Related to Ozone

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
Mortality associated with short-term exposures	Mortality (non-accidental, all ages)	*95 U.S. cities*	Bell et al. (2004)		X
	Mortality (non-accidental, all ages)	*Multiple U.S. cities*	Bell et al. (2005)	Meta-analysis of 39 studies. Found significant impact as well as publication bias.	X
	Mortality (non-accidental, all ages)	*Multiple U.S. cities*	Ito et al. (2005)	Meta-analysis	X
	Mortality (non-accidental, all ages)	*Multiple U.S. cities*	Levy et al. (2005)	Empiric Bayes meta-regression of 28 studies.	X
	Mortality (non-accidental, all ages)	*15 European cities*	Anderson et al. (2004)	World Health Organization	X
	Mortality (non-accidental, all ages)	*Multiple U.S. cities*	Levy et al. (2001)	Meta-analysis.	X
	Mortality (non-accidental, all ages)	*Multiple cities*	Stieb et al. (2002)	Meta-analysis.	X
	Mortality (non-accidental, all ages)	*Multiple cities*	Thurston and Ito (2001)		X
	Mortality (non-accidental, all ages)	*23 European cities*	Gryparis et al. (2004)		X
	Mortality (non-accidental, all ages)	*80 U.S. cities*	Samet et al. (2000), reanalyzed by Dominici et al. (2003)		
	Mortality (non-accidental, all ages)	*14 U.S. cities*	Schwartz et al. (2005)		
	Mortality (non-accidental, all ages)	*Los Angeles Co., CA*	Kinney et al. (1995)		
	Mortality (non-accidental, all ages)	*San Bernardino and Riverside Counties, CA*	Ostro (1995)		
	Mortality (non-accidental, all ages)	*Coachella Valley, CA*	Ostro et al. (2000)		
	Mortality (non-accidental, all ages)	*Santa Clara Co., CA*	Fairley (1999), reanalyzed by Fairley (2003)		

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
	Mortality (non-accidental, all ages)	*Los Angeles Co., CA*	Moolgavkar (2003a)		
Mortality associated with long-term exposures	Mortality	*Three California air basins (San Francisco, South Coast, San Diego)*	Abbey et al. (1999) Beeson et al. (1998)	This study had only 6,338 subjects, all white non-Hispanic non-smoking.	
Respiratory hospital admissions	HA, asthma	CA	Neidell (2004)		
	HA, pulmonary	Los Angeles	Linn et al. (2000)	C-R functions for age < 30 and age 30+ estimated separately. Ozone significant single pollutant model, however, unstable to inclusion of other pollutants, notably CO.	
	HA, asthma	Los Angeles	Nauenberg and Basu (1999)	Ozone not related to asthma admissions.	
	HA, all-respiratory (all ages)	3 previous studies on Canadian cities	Thurston and Ito (1999)	Meta-analysis.	X
	HA, all-respiratory (all ages)	16 Canadian cities	Burnett et al. (1997)	Ozone result significant and stable with inclusion of other pollutants. Soiling index used as a surrogate for particulate matter.	
	HA, all-respiratory	3 cities in New York State	Thurston et al. (1992)		
Effects not requiring hospitalization	School absences	12 southern CA communities	Gilliland et al. (2001)		X
	MRADs	Nationwide; workers aged 18-65.	Ostro and Rothschild (1989)		X
Respiratory symptoms among asthmatics	Morning symptoms in inner city asthmatic children	8 U.S. cities	Mortimer et al. (2002)	The study has a high percent of children from poor households and is thus not a representative sample of all asthmatic children.	
	Exacerbation of asthma in African-American children	Los Angeles	Ostro et al. (2001)	Both PM_{10} and ozone in the model. Ozone not significant.	

Health Endpoint Category	*Health Endpoint*	*Study Location*	**Study**	**Notes**	**Used in Primary Analysis**
	Symptoms interfering with daily activity among Hispanic asthmatic children	Los Angeles	Delfino et al. (2003)		
	Asthma symptoms	Alpine, CA	Delfino et al. (2004)		
	Respiratory symptoms	12 southern CA communities	Peters et al. (1999)		
	Phlegm	12 southern CA communities	McConnell et al. (1999; 2003)		
	Asthma attacks	Santa Monica, Anaheim, Glendora, Garden Grove, Thousand Oaks, and Covina, CA	Whittemore and Korn (1980)	This study was published in 1980, and uses data from the early 1970s. It measured photochemical oxidant (O_x) instead of ozone (O_3).	
Asthma onset (due to long-term exposure)	Asthma onset	California	Greer et al. (1993)		
	Asthma onset	California	McDonnell et al. (1999) (cont'd work of Greer et al. (1993))		
	Asthma onset	12 southern CA communities	McConnell et al. (2002)		
Birth outcomes	Low birth weight	California	Parker et al. (2005)		
	Preterm birth	California	Ritz et al. (2005)		

a) Mortality

There is evidence for independent effects of both PM and ozone on the risk of premature mortality. We discuss each separately.

***PM-related Mortality*.** There is a large literature examining a linkage between particulate matter and premature mortality. A number of recent studies in California (Jerrett et al. 2005; Ostro et al. 2006; Ostro et al. 2003; Fairley, 2003) have reported a significant impact; on the other hand, some (Enstrom, 2005; Moolgavkar, 2003b) have questioned this relationship. Enstrom 2005 found only a small effect on mortality with PM2.5 exposure in the early years of exposure to a cohort of elderly Californians with no effect from more recent exposures. However, this study has generated a great deal of controversy and may have a number of potential uncontrolled confounders including second hand smoke exposure. Nevertheless, the weight of the evidence in the literature points to a significant relationship.

As we discussed above, we gave preference to studies of long-term exposure, rather than short-term exposure to PM. Among the long-term exposure studies, U.S. EPA used a C-R function from Pope et al. (2002). This study extended the follow-up period for the American Cancer Society (ACS) cohort to sixteen years and published findings on the relationship of long-term exposure to $PM_{2.5}$ and all-cause mortality (as well as cardiopulmonary and lung cancer mortality). This 2002 study has a number of advantages over previous analyses, including: doubling the follow-up time and tripling the number of deaths, expanding the ambient air pollution data to include two recent years of $PM_{2.5}$ data, improving the statistical adjustment for occupational exposure, incorporating data on dietary factors believed to be related to mortality, and using more recent developments in nonparametric spatial smoothing and random effects modeling.

Recently, the Health Effects Subcommittee (HES) of the Science Advisory Board's (SAB) Clean Air Act Compliance Council indicated its preference that U.S. EPA use the results from this study rather than the results from the Krewski et al. (2000) ACS and/or Six Cities analyses to represent base case estimates for long-term exposure mortality associated with $PM_{2.5}$ concentrations for the purposes of benefits analyses (Science Advisory Board (SAB), 2004). Two periods of $PM_{2.5}$ measurements were considered in the ACS-extended study. The first, from 1979 through 1983, was the period considered in the original ACS study as well as in the Krewski reanalysis. The second was 1999-2000. The authors also report results based on an average of the two periods. The HES recommended that U.S. EPA use the results based on the average of the two periods from this study as representing the best estimates. The HES stated that this choice "may serve to reduce measurement error" (Science Advisory Board (SAB), 2004). For our benefits analysis, we used the corresponding C-R function based on $PM_{2.5}$ measurements averaging the air quality data from the two periods.

In a sensitivity discussion, we used a recent study by Jerrett et al. (2005) that examined the relationship between air pollution and mortality with small-area exposure measures in Los Angeles. This is a cohort study based on a subset of the American Cancer Society cohort used in the Pope et al. (2002) analysis. Jerrett et al. concluded that measurement error due to estimating exposure for a metropolitan area can lead to a large downward bias in the estimated impact, and that chronic impacts associated with

intra-city gradients appear much larger than previously reported across metropolitan areas. This study also suggests that these effects are closely related to traffic exposure. The authors cite confirmation of the traffic effects in a Dutch study that found a doubling of cardiopulmonary mortality for subjects living near major roads (Hoek et al. 2002). Jerrett et al. estimated a 17% increase in all-cause mortality per 10 ug/m^3 change in $PM_{2.5}$ – nearly three times larger than that seen in Pope et al. (2002). Although both the importance of intra-city gradients and the suggested relation of the effects to traffic exposure have been seen in other studies (Hoek et al. 2002), given the magnitude of the estimate and other possible models presented by Jerrett et al. (with estimated increase ranging from 11% to 17%) we elected to use this study in a sensitivity discussion, until additional work can confirm this effect.

Laden et al. (2006) extends the original Harvard Six Cities study (Dockery et al. 1993). We considered using this study as a supplementary source of a C-R function for mortality and long-term exposure to $PM_{2.5}$, because it focuses on essentially the same geographical area in which we are interested. We chose not to use it, however, for several reasons. First, $PM_{2.5}$ concentrations, while *measured* in the years from 1979 through 1988, were *estimated* in the subsequent years in the study. This introduces additional uncertainty into the resulting C-R function estimates. Second, the number of cities is relatively small, the cities are located outside of California, and the cohort is all white. Third, the reported relative risks were sufficiently high as to give us pause. This was true for the original Harvard Six Cities study and the reanalysis of that study (Krewski et al. 2000) as well. For example, Laden et al. (2006) reports a relative risk for (all cause) mortality of 1.16 – i.e., a 16% increase in mortality – associated with an increase in long-term $PM_{2.5}$ of 10 µg/m^3. The corresponding relative risk from the Krewski reanalysis of the original Harvard Six Cities study was 1.13 – a 13% increase in mortality. Both of these percent increases are over twice the percent increases that would be predicted to be associated with an increase in $PM_{2.5}$ of 10 µg/m^3 by either the reanalysis of the ACS study (Krewski et al. 2000) or the extended ACS study (Pope et al. 2002), which would predict increases of 4.7% and 6%, respectively. Nonetheless, the Laden results are in line with Jerrett et al. (2005).

Chen et al. 2005 found a greater risk of fatal coronary heart disease in females, but not males, exposed to PM2.5, PM coarse and PM10. This study is not representative of all of California since the study subjects were all white non-Hispanic. However, since the subjects are all non-smoking and detailed information was available on environmental tobacco smoke exposure in the cohort, and could be adjusted for, a large potential confounder is accounted for in the study. In addition, the majority of the cohort resides in the large urban centers of California.

Ozone-related Mortality. A number of studies have tested the significance of a relationship between ozone and premature mortality, with a number of these studies conducted in California ((Kinney and Ozkayank, 1991; Kinney et al. 1995; Moolgavkar, 2003b; Fairley, 2003). In addition, there have been a number of studies conducted in other parts of the country, including several meta-analyses (Bell et al. 2005; Ito et al. 2005; Levy et al. 2005)and a multi-city study (Bell et al. 2004)

The evidence from California is somewhat mixed. Moolgavkar (2003b) did not find a significant effect, while Kinney et al. (1995; 1991) reported a significant effect, though

the effect was sensitive to inclusion of PM; Fairley (2003) reported a significant impact even when controlling for fine PM.

The World Health Organization (WHO) conducted a meta-analysis of the 15 cities in Europe (Anderson et al. 2004). Their meta-estimates indicate a relative risk of 1.003 (95% CI = 1.001 – 1.004) for a 10 $\mu g/m^3$ change in 8-hour ozone. For standard pressure (1 atmosphere) and temperature (25º C), 1 ppb ozone equals 1.96 $\mu g/m^3$. We have assumed the ratio between 1-hour and 8-hour ozone of 1.33 and between 1-hour and 24-hour of 2.5 (Schwartz 1997). Making the conversions, the WHO estimate implies a 1.13% change (95% CI = 0.38 - 1.51) in daily mortality per 10 ppb change in 24-hour ozone. The WHO also provided an estimate correcting for possible publication bias using a trim and fill technique. Under an assumption that bias was present, the adjusted estimate is 0.75 % (95% CI = 0.19 – 1.32) per 10-ppb change in 24-hour ozone.

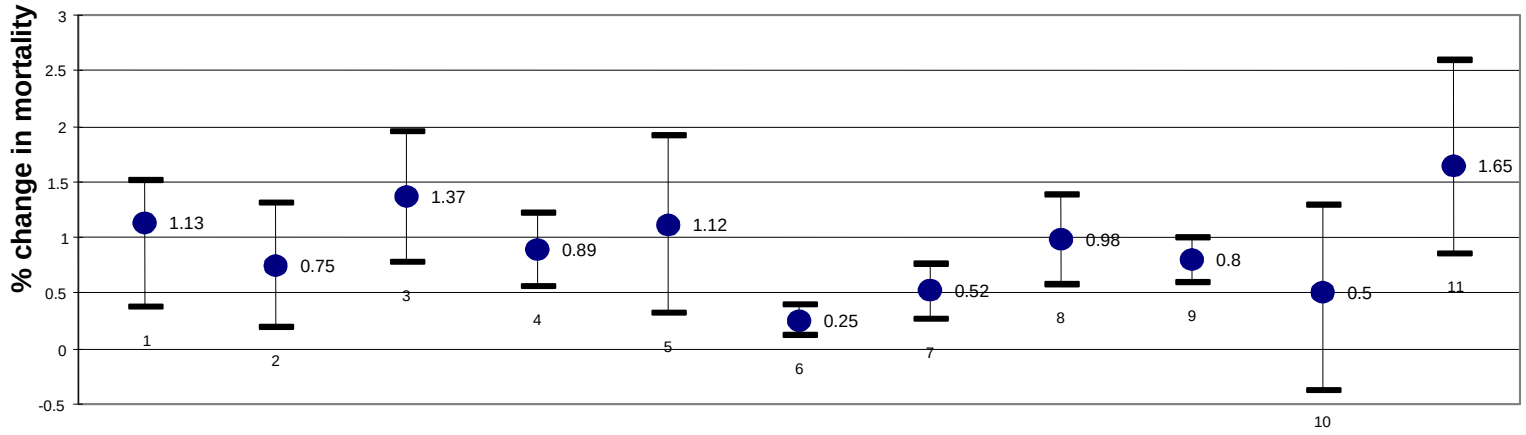
This estimate is very similar to that produced by Levy et al. (2001). In their meta-analysis they began with 50 time-series analyses from 39 published articles. A set of very strict inclusion criteria was applied, which eliminated all but four studies. Reasons for exclusion included: studies outside the U.S., use of linear temperature terms (versus non-linear and better modeled temperature), lack of quantitative estimates, and failure to include particulate matter (PM) in the regression models. Ultimately, their analysis generated an estimate of 0.98% (95% CI = 0.59 – 1.38) per 10 ppb change in 24-hour average ozone. If the criteria are loosened to include eleven more studies, the pooled estimate decreases to 0.80 (0.60 – 1.00). Stieb et al. (2002) also reported a similar effect estimate based on 109 previous studies (including those with single- and multi-pollutant models) of 1.12 (0.32 – 1.92). Thurston and Ito (2001) reviewed studies published prior to the year 2000. When the authors focused on seven studies that more carefully specified the effect of a possible confounder, daily temperature, by using non-linear functional forms, the resulting meta-estimate was 1.37% (95% CI = 0.78 – 1.96). Relaxing this constraint to include all 19 available studies, the resulting risk estimate was 0.89% (95% CI = 0.56 – 1.22) per 10-ppb change in 24-hour ozone.

Two more recent meta-analyses have been published that provide lower effect estimates. Gryparis et al. (2004) is an analysis of 23 European cities from the APEHA2 study. The study controlled for potential confounders by including average daily temperature and humidity, respiratory epidemics, day of week in the regression model. The overall full-year estimate was 0.5% (95% CI = -0.38 – 1.30) per 10-ppb change in 24-hour ozone. A meta-analysis was also conducted using summer-only data. Presumably this estimate will be less confounded by seasonality and also represent a time when the population would be spending more time outdoors. The summer-only estimate was 1.65% (95% CI = 0.85 – 2.60) per 10-ppb change in 24-hour ozone. This summer-specific estimate might be particularly relevant for California due to its milder climate. A meta-analysis of the 95 largest U.S. cities from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data base provided estimates using a similar natural spline model for every city (Bell et al. 2004). Ultimately, the model suggested an effect of 0.25% (95% CI = 0.12 – 0.39) per 10-ppb change in 24-hour ozone. The NMMAPS study may generate an underestimate of the impact of mortality due to the modeling methodology used to control weather factors. Specifically, this effort included four different controls for temperature and dewpoint, where most other times-

series analyses used only two or modeled extreme weather events more carefully and used city-specific models to ensure the best fits. In comparing the results for particulate matter (PM) for a given city with studies of individual cities by other researchers, the NMMAPS results are usually lower (Samet et al. 2000). This estimate was based on a lag consisting of today's and yesterday's ozone concentrations. When a longer period 7-day lag was used the estimate increased to 0.52% (95% CI = 0.27 – 0.77) per 10-ppb change in 24-hour ozone.

Our estimates for the effects of ozone on mortality attempt to reflect the range provided in the above cited studies. Figure A-3 provides a graphical summary of the range of effect estimates and our suggested central, low and high estimates. A low estimate of 0.5% per 10 ppb, 24-hour ozone, corresponds to the best estimates from the NMMAPS (using a one-week cumulative lag) and the APEHA2 European study, but is below most of the other central estimates. A central estimates of 1% per 10 ppb is very similar to the

Figure A-3: Percent Change in Mortality Associated with Ozone (per 10 ppb 24-hour average)



Study #	Author	# of studies	Comment
1	Anderson (2004)	15	European
2	Anderson (2004)	20	Euro, corrected for possible publication bias
3	Thurston+Ito (2001)	7	Studies using non-linear temp
4	Thurston+Ito (2001)	19	All studes
5	Stieb et al. (2003)	109	All studies
6	Bell et. al. (2004)	95	NMMAPS, lag(01)
7	Bell et. al. (2004)	95	NMMAPS,lag(06)
8	Levy et al. (2001)	4	Strict criteria
9	Levy et al. (2001)	15	Less strict criteria
10	Gryparis et al. (2004)	23	all year Europe
11	Gryparis et al. (2004)	23	summer Europe

central estimates generated by Anderson (2004), Levy et al. (2001), and Stieb (2003). Finally, as a high estimate, we use 1.5% per 10 ppb which reflects the central estimates of Thurston and Ito (using non-linear functions for temperature) and the summer-only estimates of Gryparis et al. (2004). Our range of estimates is applied to all age groups. On the 1-hour scale, a 1% change per 10 ppb of 24-hour ozone is about 0.4% per 10 ppb change in 1-hour daily maximum ozone based on an assumed the ratio between 1-hour and 8-hour ozone of 1.33 and between 1-hour and 24-hour of 2.5 (Schwartz 1997).

A more recent study (Bell et al. 2006) explores the evidence for a threshold in the ozone/mortality relationship and concludes "all results indicate that any threshold would exist at very low concentrations, far below current U.S. and international regulations and nearing background levels (Bell et al. 2006). A variety of percent increases in mortality associated with a 10 ppb increase in ozone are reported in this study, depending on the underlying model and air quality dataset being used.

In 2005, U.S. EPA funded three independent groups of researchers to assess the strength of the relationship between short-term exposures to ozone and premature death. These three recent meta-analyses (Bell et al. 2005; Ito et al. 2005; Levy et al. 2005) independently found consistent results on the association, and the results are in fair agreement with our chosen estimates.

To summarize, for ozone-related premature death, we used the following for the central estimate:

- Anderson (2004), Levy et al. (2001), and Stieb (2003)
- Bell et al. (2005); Ito et al. (2005) and Levy et al. (2005)

a) Infant Effects

A number of studies in California have associated air pollution with low birth weight, preterm delivery, and cardiovascular birth defects (Wilhelm and Ritz, 2005; Salam et al. 2005; Parker et al. 2005; Kaiser et al. 2004; Wilhelm and Ritz, 2003; Ritz et al. 2002; Ritz et al. 2000; Woodruff et al. 1997). These results have been replicated in a number of other locations both in the U.S. and around the world (Sagiv et al. 2005; Bobak, 2000; Loomis et al. 1999; Bobak et al. 2001; Ha et al. 2001; Liu et al. 2003; Yang et al. 2003a; Yang et al. 2003b; Gouveia et al. 2004; Maisonet et al. 2001). In addition, a number of studies have linked particulate air pollution to infant mortality (Ha et al. 2003; Kaiser et al. 2004; Loomis et al. 1999; Woodruff et al. 1997; Bobak and Leon, 1999; 1992).

The weight of the evidence points to air pollution, especially particulate matter, as having a significant impact on infants. In particular, we estimate the impact on infant mortality using by Woodruff et al. (1997). However, not all of the available evidence supports this conclusion, notably the work by Lipfert (2000), which examined infant mortality in the United States. As a result, we consider the infant mortality estimate in a sensitivity discussion.

The impact of air pollution on low birth weight was estimated by Parker et al. (2005). This study is California specific and examined an association with PM2.5. Ritz et al. (2000) estimated the impact of PM10 air pollution on preterm birth in southern California. Both of these estimations could not be used in a sensitivity discussion due to the many potential confounders with extrapolating their results to a California-wide estimation, and the uncertainties remaining on the association between these birth outcomes and particulate pollution exposure.

b) Hospital Admissions

For respiratory hospital admissions associated with exposure to particulate matter, we used:

- Linn et al. (2000), hospital admissions for pulmonary illness;
- Samet et al. (2000), reanalyzed by Zanobetti and Schwartz (2003), hospital admissions for COPD and hospital admissions for pneumonia; and
- Moolgavkar (2000a; 2003a).

Moolgavkar (2000a; 2003a), has the advantages of having been conducted in Los Angeles and using $PM_{2.5}$ as the measure of particulate matter, in addition, it includes ages 20 to 64 as well as ages 65 and older. Linn et al. (2000) used PM_{10} as the measure of particulate matter; however it was also conducted in Los Angeles and covers a broader range of respiratory hospital admissions. Samet et al. (2000) also used PM_{10} as the measure of particulate matter, but it has the advantage of being a 14-city study, and thus having substantially more statistical power to detect small effects over a lot of "noise." Because there is substantial overlap in the endpoints of these studies, their results (for ages 65 and older) cannot be summed. As a result, we pooled the Moolgavkar age 65+ estimare for COPD hospital admissions with the Zanobetti & Schwartz age 65+ COPD, added this to the 65+ Zanobetti & Schwartz estimate for pneumonia, and later added the result to the Moolgavkar estimate for COPD hospital admissions applied to age group 18+. This would give one central estimate of age 18+ respiratory hospital admissions. For sensitivity, Linn et al. (2000) for age 30+ could be used. However, due to the limited age range, the estimate would be viewed as an underestimate. Hence, we present the pooled estimate for age 18+ in our primary analysis.

For cardiovascular hospital admissions associated with exposure to particulate matter, we used:

- Moolgavkar (2003a; 2000b), hospital admissions for cardiovascular illness;
- Samet et al. (2000), reanalyzed by Zanobetti and Schwartz (2003), hospital admissions for cardiovascular illness.

Moolgavkar (2003a; 2000b) has the advantages of having been conducted in Los Angeles and of using $PM_{2.5}$ as the measure of particulate matter; in addition, it covered ages 20-64 as well as ages 65 and older. The advantages (and disadvantages) of Samet et al. (2000) are noted above. For ages 65 and older, we pooled the estimates based on Moolgavkar (2003a; 2000b) and Zanobetti and Schwartz (2003), and added this to the estimate for ages 18 to 64 based on Moolgavkar (2003a; 2000b) to arrive at an estimate for age 18+.

Studies of a possible ozone-hospitalization relationship have been conducted for a number of locations in the United States, including California. These studies use a daily time-series design and focus on hospitalizations with a first-listed discharge diagnosis attributed to diseases of the circulatory system (ICD9-CM codes 390-459) or diseases associated with the respiratory system (ICD9-CM codes 460-519). Various age groups are also considered which vary across studies.

For ozone, we included only respiratory hospital admissions, because the evidence for an association between cardiovascular hospital admissions and ozone is weak. For respiratory hospital admissions, the overall weight of the evidence suggests that the effect of ozone on respiratory hospital admissions is robust to the inclusion of particulate matter. To estimate ozone-related hospital admissions, we initially

considered Linn et al. (2000) because it was conducted in Los Angeles; however, they reported only the results of a single-pollutant model and noted that this result was not stable with the inclusion of other pollutants, notably carbon moNOXide. The relatively small sample-size of this study is a concern.

For this estimate, we rely on the meta-analysis by Thurston and Ito (1999). These authors used a random effects model using three studies from North America. The studies were Burnett et al. (1994), Thurston et al. (1994), and Burnett et al. (1997). The category of all respiratory admissions for all ages yielded an estimate of relative risk of 1.18 (95% CI= 1.10 – 1.26) per 100 ppb change in daily 1-hour maximum ozone. This category includes hospital admissions for asthma and bronchitis, so separate estimates of these outcomes are not necessary. The estimate converts to a 1.65% change in hospital admissions (95% CI = 0.95 – 2.31%) per 10 ppb change in 1-hour daily maximum ozone. This estimate was applied to all age groups. Additional studies of respiratory admissions for specific diseases or subpopulations provide additional support for the above relationship, but are not quantified to avoid double counting. For example, Anderson et al. (1997) reported a relative risk of 1.04 (95% CI= 1.02-1.07) for hospital admissions for COPD for all ages for a 50 µ/m change in ozone. This converts to 2.05% per 10 ppb change in 1-hour maximum ozone. Burnett et al. (2001) investigated respiratory hospitalizations in children under age 2, and reported a relative risk of 1.348 (95% CI= 1.193 – 1.523), which converts to a 6.6% increase in hospital admissions per 10 ppb change in 1-hour daily maximum ozone.

To summarize, for respiratory hospital admissions due to ozone, we used:

- Thurston and Ito (1999), hospital admissions for all respiratory symptoms.

c) Emergency Room Visits

A range of studies conducted in the United States have examined the association between air pollution and respiratory and cardiovascular emergency room visits (Peel et al. 2005; Slaughter et al. 2004; Metzger et al. 2004; Jaffe et al. 2003; Tolbert et al. 2000; Fauroux et al. 2000; Norris et al. 1999; Atkinson et al. 1999; Lipsett et al. 1997; Weisel et al. 1995; Schwartz et al. 1993; Cody et al. 1992). And there are a number of studies from Canada, Spain, United Kingdom, and other countries (Pande et al. 2002; Stieb et al. 2000; Tobias et al. 1999; Ilabaca et al. 1999; Tenias et al. 1998; Delfino et al. 1998a; Delfino et al. 1997a; Stieb et al. 1996).

Two studies by Norris et al. (1999) and Lipsett et al. (1997) were initially chosen to estimate the effect of particulate matter on emergency room visits for asthma. The Lipsett et al. study was conducted in California; however, it focused on just the winter season in a region with a lot of residential wood smoke. Moreover, it used PM_{10} as its measure of particulate matter and used interaction terms between PM_{10} and temperature when specifying the model (thus requiring temperature data to properly use the results). For these various reasons, this study was subsequently discarded. Instead, the Norris et al. study was used because it used $PM_{2.5}$ as its measure of pollution and covered the full year. However, we consider this endpoint as a potential endpoint only, since it is single-city study conducted in Seattle, Washington, and thus outside the area of interest.

Regarding ozone, the U.S. EPA (2005) Criteria Document for ozone cited both significant and non-significant results from a range of studies, and then concluded that the evidence is inconclusive regarding an association between ozone and emergency room visits. This conclusion coupled with the lack of studies from California informed the choice not to estimate ozone-related emergency room visits.

d) *Effects not Requiring Hospitalization*

A variety of respiratory symptoms and illnesses not requiring hospitalization were included in the analysis. For particulate matter, the endpoints and the studies reporting C-R functions for those endpoints are as follows:

- Lower respiratory symptoms – Schwartz and Neas (2000);
- Acute bronchitis – Dockery et al. (1996);
- Minor restricted activity days (MRADs) – Ostro and Rothschild (1989);
- Work loss days (WLDs) – Ostro (1987).

For ozone, we used:

- School loss days – Gilliland et al. (2001);
- MRADs – Ostro and Rothschild (1989).

Restricted activity day estimates are derived from a sample of an adult working population by Ostro and Rothschild (1989). This study is the same as that used for estimating this health effect for PM (see above).

School absence estimates are derived from analysis of 1,933 grade school students enrolled in the Children's Health Study (Gilliland *et al*. 2001). Illness-related absences were verified through telephone contact for respiratory-related illness including runny nose or sneeze, sore throat, cough, earache, wheezing, or asthma attack. Associations were observed between 8-hour average ozone and school absenteeism due to these respiratory illnesses. The results from this study were applied to all school-aged children.

e) Asthma-Related Effects

Particulate matter has been more closely associated with asthma-related effects, such as wheeze, cough, and other symptoms. Children appear to be particularly at risk. Ostro et al. (2001) could be used to estimate asthma-related effects (wheeze, cough, shortness of breath) and McConnell et al. (1999) to estimate acute bronchitis and chronic phlegm among asthmatic children. However, because lower respiratory symptoms (including asthma-related symptoms), acute bronchitis, and school loss days are already being estimated, there are concerns of double-counting effects in children. As a result, the asthma exacerbations are not treated separately.

Regarding ozone, the evidence suggests that asthmatic children may be at risk, though the evidence is somewhat mixed. An 8-city study by Mortimer et al. (2002) reported a significant effect for ozone on morning asthmatic symptoms in a single-pollutant model; however, the confidence bounds for this result increased with the inclusion of other pollutants and often left the estimate statistically insignificant. Studies conducted in California are mixed. In an analysis in 12 Southern California communities, McConnell et al. (1999; 2003) reported little effect for ozone on asthmatic symptoms, though they reported that children playing sports may be more likely to develop asthma (McConnell et al. 2002). Ostro et al. (2001) reported no association found between ozone and new episodes of cough or wheeze, but found some evidence that ozone is associated related asthma medication use. Similarly, Delfino et al. (2002; 2004; 1996; 1997b) have reported some significant associations between ozone and asthma; however, the results are not definitive. As a result, we have not estimated asthma-related effects associated with ozone.

The health endpoints and studies that were selected from among those considered are summarized in Exhibits 4 and 5 for PM and ozone. Endpoints and/or studies that are used only in a sensitivity discussion are shown in italics.

Exhibit 4. $PM_{2.5}$ Concentration-Response Functions

Endpoint	Location	Age	Author	Notes
Mortality, All Cause	51 U.S. cities	30+	Pope et al. (2002)	
	Los Angeles	*30+*	*Jerrett* et al. *(2005)*	*Sensitivity discussion (very large effect coefficient)*
	86 U.S. cities	*<1*	*Woodruff* et al. *(1997)*	*Sensitivity discussion*
	California	*<1*	*Woodruff* et al. *(2006)*	*Sensitivity discussion*
Hospital Admissions, All Cardiovascular	14 U.S. cities	65+	Zanobetti and Schwartz (2003)	The two 65+ estimates are pooled using fixed/random effects approach. Result summed with Moolgavkar estimate for ages 18-64.
	Los Angeles, CA	65+	Moolgavkar (2003a)	
	Los Angeles, CA	18-64	Moolgavkar (2000b)	
Hospital Admissions, Chronic Lung Disease	14 U.S. cities	65+	Zanobetti and Schwartz (2003)	Moolgavkar 65+ COPD with the Zanobetti & Schwartz 65+ COPD, add this to the 65+ Zanobetti & Schwartz Pneumonia are pooled. Result added to the 18-64 Moolgavkar COPD estimate.
Hospital Admissions, Pneumonia	14 U.S. cities	65+	Zanobetti and Schwartz (2003)	
Hospital Admissions, Chronic Lung Disease	Los Angeles, CA	18-64	Moolgavkar (2000a)	
Hospital Admissions, Chronic Lung Disease	Los Angeles, CA	65+	Moolgavkar (2003a)	
Hospital Admissions, All Respiratory	*Los Angeles, CA*	*30+*	*Linn* et al. (2000)	*Sensitivity discussion*
Lower Respiratory Symptoms (including asthma related effects)	6 U.S. cities	7-14	Schwartz and Neas (2000)	
Acute Bronchitis	24 communities	8-12	Dockery et al. (1996)	
Minor Restricted Activity Days	Nationwide	18-64	Ostro and Rothschild (1989)	
Work Loss Days	Nationwide	18-64	Ostro (1987)	
Asthma Exacerbation, Cough	*Los Angeles, CA*	*8-13*	*Ostro* et al. *(2001)*	*Sensitivity discussion (potential overlap with other endpoints, such as lower respiratory symptoms. Asthma exacerbation estimates presented separately.)*
Asthma Exacerbation, Shortness of Breath	*Los Angeles, CA*	*8-13*	*Ostro* et al. *(2001)*	
Asthma Exacerbation, Wheeze	*Los Angeles, CA*	*8-13*	*Ostro* et al. *(2001)*	
Acute Bronchitis, among asthmatics	*Southern California*	*9-15*	*McConnell* et al. *(1999)*	
Chronic Phlegm, among asthmatics	*Southern California*	*9-15*	*McConnell* et al. *(1999)*	

Exhibit 5. Ozone Concentration-Response Functions

Endpoint	Location	Age	Author	Notes
Mortality, Non-Accidental	95 U.S. cities	All ages	Bell et al. (2004)	
	15 European cities	All ages	Anderson et al. 2004	
	Multiple U.S. cities	All ages	Levy et al. (2001)	
	Multiple cities	All ages	Stieb et al. (2002)	Used in combination to develop low, central and high estimate for coefficient expressing the strength of association.
	Multiple cities	All ages	Thurston and Ito (2001)	
	23 European cities	All ages	Gryparis et al. (2004)	
	Multiple U.S. cities	All ages	Bell et al. (2005)	
	Multiple U.S. cities	All ages	Ito et al. (2005)	
	Multiple U.S. cities	All ages	Levy et al. (2005)	
Hospital Admissions, All Respiratory	Toronto, Canada	All ages	Thurston and Ito (1999)	
School Loss Days, All Cause	Southern California	6-18	Gilliland et al. (2001)	
Minor Restricted Activity Days	Nationwide	18-64	Ostro and Rothschild (1989)	

3. Unquantified Adverse Effects

As shown in Exhibit 1, there are a number of adverse health effects that have been associated with PM and/or ozone that were not included in the quantified benefits analysis. In some cases, health endpoints were excluded because they are subsets of a larger health endpoint category that is included. Cardiopulmonary mortality and lung cancer mortality were excluded, for example, because they are subsets of all-cause mortality. To include them would have resulted in double counting of benefits.

In some cases, while there is quantitative evidence of a relationship between an adverse health effect and one or both of the pollutants of concern, that evidence comes from one or more single-city studies, none of which were in California. For example, several single-city studies (Weisel, 2002; Tolbert et al. 2000; Cody et al. 1992) found a significant relationship between ozone and ER visits for asthma. However, none of these studies was in California. Moreover, the incidence of ER visits is believed to be particularly variable across locations; this argued against applying one of the statistically significant C-R functions from another location to locations within California.

For some health endpoints, although there is substantial evidence of a relationship between one of the pollutants and the health effect, there are no epidemiologically estimated concentration-response functions available.

We recognize a multitude of endpoints that may contribute to impacting health. However, the weight of evidence to date was deemed insufficient to warrant quantification in our report. These include but are not limited to: chronic bronchitis, onset of asthma, low birth weight, preterm birth, reduced lung function growth in children, psychosocial factors (stress), noise (including cardiovascular effects), light and its effects on sleep, major occupational issues including workplace exposures and injuries, traffic accidents and associated morbidity/mortality, other transportation related issues, and environmental consequences, quality of life, morbidity over extended periods of time, neurological disease, and developmental effects.

Finally, there are other adverse health effects that overlap with endpoints already included in our quantified analysis. They include myocardial infarction (heart attack) and asthma attacks.

4. Community Health Impacts

Vulnerable populations of individuals shown to be particularly susceptible to air pollution-related disease and people living in communities with high pollution burdens are two groups that are of particular concern when assessing the impacts of goods movement-related emissions. Sensitive groups, including children and infants, the elderly, and people with heart or lung disease, can be at increased risk of experiencing harmful effects from exposure to air pollution. People living in communities close to the source of goods movement-related emissions, such as ports, rail yards and inter-modal transfer facilities are likely to suffer greater health impacts and these impacts will likely add to an existing health burden.

Air pollution has been directly associated with low birth weight, preterm delivery, and cardiovascular birth defects (Maisonet et al. 2001, Ritz *et al*. 2000, Ritz et al. 2002, Ha et al. 2001, Gilboa et al. 2005, Wilhelm and Ritz 2003, 2005). Preterm delivery and low

birth weight are risk factors for infant mortality and life-long disability. Also, a number of studies have linked particulate air pollution to infant mortality (Woodruff et al. 1997, Ha et al. 2003, Bobak and Leon 1999) from respiratory causes. There is not enough information at this time to identify the levels of exposure that pose a significant risk of these adverse effects.

The health impacts of air pollution on children are of particular concern. Studies have shown associations between traffic-related pollution and effects in children, including chronic bronchitis symptoms, wheeze, cough, allergic rhinitis, asthma induction, and upper and lower respiratory tract infections (Jaakkola *et al*. 1991, Osterlee *et al*. 1996, Wjst *et al*. 1993, van Vliet *et al*. 1997, Venn et al. 2001, Kim *et al*. 2004). Recent evidence (Gauderman et al. 2004, Kunzli et al. 2004) indicates that air pollution exposure can impair lung function growth in children. The long-term consequences of lower lung function can include shorter lifespan, as lung function peaks in young adulthood and declines thereafter; lung function is the most significant predictor of mortality in the elderly (Schuneman *et al*. 2000, Hole *et al*. 1996).

For those with underlying heart disease or diabetes, increased exposure to air pollutants can compound the effects and increase the rate of adverse events. In one study, individuals with existing cardiac disease were found to be in a potentially life-threatening situation when exposed to high-levels of ultrafine air pollution (Peters *et al*. 2001). Fine particles can penetrate the lungs and may cause the heart to beat irregularly or can cause inflammation, which could lead to a heart attack. Fine particulate matter exposure in vehicles was associate with changes in heart rhythm and blood inflammatory and clotting factors in young health males (Riediker 2004). For persons with a tendency toward hyperlipidemia or diabetes, PM exposure has been found to increase their risk of underlying CVD (Kunzli *et al*. 2005). Understanding the relationships between existing disease and increased exposure is extremely important in quantifying the detrimental health effects of air pollution.

Communities surrounding many goods movement-related facilities where there may be a disproportionate exposure to air pollutants are often economically disadvantaged or ethnically or culturally diverse. People in these communities often have poor access to health care or carry a disease burden that may make them more susceptible to excess exposure. Their housing characteristics may contribute to this susceptibility. Many new areas of research are attempting to understand just how pollutant burdens, low educational attainment, poverty and access to health care, and other factors are interrelated and how these relationships might lead to increased health effects.

Several mortality studies have examined whether socioeconomic status (SES) and related factors such as education and race/ethnicity affect the magnitude of PM-mortality associations. These studies help address the question of whether factors linked with poverty or educational attainment render individuals more susceptible to the adverse effects of exposure to air pollution. To date, the findings have been mixed. The prospective cohort studies investigating the potential impacts of longer-term exposure appear to find consistent effect modification by education, whereas the acute exposure studies do not demonstrate much, if any, modification of these relationships. In their re-examination of the ACS data set originally analyzed by Pope *et al*. (1995), Krewski *et al*. (2000) conducted an exhaustive set of sensitivity analyses. They considered a wide

range of alternative specifications; their findings largely corroborated those of the original study, however, the relative risk estimates varied significantly when the analysis was stratified by educational attainment.

Zanobetti and Schwartz (2000) tested for effect modification by income or education in four large cities with daily PM10 data during the study period of 1986 to 1993 (Chicago, Detroit, Minneapolis-St. Paul, Pittsburgh). They used individual-level educational status from the death records of the National Center for Health Statistics. In three of the four cities, the PM10 effect for the cohort members with less than 12 years of education was larger than that for those with more than 12 years of education. In two of the cities, the PM effect for those in the low-education group was more than twice the other cohort. In contrast, in a study of air pollution and mortality in 10 U.S. cities, Schwartz (2000) examined whether the city-specific mortality effect was modified by several city-wide factors. No effect modification of the pollution effect was found from unemployment, living in poverty, college degree or the proportion of the population that is nonwhite, although sample size limited the ability for detection.

Some evidence exists that living near a major roadway with simultaneous exposure to traffic-related air pollution shortens life expectancy (Finkelstein et al. 2004, Hoek *et al*. 2002). A recent study (Lipfert 2006) found an association between traffic density and mortality. The investigators feel that the results of this study indicate that environmental factors other that traffic emissions, such as traffic noise, stress and socioeconomic factors that are linked to increased traffic may be having an impact as well. One study showed that myocardial infarction is triggered following short-term exposure to elevated traffic pollution in cars, public transit, or on motorcycles or bikes (Peters *et al*. 2004). Risk assessments that utilize air dispersion models to estimate “average” concentrations in a specific area may underestimate risk if that area is surrounded by major roadways. The short-term cardiovascular effects associated with traffic density are not yet quantifiable.

Cumulative impacts are very likely to be experience by communities living in close proximity to goods movement-related activity. Airborne pollutants can deposit onto surfaces and waterways, providing another source of exposure. For example, goods movement activities contribute to non-point source runoff that contaminates coastal and bay waters with a number of toxicants, including PAHs, dioxins, and metals. Exposures to pollutants that were originally emitted into the air can also occur as a result of dermal contact, ingestion of contaminated produce, and ingestion of fish that have taken up contaminants from water bodies. These exposures can all contribute to an individual’s health risk. In some cases, the risks from these kinds of exposure can be greater than the risks from inhalation of the airborne chemicals. An assessment of cumulative impacts is beyond the scope of this analysis.

In most risk assessments, chemicals are evaluated without consideration of other pollutants that may add to the risks posed by the chemicals being assessed. The typical risk assessment does consider cumulative impacts on a specific organ of the body for multiple chemicals that originate from a single source. However, there generally are no methods at present for evaluating cumulative impacts posed by exposures to multiple pollutants. For these reasons, it is often not possible to fully evaluate the health risks in a community that is impacted by multiple sources of pollution.

III. Methodology

A. Air Pollutant Emissions from Goods Movement-Related Sources

Below we describe the methodologies used to develop emissions estimates for each source category - the ships, trucks, trains, cargo handling equipment and harbor craft – associated with goods movement. In each case we built upon and refined estimates for these source categories that historically have been included in the statewide emissions inventory as either a discrete and independent category (i.e., ships and harbor craft) or combined in a more generalized category (i.e., on-road trucks) in the statewide emissions inventory. In the development of the goods movement emission inventory we took steps to ensure the inventory reflected the most up-to-date information on emission rates, activity patterns, expected growth rates and current control measures. In the following sections we provide a brief overview of how these inventories were calculated. Additional details are also provided in the Emission Inventory Technical Supplement.

1. Ocean-going Ships

Ocean-going ships can be classified into many different categories, including container ships that move goods in containers, tankers that move liquids like oil, bulk material transports, and others. Some vessel types, like container ships, directly move imported goods into the State. Other vessel types, like passenger ships, are not engaged in goods movement, but do contribute emissions to the overall port-wide total. All types of ocean-going vessels are included in this analysis, out to 24 nautical miles from shore.

The ocean-going ship category is defined by size; the category includes all ships exceeding 400 feet in length or 10,000 gross tons in weight. These ships are typically powered by diesel and residual oil fueled marine engines. Ocean-going ships have two types of engines. The main engine is a very large engine used mainly to propel the vessel at sea. Auxiliary engines are engines that in general provide power for uses other than propulsion, such as electrical power for ship navigation and crew support. Passenger vessels use diesel electric engines, where a diesel or residual oil fueled engine act as a power plant, providing power both for propulsion and general ship operations. For this reason, CARB considers engines on passenger vessels to be part of the auxiliary engine category.

ARB staff recently developed an improved emissions inventory that accounts for emissions based on a variety of factors including type of vessel, transit locations, various ship engine sizes and loads, and other factors. This inventory covers three modes of ship operation: in-transit emissions generated as a ship travels at cruising speeds, generally in between ports of call; maneuvering emissions generated as a ship slows down in anticipation of arriving, moving within or departing a port; and hoteling emissions generated by auxiliary engines as a ship is docked at port. This inventory was incorporated into the draft plan. Since that time we have further refined the ocean-going ship inventory. Specifically, the emission factor associated with maneuvering was adjusted for low-load conditions, and emissions generated by boilers operating on ships and barges were added to the inventory.

Emissions are calculated on a statewide basis for each port in California. Emissions are also calculated for hoteling and maneuvering operating modes that occur within ports

and transit emissions as ships move up and down the California coastline. Emissions calculated within 24 nautical miles of the shore are included in this emissions inventory. For emissions inventory tracking purposes, emissions are allocated to a port when they occur within three miles of shore. Emissions outside of three miles are allocated to the outer continental shelf air basin.

Estimating growth of ocean-going vessel emissions is a important issue. For this inventory, CARB staff worked with experts at the University of Delaware to compile data on the number and size of main engines visiting each port in California over time. These data account for any increase in the number of ships visiting each port over time as well as the increasing size of these ships. Using data collected representing the years 1997-2003, we developed growth rate estimates for each port. For emissions at the Ports of Los Angeles and Long Beach, we used the growth rates developed for the Port of Los Angeles' No Net Increase Report,[1] which agree with CARB growth projections based on main engine size. As a result, growth rate estimates for 2025 used in this plan are consistent with the No Net Increase report. Our estimates for container growth at the Port of Oakland were also consistent with previous estimates.[2]

Table A-4-a presents statewide emissions by pollutant and ship type from 2001-2020. Container ships are the dominant ship type, although major growth is also forecast for passenger ships, which has a significant on emissions in the San Diego air basin. Table A-4-b presents those same emissions by mode: hoteling, maneuvering, and transit.

Table A-4-a
Statewide Ship Emissions to 24 Miles from Shore by Ship Type*
(tons per day)

	NO_X				Diesel PM				SO_X			
Ship Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Container Ship	59	102	127	156	4.8	8.7	11.0	13.9	37	66	84	106
Tanker	10	15	18	22	0.8	1.3	1.6	1.9	6	10	12	15
Passenger Ship	7	18	29	48	0.7	1.8	2.9	4.9	5	14	23	39
Other Cargo Ships	18	22	25	28	1.5	1.9	2.2	2.6	11	15	17	21
Sum	**95**	**158**	**200**	**254**	**7.8**	**13.8**	**17.8**	**23.4**	**60**	**106**	**137**	**180**

** Includes benefits of regulations passed through October 2005; does not include Auxiliary Engine regulation.*

[1] Report to Mayor Hahn and Councilwoman Hahn by the No Net Increase Task Force: June 24, 2005. Available at: http://www.portoflosangeles.org/DOC/NNI_Final_Report.pdf

[2] Metropolitan Transportation Commission, Regional Goods Movement Study for the San Francisco Bay Area: Final Summary Report. Available at: http://www.mtc.ca.gov/pdf/rgm.pdf

Table A-4-b
Statewide Ship Emissions to 24 Miles from Shore by Operating Mode
(tons per day)

	NO_X				Diesel PM				SO_X			
Operating Mode	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Hoteling	15	33	40	49	1.3	3.0	3.7	4.6	10	25	31	38
Maneuvering	2	5	7	8	0.2	0.4	0.5	0.6	1	3	4	5
Transit	77	120	153	197	6.4	10.5	13.6	18.2	48	79	103	137
Sum	**95**	**158**	**200**	**254**	**7.8**	**13.8**	**17.8**	**23.4**	**60**	**106**	**137**	**180**

** Includes benefits of regulations passed through October 2005; does not include Auxiliary Engine ATCM*

2. Commercial Harbor Craft

Harbor craft are commercial boats that operate generally within or near harbors, or are smaller vessels that support a commercial or public purpose. The harbor craft category includes many types of vessels including crew and supply vessels, pilot vessels, tug and workboats, fishing vessels and ferries. This category does not include recreational vessels used for private use.

ARB staff recently developed an improved statewide emissions inventory for the harbor craft category. This emissions inventory was developed using the statewide population of harbor craft, in conjunction with information about the size and activity of propulsion engines by vessel type obtained by survey to estimate emissions. Harbor craft have both propulsion and auxiliary engines; both are generally powered by diesel fuel. For most commercial harbor craft, the propulsion engines are the primary engines and move the vessel through the water. The auxiliary engines generally provide power to the vessels electrical systems and can also provide additional power to unique, essential vessel equipment (e.g., refrigeration units) during the normal day-to-day operation of the vessel.

Growth in harbor craft emissions was assessed by vessel category. Growth in tug boat emissions were assumed proportional to growth in the number of visits to each port by ocean-going ships in each year, which is not projected to increase with time. No growth was assumed in other harbor craft ship types unless location specific information was provided by local authorities.

For the goods movement inventory, we are using the statewide inventory for harbor craft. However, since the release of the draft plan we have refined our estimates. Specifically, to be consistent with the ocean-going ship inventory, only emissions released within 24 nautical miles of shore are now included in the goods movement inventory. In addition, emission factors were updated to account for fleet turnover, current engine standards, and the increase in emission factors with engine age. The combined effect of these assumptions is to reduce future year emissions. Table A-5-a provides emissions by harbor craft type by pollutant for 2001-2020.

Table A-5-a
Statewide Harbor Craft Emissions to 24 Miles from Shore by Ship Type
(tons per day)

	NO_X				Diesel PM			
Ship Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Fishing Vessels	19	14	11	10	1.0	0.8	0.5	0.5
Tug Boats	15	11	8	7	0.8	0.6	0.4	0.4
Ferry/Excursion	35	26	20	18	1.6	1.3	0.9	0.8
All Others	6	5	4	4	0.3	0.3	0.2	0.2
Sum	**75**	**56**	**44**	**39**	**3.8**	**2.9**	**2.1**	**1.8**

3. Cargo Handling Equipment

The cargo handling equipment category includes many different types of off-road vehicles that are used to move goods through California's ports and intermodal facilities. CARB staff recently developed a new statewide emissions inventory representing cargo handling equipment that estimates the emissions from cranes, forklifts, container handling equipment such as yard hostlers, top picks and side picks, bulk handling equipment such as excavators, tractors, and loaders used at ports and intermodal rail yards.

The goods movement inventory provides emissions by equipment type and for each port and major intermodal facility in California. The inventory reflects updated population and activity data for cargo handling equipment statewide by equipment type based on a survey conducted by CARB in early 2004 and recent emission inventories prepared for the ports of Los Angeles and Long Beach. Growth rates were developed by equipment type from survey responses. The cargo handling equipment inventory in the draft plan has not changed. Table A-5-b presents cargo handling equipment emissions estimated for 2001 and 2025 by pollutant and equipment type.

Table A-5-b
2001 Statewide Cargo Handling Equipment Emissions (tons per day)

	NO_X				Diesel PM			
Equipment Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Yard Tractor	15	10	7	3	0.6	0.3	0.2	0.1
Material Handling Equip	3	3	3	2	0.1	0.1	0.1	0.1
Crane	2	2	2	1	0.1	0.1	0.05	0.04
All Others	1	1	1	0	0.1	0.03	0.02	0.01
Sum	**21**	**16**	**11**	**6**	**0.8**	**0.5**	**0.4**	**0.2**

** Includes benefits of regulations passed through October 2005; it does not reflect the Cargo Handling Equipment regulation adopted by the CARB in December 2005.*

4. Trucks

Trucks are an integral and important component of California's goods movement transportation system. Nearly all goods moved through California are moved by a truck at some time during their transport. Emissions released by trucks are a substantial component of statewide, regional, and goods movement emissions inventories.

The calculation of emissions from trucks is not a simple process. Estimating emissions requires some knowledge about population / engine characteristics, travel activity, and emission factors for individual types of trucks. Engine characteristics include engine model year, manufacturer and technologies. Travel activity includes not just an assessment of the number of trucks and the distance each truck travels in an area, but also the distribution of speeds at which trucks travel and the number of miles the average truck travels per year. Both fleet characteristics and travel activity are typically provided by local and state governments to CARB.

Emission factors relate a given activity level to emissions of each pollutant. These data are obtained by conducting controlled tests of many individual vehicles and then analyzing resulting data to extract average emission factors and trends for different types and ages of engines. Emission factors also include estimates of how emissions change at different speeds, and how emissions increase as engines in trucks become older. All of this information is integrated across a predicted fleet of trucks in a region to calculate emissions. CARB's motor vehicle emissions model, EMFAC, incorporates these factors for the calculation of vehicle emissions.

Truck emissions estimates have changed substantially since the draft goods movement plan was released in December, due to a number of different changes. Most significantly, the inclusion of domestic goods movement has led to a major increase in emissions for the category. Two additional changes led to major changes in the inventory.

- **This plan includes new information regarding motor vehicle emissions.**

ARB staff is currently in the process of developing a new version of EMFAC. This model has not yet been completed, but staff has developed draft emissions calculation methods that include new information about engine populations and characteristics; travel activity; and emission factors. To ensure truck emissions estimates are as accurate as possible in this plan, staff included the new data and assumptions into the goods movement truck emissions inventory. Incorporating new data and assumptions increased emissions estimates and changed the statewide spatial allocation of truck emissions. The current version of the EMFAC model allocates heavy duty truck emissions spatially based upon where vehicles are registered. For this plan, staff allocated emissions based on where trucks are expected to travel. This change results in travel decreases in areas like South Coast and the Bay Area where most trucks in California are registered, and travel increases in areas like the San Joaquin Valley and Mojave Desert where trucks tend to travel on longer routes. Second, truck emission factors in the current version of EMFAC are based upon an extremely limited set of data representing tested trucks. Over the past several years CARB and other organizations have funded new studies to test emissions from trucks. These data, representing chassis dynamometer tests on more than 30 trucks, were integrated into truck

emissions estimates for this plan. Generally truck emission factors for NO_X and diesel exhaust particulate matter increased substantially, leading to a significant increase in emissions relative to the current EMFAC model.

- **This plan includes significant revisions to methods for estimating truck emissions associated with international goods movement.**

The EMFAC model provides emission estimates by vehicle class and by county. It does not provide emission estimates for a specific industry or sector of the economy, such as goods movement. As a result, estimating emissions associated with international goods movement required the development of new methods. The goal of these new methods was to estimate the VMT associated with trucks that haul international goods. The fraction of total truck VMT attributable to international goods movement in a region is then multiplied by emissions in that region to estimate international goods movement emissions. This section describes the development of those methods, which have changed significantly since the release of the draft plan. Our new method is based on the concept of balancing the number of inbound containers to California, outbound containers from California, and empty containers moved out of California. Our assumption is that the number of containers should be balanced; and the flow of containers on ships needs to be consistent with the number of containers moved by trucks and trains.

To illustrate this assumption, it is useful to consider how international goods move in California. Imported goods enter California through the Ports of Los Angeles, Long Beach, Oakland, and others. These goods arrive on ocean-going ships, much of which are packaged in containers. Once at port containers are removed from the ship and staged for land-side transportation. Containers may be moved directly on to a train without the assistance of a truck. This is referred to as “on-dock” rail. Containers may also be moved by a truck to a rail yard, such as the Intermodal Container Transfer Facility in Long Beach, only a few miles away from the port. This is referred to as “near-dock” rail. Containers may also be moved by a truck to a more distant rail yard, such as the Hobart yard in Los Angeles. This is referred to as “off-dock” rail. Rail transportation is most cost-effective over long distances and most containers loaded on to rail at California’s ports are moved out of California.

Other containers are moved by truck directly to their destination, which is most often a distribution center. When trucks carry containers to a distribution center, several things may happen. In many cases the container contents are distributed to smaller trucks for local delivery. Emissions associated with these local deliveries are not included in this plan. In other cases a container may be picked up by a long-haul trucking firm and the container may be moved out of state. In some cases the container is transloaded. Transloading is the practice of repacking generally 40 foot containers into 53 foot containers. Since the cost to move a container is about the same regardless of container size it is more cost effective to move larger containers by truck of rail than smaller containers. Over longer distances transloading can be a cost-effective and efficient method to transport goods.

Our container balancing method was first applied to the South Coast region. Staff collected data from the Ports and local government agencies in the South Coast region. Based on these data staff developed an estimate of the number of containers moving

into the region's ports, and projected these numbers into the future. The total number of containers in each year was then allocated to different travel modes. Table A-5-c presents our estimate of the number of containers in 2001 and 2025 moved by each travel mode. The data indicate more than 50% of containers passing through the Ports of Los Angeles and Long Beach travel by rail.

Table A-5-c
Container Balance by Travel Mode: South Coast
(number of containers)

Mode		Containers		
		Year 2000	**Year 2010**	**Year 2025**
Rail	On-Dock	933,476	2,624,477	3,118,943
Truck	Near-Dock (ICTF)	375,899	1,286,991	1,976,471
	Off-Dock (Hobart)	658,070	1,164,786	2,513,832
	Transload	1,568,539	2,018,570	5,947,318
	Local	1,730,801	2,227,388	6,562,559
Total		5,266,785	9,322,212	20,119,123

About 10,000 trucks are estimated to service the Ports by moving containers on short routes to and from rail yards and distribution centers. These trucks, called Port Trucks in this plan, are generally older than other truck fleets in the South Coast region[1]. Because trucks emit more as they get older, the port truck fleet is dirtier than the regional average fleet.

To estimate port truck emissions in South Coast, staff estimated an average distance traveled per container for each travel mode. The number of containers was then multiplied by the average distance traveled by truck in each mode to calculate VMT. Staff calculated a ratio of port truck VMT to total VMT in South Coast, and adjusted this ratio to account for the higher emission rate of port trucks based on model year distribution. This ratio was then multiplied by truck total truck emissions in South Coast to estimate emissions generated by port trucks.

A fraction of goods transported to distribution centers, primarily transloaded containers, are moved by truck through and potentially out of California to other regional destinations such as Oregon, Utah, Nevada, and other states. Using technical reports generated by local transit agencies in the Los Angeles region, we estimated an additional amount of heavy-duty truck miles traveled in each air basin in California as a result of these secondary transload trips. We adjusted the ratio of transload VMT to air basin total VMT to account for the fact that trucks pulling transloads likely involve national fleets that are much cleaner than the air basin average. This adjusted ratio was also multiplied by emissions in each air basin to calculate emissions associated with transloaded containers originating from the Ports of Los Angeles and Long Beach.

[1] Port of Los Angeles (2004). Port of Los Angeles Baseline Air Emissions Inventory – 2001. Available at: http://www.portoflosangeles.org/DOC/POLA_Final_BAEI.pdf

To estimate the fraction of port truck and transload truck emissions associated with other ports in California we applied the method used for South Coast to the Bay Area. Port trucks servicing the Port of Oakland were assumed to travel in the Bay Area and San Joaquin Valley, and transload VMT generated for containers originating in Oakland was estimated in each air basin of California. For ports outside of the Bay Area, we scaled port truck VMT by the total non-petroleum related tonnage throughput at each port. Only Oakland and the San Pedro Bay ports were assumed to generate transload long-haul truck trips.

Table A-6-a presents domestic truck, port truck, and transload truck emissions projected on a statewide basis between 2001 and 2020. International emissions decreased from the draft plan because we used the container balance method. We believe current emissions more accurately reflect international goods movement, and projections in the draft plan were over-estimated. One might expect port truck emissions to increase with projected to container growth, but as Table A-6-a shows it does not. Container growth is accounted for in the calculation; however existing controls on the truck fleet are projected to reduce emissions more quickly than container growth would increase emissions. Overall, the inclusion of all goods has led to a dramatic increase in total diesel PM and NO_X emissions attributable to goods movement from the draft plan. NO_X emissions are five times higher, and diesel PM estimates ten times higher than estimates in the previous draft plan.

Table A-6-a
Statewide Truck Emissions (tons per day)

	NO_X				Diesel PM			
Truck Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Domestic Trucks	623	492	336	234	36.0	18.5	10.4	5.7
Port Trucks	19	20	21	18	1.4	0.7	0.6	0.4
International Long Haul Trucks	13	5	3	3	0.3	0.2	0.1	0.1
Sum	**655**	**517**	**359**	**255**	**37.7**	**19.4**	**11.1**	**6.22**

Emissions in the South Coast and Bay Area reflect container balancing, as shown in Tables A-6-a and A-6-c. Table A-6-d provides results for the San Joaquin Valley. While the San Joaquin Valley has significant transload traffic, these trucks are relatively cleaner than domestic truck fleets that are likely to be generally older and dirtier.

Table A-6-b
Truck Emissions in the South Coast Region (tons per day)

	NO_X				Diesel PM			
Truck Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Domestic Trucks	120	104	68	44	7.0	4.0	2.2	1.1
Port Trucks	16	17	17	15	1.2	0.6	0.5	0.4
International Long Haul Trucks	4	2	1	1	0.1	0.0	0.0	0.0
Sum	**140**	**122**	**87**	**60**	**8.2**	**4.6**	**2.7**	**1.50**

Table A-6-c
Truck Emissions in the Bay Area (tons per day)

	NO_X				Diesel PM			
Truck Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Domestic Trucks	49	37	23	16	2.4	1.2	0.6	0.3
Port Trucks	3	3	3	2	0.2	0.1	0.1	0.1
International Long Haul Trucks	1	0	0	0	0.0	0.0	0.0	0.0
Sum	**52**	**40**	**26**	**18**	**2.5**	**1.3**	**0.7**	**0.39**

Table A-6-d
Truck Emissions in the San Joaquin Valley (tons per day)

	NO_X				Diesel PM			
Truck Type	**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Domestic Trucks	179	133	92	64	9.9	4.7	2.7	1.5
Port Trucks	0	0	0	0	0.0	0.0	0.0	0.0
International Long Haul Trucks	4	2	1	1	0.1	0.0	0.0	0.0
Sum	**183**	**135**	**93**	**65**	**10.0**	**4.8**	**2.7**	**1.49**

5. Locomotives

Trains, and the diesel-fueled locomotives that power them, travel throughout California. The vast majority of trains in California move freight; a fraction of this freight is imported into and through California from overseas, while the balance represents freight generated in California that is bound for export, and freight generated and consumed within California.

ARB's inventory of emissions from locomotives was first developed in 1987 and has been updated periodically since that time. The inventory accounts for generalized locomotive activity patterns over broad geographical regions. The inventory covers two types of train locomotives. Line-haul locomotives are larger, more modern locomotives that are used to move trains over long distances. Switchers are smaller, older locomotives used to transport trains within a rail yard or over short distances. Line-haul locomotives operate in rail yards as they travel through to their final destination.

To estimate both domestic and international locomotive emissions generated in California CARB staff updated the statewide locomotive inventory. The statewide inventory accounts for several types of line haul trains, all of which are pulled by the same fleet of locomotives. These types include intermodal trains that haul containers; mixed trains that haul bulk materials and other goods such as wood products, agricultural products and petroleum products; and local trains that operate on privately owned local runs. This inventory also includes passenger trains. To update the inventory we reassessed the fraction of intermodal trains operating in each air basin. We then estimated the fraction of international intermodal trains operating in each air basin based on rail yard specific data provided to CARB by class I rail companies. We then reassessed growth to be consistent with expected growth in the number of

containers that will be moving through each air basin in California. These estimates were calibrated using the container balancing method developed for trucks, as discussed above. Switching associated with international intermodal trains was considered international; all other switching emissions were considered domestic. Table A-6-e presents international line haul, international switching, domestic line haul, and domestic switching by pollutant for the years 2001 and 2025.

Table A-6-e
Projected Locomotive Emissions: Baseline Projections 2001-2020
(tons per day)

		Diesel PM				NO_X			
Train Type		**2001**	**2010**	**2015**	**2020**	**2001**	**2010**	**2015**	**2020**
Line Haul									
	International	1.2	1.4	1.6	1.8	49	34	42	51
	Domestic	3.3	2.7	2.6	2.5	144	76	81	82
Switching									
	International	0.04	0.03	0.03	0.03	2	1	1	1
	Domestic	0.2	0.1	0.1	0.1	9	6	5	5
Sum		**4.7**	**4.3**	**4.3**	**4.4**	**203**	**116**	**128**	**139**

B. Adjustment Factor for Ship Emissions

Diesel PM emissions released off-shore do not result in nearly as much population exposure as occurs when the emissions are released on-shore within populated regions. There are two reasons for this. First, diesel PM emissions released off-shore are diluted before they reach shore. As a result, there is no near-source population exposure where pollutant levels are highest. Second, a portion of off-shore diesel PM emissions never reaches the shore, depending on wind direction and over-water deposition rates.

To account for the differing impact of diesel PM emission from off-shore sources, CARB staff developed a South Coast and a statewide diesel PM emissions impact adjustment factor. For the South Coast, the adjustment factor for ship diesel PM emissions release off-shore was estimated to be 0.1, based on dispersion modeling. That is, 100 tons per year of emissions from ships released off-shore would have the same populated-weighted diesel PM concentration (and health impacts) as 10 tons per year of diesel PM emissions released in residential areas near the ports. For the rest of the state, the adjustment factor was estimated to be 0.25.

In calculating the impact of off-shore emissions, the mass of directly emitted diesel PM associated from ships operating off-shore was multiplied by 0.1 for the South Coast Air Basin and by 0.25 for the rest of the State. The resulting emissions were then assigned to the appropriate coastal county. No adjustment was made for secondary PM formation from NO_X, SO_X, and ROG emissions, since these pollutants require at least several hours to form particle nitrate, particle sulfate, and secondary organic aerosol. For the

same reason, offshore sources of NO_X and VOC that contribute to ozone formation were also not adjusted. This latter assumption probably overestimates the impact of offshore emissions, as there will be some losses due to offshore winds and over-water deposition; however, there is the possibility that this could be offset by enhanced chemical conversion rates due to the chlorine radicals (from sea salt spray) and the humid conditions encountered over the ocean. These issues are being studied as part of the technical analysis for a possible North American SO_X Emissions Control Area, described in Section V-C.

The 0.1 adjustment factor for the South Coast Air Basin was derived from dispersion modeling results for the Ports of Los Angels and Long Beach (CARB 2005a) and from modeling results for off-port truck and rail activity that was conducted as part of this report.

Diesel PM emissions from transiting and maneuvering ships associated with the Ports of Los Angels and Long Beach were estimated to be 942 tons per year. Modeling analysis results estimated the annual average population-weighted diesel PM concentration within the study area (20 miles by 20 miles) from these emissions to be 0.11 microgram per meter cubed ($\mu g/m^3$). This would result in an annual average population-weighted diesel PM concentration of 0.0117 $\mu g/m^3$ per 100 tons per year emissions from ships operating offshore. Since this concentration only represents the emissions impact within the study domain, the value was adjusted to account for the impact of ship emissions that extend beyond the study area. As discussed elsewhere in this report, CARB staff estimated that about 40% of the impact from ships operating offshore were outside the study area. Taking this into consideration, the population-weighted diesel PM concentration was adjusted to 0.0164 $\mu g/m^3$ per 100 tons per year emissions from ships operating offshore.

The population-weighted diesel PM concentrations from truck and rail activity within the study area, but off the port property, were estimated to be 0.18 $\mu g/m^3$ resulting from 114 tons per year of diesel PM emissions. Normalized to 100 tons of emissions, the annual average population-weighted concentration would be 0.158 $\mu g/m^3$ per 100 tons of diesel PM emissions off the port property. Comparing the ratios of the population-weighted concentration per 100 tons of diesel PM emissions from offshore ships to off-port truck and rail source (0.0164/0.158), results in a value of about 0.1. This is the value used to adjust the impact of ship emission released offshore the South Coast Air Basin.

The adjustment factor selected for the remainder of the State was 0.25. There is insufficient information to develop adjustment factors for other areas using the same approach as used for the South Coast Air Basin. Given the resulting uncertainty, a more conservative (health protective) adjustment factor of 0.25 was selected for use until additional analyses can be performed. For the San Francisco Bay, it seems reasonable to use a greater adjustment factor than used for the South Coast Air Basin because once a ship enters the Bay the emissions are likely to impact urbanized area regardless of the wind direction. CARB staff will continue work to refine these estimates using region-specific models.

C. Exposure Estimates

1. Diesel PM

In 1998 CARB identified diesel particulate exhaust as a toxic air contaminant (CARB 1998). As part of the identification process, 3 staff estimated the ambient PM10 concentrations of diesel exhaust throughout California. In this estimation, CARB staff used receptor modeling techniques, which includes chemical mass balance model results from several studies, ambient 1990 PM10 monitoring network data, and 1990 PM10 emissions inventory data. The staff used the 1990 PM10 inventory and monitoring data because it would best represent the emission sources in the years when the ambient data were collected for the studies used to estimate 1990 diesel exhaust PM10 outdoor concentrations. The staff has also estimated outdoor exposure concentrations for 1995 and 2000 based on linear extrapolations from the base year 1990 to the respective emissions inventories (CARB 1998).

2. Particle Nitrate and Particle Sulfate

This section provides information on the population-weighted exposure calculation of annual geometric means for particle nitrate and particle sulfate to which people in different parts of California are potentially exposed. The term “potentially” is used because daily activity patterns influence a person’s exposure. For example, being inside a building will decrease a person’s exposure to outdoor nitrate and sulfate concentrations in their vicinity. However, any person who is outdoors will be exposed to a variable concentration. Furthermore, the exposures presented here provide an integrated regional perspective rather than an indication of exposure at any individual location. This exposure analysis is based solely on “outdoor” nitrate and sulfate data, as measured by the Statewide Routine Monitoring Network and additional special monitoring networks IMPROVE and Children’s Health Study.

a) PM Data Description

Airborne particulate matter (PM) is not a single pollutant, but rather a mixture of primary and secondary particles. Particles vary widely in size, shape, and chemical composition, and may contain inorganic ions, metallic compounds, elemental carbon (EC), organic carbon (OC), and compounds from the earth’s crust. A large variety of emission source types, both natural and man-made, are responsible for atmospheric levels of PM. These emission sources directly emit PM (“primary” particles), which then, over time, become coated with the low-vapor-pressure products of atmospheric chemical reactions (“secondary” particles) involving ozone and other oxidants, oxides of sulfur (SO_X), oxides of nitrogen (NO_X), ammonia (NH_3), and volatile organic compounds (VOC). In California, the proximity of a location to a variety of sources, in addition to the diurnal and seasonal variations in meteorological conditions, causes the size, composition, and concentration of particulate matter to vary in space and time.

In urban areas of California, nitrate represents a larger fraction of PM mass compared to the rest of the nation due to wide use of low-sulfur fuels for mobile and stationary sources. The formation of secondary ammonium nitrate (NH_4NO_3) begins with the oxidation of oxides of nitrogen (NO_X) into nitric acid (HNO_3). The nitric acid then reacts with gaseous ammonia to form NH_4NO_3 . The sea influences the chemical composition

of aerosols in the coastal zone. Sodium chloride (NaCl) is always present in aerosols in the form of large particles originating from seawater. Several studies have indicated the importance of HNO_3 reaction on the sea salt particles, leading to thermally stable sodium nitrate ($NaNO_3$) production in the particle phase accompanied by liberation of gaseous hydrochloric acid (HCl) from the particles. This reaction may be the principal source of coarse (2.5 to 10 µm) nitrate, and plays an important role in atmospheric chemistry because it is a permanent sink for gas-phase nitrogen oxide species.

Sulfur dioxide emissions result almost exclusively from the combustion of sulfur-containing fuels. Other sulfur compounds, such as sulfur trioxide (SO_3), sulfuric acid (H_2SO_4), and sulfate, may also be directly emitted during combustion of sulfur-containing fuels, although usually only in small amounts. In the atmosphere, sulfur dioxide is chemically transformed to sulfuric acid, which can be partially or completely neutralized by ammonia and other alkaline substances in the air to form sulfate salts. Sulfate concentrations in the SoCAB are much greater than other areas. However, nationwide, large reductions in ambient SO_2 concentrations have resulted in reductions in sulfate formation that would have been manifest in PM2.5 concentrations on the regional scale.

b) Nitrate and Sulfate Population-weighted Exposures

This analysis is based on the Inverse Distance Weighting method from the Geostatistical Analyst 9.0 software. For this discussion, the nitrate and sulfate annual geometic mean values and population counts were associated by census tract group block and merged to assemble a distribution of exposures across a range of concentrations. Concentrations of many air pollutants, including nitrate and sulfate, may change substantially from place to place. Accordingly, population exposure estimates tend to be more accurate when the population data and air quality data on which they are based are highly resolved, geographically. Population counts by census tract group block provide a convenient source of highly resolved population data. A typical census tract group block contains several thousand people. As a result, densely populated areas have many census tract group blocks, while sparsely populated areas have very few.

The interpolated nitrate and sulfate concentrations from the Statewide Routine Monitoring Network plus the special monitoring networks, IMPROVE and Children's Health Study, were assigned to a census tract group block. The interpolation was a weighted-average of the concentrations measured at the monitors. The weight assigned to each monitor was a function of its distance from the point in space within the state, using an inverse distance weighting function (1/distance to a power). In this way, close monitors are more influential than are distant monitors to the point. Using a weighting factor of 1/distance squared is a common practice. So it was used by staff in this assessment. In addition for the weighting factor, a minimum of 3 monitoring stations were used even if those sites were beyond the search radius of 50 kilometers. Up to a total of 15 could be used within the radius. Geographical barriers such as mountain ranges that may impede the movement of emissions and pollutants were not considered in the exposure calculations, but this omission had little impact on the results since monitors typically collect data in populated areas on both sides of such barriers.

c) Nitrate and Sulfate Monitored Data

The PM nitrate and sulfate data used for the interpolated exposure have been derived from a variety of routine and special monitoring programs and databases. 1998 provide the best data availability with maximum spatial resolution for both routine monitoring network and special study PM network, so this study used mean annual sulfate and nitrate concentrations based on the 1998 data. The PM data that were used in this study generally met U.S. EPA's minimum data completeness criterion, i.e., 11 of 15 samples per calendar quarter. Three different data sets for 1998 were used to provide the ambient nitrate and sulfate concentrations.

- PM10 nitrate and sulfate data from Size Selective Inlet (SSI) monitors. In 1998 the SSI sampling network consisted of 91 sites, however the data completeness criterion reduced the number of sites used in this analysis to 60. Compositional analysis in a laboratory provides the mass of certain ions, including nitrate and sulfate, present in the SSI samples.
- PM2.5 sulfate and nitrate data from Two-Week Samplers (TWS) used in the Children's Health Study. The TWS network was deployed to provide information for an on-going study of the chronic respiratory effects in children from long-term exposure to air pollution in southern California. Because estimates of long-term average concentrations (seasonal and annual) of vapor-phase acids and PM2.5 mass and inorganic ions were needed, it was decided that two-week integrated sampling would be more appropriate than every 6th day sampling.
- PM2.5 nitrate and sulfate data from the Interagency Monitoring of Protected Visual Environments (IMPROVE) program. The IMPROVE program monitoring sites are located in federally protected Class 1 areas and are outside of urban areas. Data from 11 California sites are used in this study.

The concentrations used are a mixture of both PM10 and PM2.5. For annual averages, we believe that mixing PM2.5 and PM10 sulfate and nitrate data is reasonable because most sulfate and nitrate occur in the PM2.5 fraction. To confirm this, we have estimated ratio of PM10 sulfate to PM2.5 sulfate using PTEP data at six monitoring sites in southern California. In general, the annual mean fine PM-sulfate fraction at these sites ranges between 0.8 to 0.9. A similar relationship between PM10 nitrate and PM2.5 nitrate has also been observed at several heavily populated urban locations in California.

Two additional set of data provided information used in estimating background sulfate concentrations. They were:

- The dichotomous (dichot) sulfur data. Dichot sampler uses a low-volume PM10 inlet followed by a virtual impactor which separates the particles into the PM2.5 and PM10-2.5 fractions. The sum of PM2.5 and PM10-2.5 provides a measure of PM10. With the routine monitoring program, samples of PM10 are collected over a 24-hour period using a PM10-SSI) sampler and Dichot sampler. Samples are usually collected from midnight to midnight every sixth day.
- PM2.5 and PM10 sulfate data from the PM Technical Enhancement Program (PTEP 1995). A one-year PM10 Technical Enhancement Program (PTEP)

monitoring was conducted at six sites: downtown Los Angeles, Anaheim, Diamond Bar, Rubidoux, Fontana, and San Nicolas Island. At each location, the sampling equipment was deployed to collect fine and coarse particulate fractions for speciation.

Since the annual California ambient air quality standard for PM is based on the geometric mean (useful for characterizing lognormal data), the geometric means of SSI-PM10 nitrate and sulfate and IMPROVE nitrate and sulfate mass concentrations were calculated for this study. However, the annual arithmetic mean was calculated for the PM2.5 sulfate and nitrate data from Two-Week Samplers. Because the two-week sampler provides an integrated two-week average measurement at each air monitoring station.

Since nitrate and sulfate measurements represent only the mass of the anion, the concentration data need to be adjusted to represent the total mass of the collected particulate molecules, i.e., including the anion, cations, and associated water. The ammonium cation (NH_4^+) is expected to be the major cation for nitrate and sulfate ions in California. There is considerable uncertainty regarding the amount of water associated with ammonium nitrate and ammonium sulfate, but ambient conditions are relatively dry in California for most of the year. In this data analysis, the mass associated with dry ammoniated nitrate and sulfate (i.e., zero molecule of water per XNO_3 or XSO_4 molecule) can be estimated by multiplying the nitrate values by the ratio of the molecular weight of ammonium nitrate to the molecular weight of nitrate, a factor of 1.29, and multiplying the sulfate values by a factor of 1.38.

d) Background Estimation for Particle Sulfate

At the time of release of the December 2005 draft, this report did not specifically address population exposure due to secondary sulfate due to goods movement emissions. Analyses of ambient air quality data conducted in the intervening period now permit an estimate of sulfate effects (see Section A of the Technical Supplement).

Stringent regulations on the sulfur content of fuels have minimized sulfur emissions from most California sources, but despite low sulfur content, the large volume of motor fuel used in California still results in significant statewide SO_X emissions, of which goods movement sources such as locomotives, trucks, etc. are a significant fraction. The largest uncontrolled fossil fuel sulfur source in California is the burning of residual oil as fuel in ocean-going vessels.

Sulfate analysis is complicated by the fact that, in addition to sulfate formed from fossil fuel use in California, there are three other sources of atmospheric sulfate in California – natural “background” sulfate formed over the ocean, global “background” sulfate that is distributed throughout the Northern Hemisphere by the upper air westerly winds, and sulfate blown into Southern California from combustion in Mexico.

Estimating the public exposure to goods movement sulfate is a step-wise process. First, measured ambient sulfate levels must be partitioned among three general source categories (natural, transported, and local), and the “local” fraction must be further subdivided between goods movement sulfate and that from all other emissions. Next, population-weighted exposure due to goods movement sulfate is computed by

overlaying the geographic distributions of goods movement sulfate and population. Finally, health effects are computed by applying appropriate risk factors to the population exposure data.

Natural sulfate concentrations from the ocean were estimated from a review of open ocean measurements and California-specific shore-line and offshore island monitoring data. Sulfate carried by the sea breeze will be reduced by deposition and diluted by dispersion as the air moves inland. Concentrations inland from the shoreline were estimated from the residuals of regressions between fossil fuel emissions and measured sulfate over the period 1985-2000, and found to agree with expected fall-off going inland.

Particle sulfate in the upper air from sources throughout the Northern Hemisphere have been detected at multiple mountain locations in North America, and California-specific data are available from studies in northern California. Since this sulfate is widely distributed over the mid-latitudes, a single upper air “background” level was assigned to all high altitude sites.

Annual average “local” source sulfate at most California monitoring sites was estimated by subtracting site-specific estimates of oceanic and Northern Hemisphere sulfate from the observed values. In extreme southern California (San Diego and Salton Sea Air Basins), where transport from Mexico adds significantly to the measured sulfate, additional adjustments were made based on regression analyses and comparison of ambient sulfate concentrations with analogous population centers farther north.

Population-weighted sulfate exposure was computed by estimating local sulfate concentrations at the census block level using spatial interpolation of the monitoring data. Finally, aggregated Air Basin health effects were estimated from the population-exposure data and the fraction of those effects due to GM emissions determined based on local emission inventories.

New analyses of air quality and emissions data conducted since December 2005 indicate that uncontrolled SO_X emissions from ships increase the estimates of total goods movement-related health effects by about one quarter. However, this preliminary estimate contains several uncertainties and a fully quantitative analysis must await the completion (by end of 2006) of research being jointly conducted by CARB staff, five university groups, the U.S. EPA, and Environment Canada as part of a feasibility study for establishing a SO_X Emission Control Area (SECA) to reduce sulfur emissions from West Coast shipping. The research includes a refined inventory of ship activity and ship emissions, analysis of historical PM data from sites along the West Coast to look for evidence of ship emissions, development of new monitoring methods that can distinguish fossil fuel sulfate from that due to biologic activity in the ocean, and model development to allow simulation of sulfate formation and transport over the ocean and land areas of coastal California.

e) Uncertainties

Secondary nitrate and sulfate particle formation are influenced by a combination of precursor pollutant concentrations and weather conditions. Conversion of SO_X to sulfate aerosols is accelerated by the presence of oxidants in the air (as during ozone

episodes) and is accelerated even more under humid conditions when the conversion can occur inside water droplets. NO_X conversion to nitrate is even more sensitive to weather conditions, as formation rates must compete with dissociation back to gases, so that nitrate is generally a cool-wet (e.g., winter) weather phenomenon. Due to the influences of these factors, the same emissions can result in high PM concentrations on one occasion, and low concentrations on another.

There is uncertainty in these estimates of the secondary fraction of PM2.5 mass. For example, limited ambient speciated data in many areas, particularly rural areas, and forced us to rely on a very limited data in the same region of the air basin. Additionally, these estimates do not account for the volatilization of NO_3 from the particulate filters during sampling and before analysis. Volatilization could be as high as 50%. Overall, it seems that our relatively simple method provides reasonable estimates of the contribution of secondary PM in most of the heavily populated air basins.

To partially assess the uncertainty associated with the interpolation methods, we compared the actual measurements and the interpolated values at the monitoring stations. The mean-squared errors were 0.28 $\mu g/m^3$ and 0.08 $\mu g/m^3$ for nitrate and sulfate calculations, respectively.

3. Secondary Organic Aerosols

Atmospheric particulate carbon consists of both elemental carbon (EC) and organic carbon (OC). Elemental carbon has a chemical structure similar to impure graphite and is emitted directly by sources. Organic carbon can either be emitted directly by sources (primary OC) or can be the result of the condensation of gas-phase oxidation products of volatile organic compounds (VOCs) in the air, here after is referred to secondary organic aerosol (SOA). The initial PM analysis for goods movement only addressed primary carbonaceous material. To complete the assessment of goods movement, PM effects on the contribution to SOA must also be obtained.

Routine OC measurements do not distinguish the primary and secondary components of OC. Even detailed laboratory molecular analyses of organic species in PM can not differentiate properly all of the primary and secondary organic aerosols.

Because direct chemical determination of SOA requires more detailed analysis than is available in routine PM data, the ratio of OC to EC can be used to estimate the amount of SOA in a given sample [Strader at al 1999; Turpin and Huntzicker (1991) Turpin and Lim (2001)]. If an OC/EC ratio that is both characteristic of primary emissions and relatively constant within the period of interest can be determined, then additional OC that drives the ambient ratio above this base level can be assumed to be secondary.

The OC/EC method was used to determine the contribution of SOA at PM monitoring sites in California in 2000. Using this ratio, the contribution of SOA at about 50 sites in California range from 0.15 $\mu g/m^3$ to 2.40 $\mu g/m^3$. Population-weighted SOA exposure was computed by estimating local SOA concentrations at the census block level using spatial interpolation of the monitoring data, based on a methodology similar to that used for particle nitrate and particle sulfate. Finally, aggregated air basin health effects were estimated from the population-exposure data and the fraction of those effects due to goods movement emissions determined based on local emission inventories. The

effects of the uncontrolled ship emissions on port-area air quality show up in these calculations: roughly less than 1% of the health effects due to goods movement (i.e., shipping and port operations) are due to SOA.

4. Ozone

For ozone, California has a monitoring network of approximately 175 monitors located throughout the State. In our ozone staff report (CARB/OEHHA 2005b), hourly observations were input into the estimation of the health impacts of ozone exposures above the standard. Several scenarios of characterizing the ozone exposures were considered: averaging monitored values across each county, assigning portions of populations to monitored concentrations within each county, and interpolating exposures for each census tract. All three options led to very similar results.

D. Health Impacts Methodology

A number of adverse health impacts have been associated with the increase in pollutant emissions associated with goods movement-related emissions. For many of these impacts there is insufficient scientific information to estimate the number of new cases that could result from increased ambient concentrations of the respective pollutant. For this analysis, staff used the same basic methodology and peer-reviewed epidemiologic studies discussed in the Particulate Matter and Ozone Standards reviews (CARB/OEHHA 2002, 2005b) to determine concentration-response functions for several health endpoints, with one exception. An updated study on PM mortality effects was substituted to determine premature deaths associated with diesel PM.

The following goods movement-related health impacts were quantified in this analysis:

Particulate Matter

- Premature deaths
- Hospital admissions for respiratory diseases
- Hospital admissions for cardiovascular diseases
- Acute bronchitis
- Asthma and other lower respiratory symptoms
- Work Loss Days
- Minor Restricted Activity Days

Ozone

- Premature deaths
- Hospital admissions for respiratory diseases
- Minor Restricted Activity Days
- School Absence Days

In a sensitivity discussion, we address premature deaths and respiratory hospital admissions using other studies, infant mortality, and other potential health endpoints.

Concentration-response functions are equations using coefficients derived from epidemiologic studies that relate the change in the number of adverse health effect incidences in a population to a change in pollutant concentration experienced by that population. Due to the form of the models used in many epidemiologic studies, a logarithmic function is usually needed to characterize the non-linear relationship between changes in pollution concentration and occurrences of adverse health outcomes as follows:

$$\Delta y = y_0 \left(e^{-\beta \Delta conc} - 1\right) \times pop$$

where:

Δy= changes in the number of occurrences of a health endpoint corresponding to a particular change in concentration;

y_0 = baseline incidence rate per person;

β= coefficient; usually derived from the percent change in the health endpoint extracted from an epidemiologic study or meta-analysis;

$\Delta conc$ = change in PM or ozone concentration; and

pop= population being exposed to the change in concentration.

Baseline incidence rates for these functions are determined using data available from a variety of databases assembled by California state health agencies. These include the California Office of Statewide Health Planning and Development and the Department of Health Services.

1. Particulate Matter

To determine concentration estimates for each pollutant related to goods movement an emissions inventory approach was used. It is not possible to estimate total diesel PM-related concentrations based on emissions estimates alone—because not all PM is directly emitted. Primary diesel PM, or directly emitted diesel PM, can be estimated directly from the emissions inventory. Secondary diesel-related PM is formed in the atmosphere from the precursors: SO_2, NO_X and other organic compounds. An estimate of the particle nitrate formed from goods movement-related NO_X must be calculated to derive secondary diesel PM estimates; similarly, diesel PM formed from goods movement-related ROG must also be estimated to address secondary organic aerosols (SOA). Details on how each of the pollutant concentrations was derived are provided above and in the Technical Supplement. To quantify the health impacts of diesel PM, four basic steps are required:

1. Estimate the basin-specific PM2.5 concentrations attributed to diesel sources.
2. Calculate the health impacts for the base year 2000 by applying a concentration-response (C-R) function to the exposed population for each basin; details on the selection of health endpoints and C-R functions are discussed earlier in Section II.D above. Without available studies addressing the relative toxicity of diesel PM compared to PM2.5, we assumed it's equally toxic. In reality, diesel PM may be more toxic than other components of PM2.5; hence, our assessment may underestimate the true effects.

3. Associate the health impacts with the related emission inventory in the base year (diesel PM, NO_X and ROG for primary diesel PM, particle nitrate, and SOA respectively) to determine the specific factors of tons per annual case of health endpoint.
4. Apply factors to the Goods Movement emission inventory (adjusted to reflect lower impacts from emissions over the oceans and bays) to estimate the average annual impacts for each health endpoint (with population growth adjustment) for years 2005, 2010, 2015, and 2020.

Sources such as tire wear, brake wear, and ship boilers emit PM2.5, which are not captured by primary diesel PM. To address these sources, health impacts for total PM2.5 and primary diesel PM were calculated based on diesel PM factors. Since diesel PM emissions come from a PM10 inventory, and about 92% are PM25, the health impacts due to non-diesel PM2.5 sources were estimated as: PM2.5 impacts – 0.92 * diesel PM impacts. Note that the concentration-response functions between PM and mortality were based on PM2.5, so this is a reasonable approximation of the non-diesel PM2.5 effect.

A critical issue here is the categorization of volatile organic compounds (VOC) emissions, and how that relates to formation of SOA. Many different types of VOCs are emitted into the atmosphere, where they can affect SOA formation at different rates. One of the major uncertainties is the assumption of all ROG emissions have equal propensity for form SOA. Diesel emissions are supposed to contain a high fraction of high molecular weight compounds (especially from ships), which could also influence SOA production.

Currently, the details of SOA formation are not well known, and the implications for needs related to the development of emission factors and other emissions estimation tools to characterize the precursor emissions remain uncertain. Large carbon number organic compounds that have an affinity to stick together could contribute significantly to these processes. Future development efforts may need to be directed to expand VOC speciation profiles to include compounds that improve the methods for characterizing SOA formation. Additional uncertainties are associated with lack of proper time and spatial resolution in ambient measurements of both primary and secondary organic species. These detailed measurements are critical in evaluating influence of meteorology and diurnal and seasonal changes in emissions.

2. Ozone

For health effects due to goods movement-related ozone concentrations, staff followed the same basic procedure outlined in the CARB and OEHHA's Review of the Ozone Standards (CARB/OEHHA 2005b), which itself was based on methods developed by the U.S. EPA for assessment of health benefits (Hubbell *et al.* 2005). The basic approach is the same as that for PM discussed above. However, concentrations by basin are based on the actual 2001-2003 daily measurements, used to calculate the health impacts due to exposures above the newly approved State 8-hour standard of 0.070 ppm. In that calculation, staff estimated the daily concentrations that would result in a hypothetical setting of attainment of the 8-hour standard. The difference between the two sets of measurements, considered at the daily level to account for day-of-week

variation in ozone measurements (the “weekend” effect), was used to quantify the health impacts. As detailed in the Ozone Standard Staff Report (CARB/OEHHA 2005b), ozone concentrations in the SoCAB, where a majority of the population reside, have declined at a consistent rate throughout the distribution of the ozone levels. Consequently, strategies to control both ROG and NO_X are considered to be equally effective. The basin-specific health impacts due to ozone exposures above the 8-hour standard are associated with total emissions from reactive organic gas (ROG) and NO_X emissions that would need to be reduced to attain the standard to determine health impact factors. These factors are then applied to the Goods Movement total inventories of ROG and NO_X to determine the health impacts. Further details on the peer-reviewed studies used to derive coefficients for ozone health impacts can be found in the Ozone Standard Staff Report (CARB/OEHHA 2005b) and in Ostro et al. 2006.

3. Port-Specific Modeling

To estimate potential non-cancer health impacts associated with exposures to directly emitted diesel PM from the Ports of Los Angeles and Long Beach, we used air dispersion modeling of ambient directly emitted diesel PM (primary diesel PM). The detailed methodology for this analysis is presented in the October 2005 draft report “Diesel PM Exposure Assessment Study for the Ports of Los Angeles (POLA) and Long Beach (POLB)” (CARB 2005a). The non-cancer health effects evaluated include premature death, hospital admissions, asthma and other lower respiratory symptoms, acute bronchitis, work loss days, and minor restricted activity days – as was done for PM in the rest of the state.

To estimate the ambient concentration levels of primary diesel PM resulting from port operations, CARB staff conducted air dispersion modeling. We evaluated the impacts from the 2002 estimated on-port property and over-water emissions for five categories of emission sources at the ports: cargo handling equipment, on-road heavy-duty trucks, locomotives, ocean-going vessels, and commercial harbor craft. Meteorological data from Wilmington was used for this study. The Wilmington site is about one mile away from the ports, and the measurements were collected in 2001. The U.S. EPA’s ISCST3 air dispersion model was used to estimate the annual average offsite concentration of diesel PM in the area surrounding the two ports. The modeling domain (study area) spans a 20 x 20 mile area, which includes both the ports, the ocean surrounding the ports, and nearby residential areas in which about 2 million people live. The land-based portion of the modeling domain, excluding the property of the ports, comprises about 65% of the modeling domain. A Cartesian grid receptor network (160 x 160 grids) with 200 x 200 meter resolution was used in this study.

The annual average above ambient diesel PM concentration in each grid cell was calculated using the U.S. EPA ISCST3. The population within each grid cell was determined from U.S. Census Bureau year 2000 census data. Using the methodology peer-reviewed and published in the Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates, (PM Staff Report) (CARB, 2002), we calculated the number of annual cases of death and other health effects associated with exposure to the above ambient PM concentrations modeled for each of the grid cells. For each grid cell, each health effect was estimated based on concentration-response functions derived from published

epidemiological studies relating changes in ambient concentrations to changes in health endpoints, the population affected, and the baseline incidence rates. The total impacts for the affected population in the modeling domain were obtained by summing the results from each grid cell.

To estimate the non-cancer health effects in areas outside the modeling domain, we interpolated the diesel PM concentrations from the modeling domain (20 mile x 20 mile) into an area of 40 mile x 30 mile in the north direction and another area of 20 mile x 20 mile in the east direction of the modeling domain. Concentrations into the south and west directions of the modeling domain were not interpolated because these areas are located over the ocean. The expanded model receptor domain covers an area of 40 mile (east-west) and 50 mile (north-south) and includes a population of about 10 million people. The non-cancer health effects presented in this report are derived from the expanded modeling domain, i.e., 40 mile x 50 mile.

E. Economic Valuation of Health Effects

This section describes the methodology for monetizing the value of avoiding the adverse impacts associated with goods movement-related emissions as discussed in the previous section. The most significant inputs into the analysis are the incident rates as previously discussed and the valuations associated with each endpoint (e.g., premature death). In addition, the discount rates that are chosen for valuing the avoidance of the adverse impacts are also discussed.

The U.S. EPA has established $4.8 million in 1990 dollars at the 1990 income level as the mean value of avoiding one premature death (U.S. EPA, 1999, pages 70-72). This value is the mean estimate from five contingent valuation studies and 21 wage-risk studies, with estimates ranging from $0.6 million to $13.5 million in 1990 dollars, (or $0.9 million to $20.1 million in 2005 dollars).

Contingent valuation and wage-risk studies examine the willingness to pay (or accept) for a minor decrease (or increase) in mortality risk. For example, if 10,000 people are willing to pay $800 apiece for risk reduction of 1/10,000 then collectively the willingness-to-pay for avoiding a premature death, in this example, would be $8 million. This is also known as the “value of a statistical life” or VSL.

Contingent valuation studies provide stated preference data about willingness-to-pay for decreased levels of risk. Such studies pose a market situation to survey respondents who are asked how much they would be willing to pay. The approach is useful for getting estimates on willingness-to-pay (WTP) for policies that have not yet been implemented. The earliest techniques involved asking people directly how much they value incremental risk avoidance. Today, the more effective referendum format suggests a specific dollar amount and then asks respondents whether they would be willing to pay that amount to decrease the probability of experiencing a well-defined adverse health outcome (Freeman, 2003).

Wage-risk studies provide revealed preference data about willingness to accept increased levels of risk. Willingness-to-pay and willingness-to-accept result in very close estimates when the change in risk is small. Such studies look at comparisons between different jobs in terms of wages and risks of death on the job. The comparisons focus on

risk by controlling for other differences in job attributes. The compensating wage approach may underestimate the value of preventing premature mortality, because people who are willing to be paid to accept increased risk may value risk reduction less than the average person (Freeman 2003).

Table A-7 provides some information about the 26 studies that U.S. EPA used to calculate its estimate for the value of avoiding a premature death, or VSL. U.S. EPA averaged the 26 estimates to get a value of 4.8 million in 1990 dollars. This value applies to both adult and infant mortality.

Table A-7 Collected Valuations of Premature Deaths Prevented

Authors	Year	Type of Estimate	Valuation (millions 1990$)	Annual risk reduction	Implied compensating wage (1990$/year)
Kneisner and Leeth	1991	Wage-risk	0.6	0.0004	240
Smith and Gilbert	1984	Wage-risk	0.7		
Dillingham	1985	Wage-risk	0.9		
Butler	1983	Wage-risk	1.1	0.00005	60
Miller and Guria	1991	Cont. Valu.	1.2		
Moore and Viscusi	1988	Wage-risk	2.5		
Viscusi, Magat, and Huber	1991	Cont. Valu.	2.7		
Gegax et al.	1985	Cont. Valu.	3.3		
Marin and Psacharopoulos	1982	Wage-risk	2.8		
Kneisner and Leeth	1991	Wage-risk	3.3		
Gerking, de Haan, and Schulze	1988	Cont. Valu.	3.4		
Cousineau, Lacroix, and Girard	1988	Wage-risk	3.6		
Jones-Lee	1989	Cont. Valu.	3.8		
Dillingham	1985	Wage-risk	3.9		
Viscusi	1979	Wage-risk	4.1	0.0001	410
Smith	1976	Wage-risk	4.6	0.0001	460
Smith	1976	Wage-risk	4.7	0.0001	470
Olson	1981	Wage-risk	5.2	0.0001	520
Viscusi	1981	Wage-risk	6.5	0.0001	650
Smith	1974	Wage-risk	7.2	0.000125	900
Moore and Viscusi	1988	Wage-risk	7.3	0.00006	440
Kneisner and Leeth	1991	Wage-risk	7.6		
Herzog and Schlottman	1987	Wage-risk	9.1	0.000097	880
Leigh and Folson	1984	Wage-risk	9.7	0.0001	970
Leigh	1987	Wage-risk	10.4		
Garen	1988	Wage-risk	13.5	0.000108	1,460

U.S. EPA's most recent regulatory impact analyses, (U.S. EPA 2004, 2005), apply a different estimate of the value of avoiding one premature death, ($5.5 million in 1999 dollars). This revised value is based on more recent meta-analytical literature, and has not yet been assessed or endorsed by the Environmental Economics Advisory Committee (EEAC) of U.S. EPA's Science Advisory Board (SAB). Unless and until U.S. EPA's SAB reviews and endorses the revised estimate, CARB staff will continue to use the last VSL estimate approved for use by the SAB, i.e., $4.8 million in 1990 dollars.

As real income increases, people are willing to pay more to prevent premature death. U.S. EPA adjusts the 1990 value of avoiding a premature death by a factor of 1.201[1] to account for real income growth from 1990 through 2020, (U.S. EPA, 2004, page 9-121). Assuming that real income grows at a constant rate from 1990 until 2020, we adjusted VSL for real income growth, increasing it at a rate of approximately 0.6% per year. We also updated the value to 2005 dollars. After these adjustments, the value of avoiding one premature death is $7.9 million in 2005, $8.1 million in 2010 and $8.6 million in 2020, all expressed in 2005 dollars.

The U.S. EPA also uses WTP methodology for some non-fatal health endpoints, including lower respiratory symptoms, acute bronchitis and minor restricted activity days. WTP values for these minor illnesses are also adjusted for anticipated income growth through 2020, although at a lower rate, (1.066 in lieu of 1.201).

For school absences and work-loss days, the U.S. EPA uses an estimate of the parent's lost wages, (U.S. EPA, 2004), which CARB adjusts for projected real income growth.

"The Economic Value of Respiratory and Cardiovascular Hospitalizations," (ARB, 2003), calculated the cost of both respiratory and cardiovascular hospital admissions in California as the cost of illness plus associated costs such as loss of time for work, recreation and household production. CARB adjusts these COI values by the amount that annual medical care price increases for hospitalization exceed "all-item" price increases (CPI).

Table A-8 lists the valuation of avoiding various health effects, compiled from CARB and U.S. EPA publications, updated to 2005 dollars. The valuations based on WTP, as well as those based on wages, are adjusted for anticipated growth in real income.

[1] U.S. EPA's real income growth adjustment factor for premature death incorporates an elasticity estimate of 0.4.

Table A-8 Undiscounted Unit Values for Health Effects (in 2005 Dollars and current income levels) [1]

Health Endpoint	2005	2010	2020	References
Mortality				
Premature death ($ million)	7.9	8.1	8.6	U.S. EPA (1999), (2000), (2004)
Hospital Admissions				
Cardiovascular ($ thousands)	41	44	49	CARB (2003), p.63
Respiratory ($ thousands)	34	36	40	CARB (2003), p.63
Minor Illnesses				
Acute Bronchitis	422	440	450	U.S. EPA (2004), 9-158
Lower Respiratory Symptoms	19	19	20	U.S. EPA (2004), 9-158
Work loss day	180	195	227	2002 California wage data, U.S. Department of Labor
Minor restricted activity day (MRAD)	60	62	64	U.S. EPA (2004), 9-159
School absence day	88	95	111	U.S. EPA (2004), 9-159

[1]The value for premature death is adjusted for projected real income growth, net of 0.4 elasticity. Wage-based values (School absences, Work Loss Days) are adjusted for projected real income growth, as are WTP-derived values, (Lower Respiratory Symptoms, Acute Bronchitis, and MRADs). Health endpoint values based on cost-of-illness, (Cardiovascular and Respiratory Hospitalizations), are adjusted for the amount by which projected CPI for Medical Care (hospitalization) exceeds all-item CPI.

F. Uncertainty Calculations

Health impacts, (the number of cases), were estimated with a range that reflects the uncertainty of the underlying concentration-response functions. Per-case economic valuations of health impacts also reflect the uncertainty of the economic estimation. For estimates of the value of premature death, or VSL, this uncertainty is considerable.

Calculating an economic value for any health endpoint entails multiplying the health impacts (number of cases) by the per-case economic valuations. To calculate the uncertainty of the economic value of premature deaths, staff used standard statistical analysis to combine the uncertainty of the concentration-response function (used to derive the number of cases) with the uncertainty of the per-case economic valuation. Based on this method,[1] staff estimated the upper and lower bounds of the 95-percent

[1] The valuation of premature death is the product of multiplying two quantities together: the number of premature deaths times the value of statistical life (VSL). The uncertainty in the valuation depends on the uncertainties in these two quantities. The number of premature deaths appears to have a normal

confidence interval for the economic value of premature deaths avoided by the regulation.

The uncertainty range of our estimates for GM-related premature mortality impacts far exceeds the total uncertainty from all non-mortality health impacts combined. For non-mortality health endpoints, therefore, we did not develop procedures for combining health impact uncertainty with economic valuation uncertainty. For all non-mortality health endpoints our estimates of economic impact reflect only the uncertainty of underlying concentration-response functions.

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distribution. VSL has a lognormal distribution. Because their product does not have a recognized statistical distribution, we calculate it by numerical integration. From numerical integration, we obtained: 2.5^{th} percentile = 0.31; and 97.5^{th} percentile = 1.88. Therefore the lower bound of the 95% CI equals 0.31 of the calculated mean and the upper bound equals 1.88 times the calculated mean. We used these factors to calculate the upper and lower 95% CI for our dollar estimate of premature mortality impacts.

IV. Results

A. Emissions Estimates

The mass-based calculation of health impacts requires a statewide emissions inventory, and an emissions inventory representing goods movement. Both of these inventories are adjusted to account for the dispersion of emissions generated by ocean-going ships and harbor craft, as described above.

Table A-9 provides ports and goods movement emissions, by pollutant, that have been adjusted to reflect the dispersion adjustment factor for diesel PM. To adjust for dispersion, all emissions over water were discounted by 90% except for emissions within 3 miles of the San Diego and San Francisco Bay Area Air Basins, which were discounted by 75%. Diesel PM emissions associated with the health risk assessment of the Port of Los Angeles and Long Beach are excluded from Table A-9. Those emissions are excluded because they are not used to calculate health impacts; instead, the Ports' health risk assessment is used to calculate health impacts.

Table A-9 Dispersion-Adjusted Goods Movement Emissions Inventory

Pollutant	2005	2010	2015	2020
Diesel PM	42	30	21	17
NO_X	1,079	892	771	721
ROG	90	72	57	51
SO_X	95	108	138	182

Table A-10 provides a summary of the dispersion-adjusted draft 2006 statewide emissions inventory, including ocean-going ships out to 24 nautical miles from shore. To adjust for dispersion, all emissions over water were discounted by 90% except for emissions within 3 miles of the San Diego and San Francisco Bay Area air basins, which were discounted by 75%.

Table A-10 Dispersion-Adjusted Statewide Emissions[1]

Pollutant	1998	2000	2005	2010	2015	2020
Diesel PM	74	71	67	57	48	43
NO_X	3,865	3,787	3,161	2,651	2,226	2,021
ROG	3,340	3,126	2,424	2,155	2,031	1,985
SO_X	228	265	264	290	329	381

[1]Biogenic, geogenic, wildfires, windblown dust are included for NO_X and SO_X, but not for other pollutants.

B. Exposure Estimates

Table A-11 summarizes the exposure estimates used in the analysis of the health impacts. These are estimated population-weighted concentrations for each air basin of California using the methodology described in the previous section. They provide an integrated regional perspective rather than an indication of exposure at any individual

location, but are consistent with how the concentration-response functions are derived in the epidemiological studies.

Table A-11 Exposure Estimates by Air Basin.

Base Year	1998	1998	2000	2000	2003
AIR BASIN	Nitrate[1] (μg/m^3)	Sulfate[2] (μg/m^3)	SOA[3] (μg/m^3)	DPM[4] (μg/m^3)	O_3[5] (ppm)
Great Basin Valleys	0.77	0.49	0.40	0.10	0.084
Lake County	0.80	0.39	0.51	0.20	0.071
Lake Tahoe	0.32	0.19	0.30	0.40	0.081
Mojave Desert	2.71	0.95	0.61	0.40	0.117
Mountain Counties	1.00	0.63	0.70	0.40	0.122
North Central Coast	1.00	0.43	0.61	0.80	0.089
North Coast	0.55	0.30	0.34	0.80	0.068
Northeast Plateau	0.32	0.20	0.32	0.70	0.072
Sacramento Valley	1.13	0.62	0.98	1.20	0.111
Salton Sea	2.32	1.29	0.32	1.50	0.119
San Diego	2.64	0.82	0.63	1.40	0.101
San Francisco Bay	1.05	0.52	0.73	1.60	0.098
San Joaquin Valley	1.79	1.31	0.73	1.30	0.122
South Central Coast	1.58	1.07	0.62	1.10	0.103
South Coast	4.63	1.16	1.11	2.40	0.146
CALIFORNIA	2.87	0.94	0.88	1.80	N/A

[1] Particle nitrate exposure based on inverse-distance-weighted and population-weighted annual geometric means for particle nitrate.

[2] Particle sulfate exposure based inverse-distance-weighted and population-weighted annual geometric mean for particle sulfate. Although it is presented here, particle sulfate was not part of our health impacts assessment in this report.

[3] Secondary organic aerosol (SOA) exposure based on inverse-distance-weighted and population-weighted annual arithmetic means for secondary organic aerosols.

[4] Diesel PM (DPM) is derived from receptor modeling results, emissions, and monitoring data.

[5] Ozone 1-hour peak indicator is based on 2001-2003 data and provides the basis for the assessment of the health impacts of exposures above the ozone ambient air quality standards. For details, see Appendix B of the ozone standard staff report (CARB/OEHHA 2005b).

C. Health Impacts Assessment

The next series of tables present the results of our health impacts assessment. Tables A-12 through A-15 present results that include those modeled for the SoCAB ports. In other words, information from Table A-16 is already incorporated into Tables A-12 through A-15. All results have been rounded to two significant figures; hence, the totals may not add up exactly.

1. Statewide Impacts

Shown in Table A-12-a is a summary of the combined statewide health effects from PM and ozone exposure linked with goods movement. We estimate that 2,400 premature deaths (720 – 4,100, 95% confidence interval (95%CI)) can be associated with goods movement emissions, annually on a statewide basis. Table A-12-b shows the statewide valuation of health effects associated with goods movement within California. The values reported in this table result from multiplying number of health effects cases reported in Table A-12-a by the unit valuations of Table A-8, discounted at 3% and 7% per year, using the discount rates recommended by U.S. EPA's guidance on social discounting (U.S. EPA, 2000). A detailed discussion of the discount rates can be found in Section D.

2. Air Basin-Specific Impacts

Since the majority of the economic impact arises from the estimated number of premature death, more detailed analysis of this health endpoint was conducted. For example, the number of premature deaths was calculated for each air basin (Table A-13). Our analysis showed about 50% of the premature deaths associated with goods movement occur in the SoCAB, while the San Diego County, San Francisco Bay Area, and San Joaquin Valley Air Basins collectively accounted for 27%. Moreover, for the SoCAB, goods movement-related health impacts account for a large portion of the total impact of ozone and PM pollution from all sources.

Table A-12-a Statewide PM and Ozone Health Effects Associated with Ports and Goods Movement[1] (Uncertainty range in parentheses)

Health Outcome	2005	2010	2020
Premature Death	2,400 (720-4,100)	2,000 (610-3,400)	1,700 (500-2,800)
Hospital Admissions (respiratory causes)	2,000 (1,200-2,800)	1,700 (1,000-2,400)	1,500 (860-2,100)
Hospital Admissions (cardiovascular causes)	830 (530-1,300)	710 (450-1,100)	580 (360-890)
Asthma and Other Lower Respiratory Symptoms	62,000 (24,000-99,000)	52,000 (20,000-83,000)	42,000 (16,000-66,000)
Acute Bronchitis	5,100 (-1,200-11,000)	4,300 (-1,000-9,300)	3,400 (-820-7,500)
Work Loss Days	360,000 (310,000-420,000)	310,000 (260,000-350,000)	250,000 (210,000-290,000)
Minor Restricted Activity Day	3,900,000 (2,200,000-5,800,000)	3,300,000 (1,900,000-5,000,000)	2,800,00 (1,500,000-4,100,000)
School Absence Days	1,100,000 (460,000-1,800,000)	1,000,000 (410,000-1,600,000)	860,000 (350,000-1,400,000)

[1]Does not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates. A negative value as a lower bound of the uncertainty range is not meant to imply that exposure to pollutants is beneficial; rather, it is a reflection of the adequacy of the data used to develop these uncertainty range estimates. Additional details on the methodology and the studies used in this analysis are given in earlier sections of the Appendix.

Table A-12-b Economic Valuation of Statewide PM and Ozone Health Effects Associated with Ports and Goods Movement in present value dollars[1]

(Uncertainty range in parentheses)

Health Outcome	2005 ($million)	2010 ($million)	2020 ($million)
Premature Death	$19,000 ($5,900-$36,000)	$13,000 to $15,000 ($3,900-$28,000)	$5,500 to $9,400 ($1,700-$18,000)
Hospital Admissions (respiratory causes)	$67 ($40-$93)	$47 to $55 ($28-$77)	$23 to $39 ($13-$55)
Hospital Admissions (cardiovascular causes)	$34 ($22-$53)	$23 to $27 ($15-$42)	$11 to $19 ($6.9-$29)
Asthma and Other Lower Respiratory Symptoms	$1.1 ($0.44-$1.8)	$0.77 to $0.89 ($0.30-$1.4)	$0.32 to $0.54 ($0.12-$0.87)
Acute Bronchitis	$2.2 ($-0.52-$4.7)	$1.4 to $1.7 ($-0.35-$3.7)	$0.60 to $1.0 ($-0.14-$2.2)
Work Loss Days	$65 ($55-$75)	$46 to $53 ($39-$61)	$22 to $37 ($19-$43)
Minor Restricted Activity Day	$230 ($130-$350)	$160 to $190 ($88-$280)	$69 to $120 ($38-$170)
School Absence Days	$100 ($41-$160)	$72 to $84 ($29-$140)	$37 to $63 ($15-$100)
Total	$19,000 ($6,000 - $36,000)	$13,000 to $15,000 ($4,000 - $28,000)	$5,700 to $9,700 ($2,000 - $18,000)

[1]Valuation in millions of 2005 dollars. @ 3% - discounted at 3% per year, @ 7% - discounted at 7% per year. The health impacts associated with the economic values in this table do not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions for morbidity endpoints and combined uncertainty in concentration-response functions and economic values for premature death, but not in emissions or exposure estimates. A negative value as a lower bound of the uncertainty range is not meant to imply that exposure to pollutants is beneficial; rather, it is a reflection of the adequacy of the data used to develop these uncertainty range estimates. Additional details on the methodology and the studies used in this analysis are given in earlier sections of the Appendix.

Table A-13 Basin-Specific Mortality Effects Associated with Ports and Goods Movement[1]

Year	2005		2010		2020	
Air Basin	Mean Deaths	Uncertainty Range	Mean Deaths	Uncertainty Range	Mean Deaths	Uncertainty Range
Great Basin Valleys	<1	(<1)	<1	(<1)	<1	(<1)
Lake County	<1	(<1)	<1	(<1)	<1	(<1)
Lake Tahoe	1	(<1-1)	<1	(<1-1)	<1	(<1-1)
Mountain Counties	16	(5-27)	12	(4-20)	8	(3-14)
Mojave Desert	150	(54-250)	120	(43-200)	90	(31-140)
North Coast	2	(1-3)	2	(<1-3)	1	(<1-2)
North Central Coast	14	(4-24)	10	(3-17)	6	(2-11)
Northeast Plateau	5	(1-8)	3	(1-6)	2	(1-4)
South Coast	1,200	(360-2,100)	1100	(310-1,800)	800	(240-1,400)
South Central Coast	69	(21-120)	73	(22-120)	97	(30-160)
San Diego	150	(44-260)	140	(41-240)	200	(57-340)
San Francisco	220	(61-380)	190	(53-330)	180	(50-300)
San Joaquin Valley	270	(84-460)	200	(63-340)	120	(39-210)
Salton Sea	140	(43-230)	110	(36-190)	79	(25-130)
Sacramento Valley	140	(42-240)	110	(33-180)	75	(23-130)
Total	2,400	(720-4,100)	2,000	(610-3,400)	1,700	(500-2,800)

[1] Values are rounded. Mortality impacts do not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates.

3. Source-Specific Impacts

We also investigated the contribution of specific goods movement-related sources to air pollution problems. We found that the source of air emissions most responsible for estimated the health impacts is trucking, with ocean going ships, rail and harbor craft as significant contributors (Table A-14). The relative ranking was similar for statewide estimates and for estimates of the health impacts in the major air basins (data not shown).

Table A-14 Mortality Effects Associated with Ports and Goods Movement: Contributions of Source Categories[1] (Uncertainty range in parentheses)

Source Category	2005 Number of deaths	2010 Number of deaths	2020 Number of deaths
Commercial Harbor Craft	140	120	85
	(41- 240)	(35-200)	(25-150)
Cargo Handling Equipment	43	38	16
	(13-73)	(11-64)	(5-28)
Ocean-Going Ships	210	290	540
	(63-360)	(86-490)	(160-910)
Rail (Locomotives)	270	230	290
	(84-460)	(69-380)	(89-490)
SoCAB Ports (modeled)	67	75	96
	(18-120)	(20-130)	26-170
Truck	1,500	1,200	580
	(460-2,600)	(360-2,000)	(180-990)
Transport Refrigeration Units	130	99	48
	(36-220)	(29-170)	(15-81)
STATEWIDE TOTAL	2,400	2,000	1,700
	(720-,4100)	(610-3400)	(500-2,800)

[1]Does not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates.

4. Pollutant-Specific Impacts

The contribution of primary diesel PM, secondary particle nitrate, secondary organic aerosols, other primary PM2.5, and ozone to the mortality estimates are summarized in Table A-15.

Table A-15
Mortality Effects Associated with Goods Movement: Pollutant Contributions[1]
(Uncertainty range in parentheses)

Pollutant	Number of Deaths in Each Year		
	2005	2010	2020[2]
Primary Diesel PM	1,200 (330-2,000)	920 (260-1,600)	630 (170-1,100)
Secondary Diesel PM (Nitrate)	940 (260-1600)	850 (240-1,500)	790 (220-1,400)
Secondary Organic Aerosols	29 (8-50)	25 (7-43)	20 (5-34)
Other Primary PM2.5[3]	23 (6-39)	26 (7-44)	41 (11-71)
Ozone	240 (120-350)	210 (100-310)	180 (88-260)
Statewide Total	2,400 (720-4,100)	2,000 (610-3,400)	1,700 (500-2,800)

[1]Does not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates.

[2]These values may overestimate the health impacts if the state ambient air quality standards for particulate matter and ozone are attained by the year 2020.

[3]PM2.5 includes truck tire wear and brake wear, and particles from ship boilers, which are not covered under primary diesel PM.

5. Cancer Risk

For diesel PM, the regional "background" risk in urban areas is 500-800 potential cancers per million people over a 70-year period. For areas in close proximity to major diesel sources, the increase in potential cancer risk can exceed 500 potential cancers per million people over a 70-year exposure period, effectively doubling the risks of those exposed. Since the concentration of diesel PM in the air declines with distance from the source, risks decrease the farther one moves away from goods movement activity centers. However, even several miles away, the elevated cancer risk can still exceed 10 expected cancers per million people exposed. To put these risk numbers into perspective, new stationary sources of air pollution, such as power plants and other industrial facilities are currently required to be designed to ensure that cancer risk from an individual source do not exceed 10 potential cancers per million persons exposed.

Based on CARB's preliminary work, cargo-handling equipment and ship hotelling activities are anticipated to be the largest contributors of toxic pollutants to neighboring communities. While ocean-going vessel transiting emissions contribute a substantial portion of the total port-related diesel PM, they do not produce a comparable cancer risk because those emissions are distributed over a very wide area. Most of the diesel PM emissions (90%) are emitted during transit in California Coastal Waters. In addition, the emission plume from ocean- going vessels has a much higher dispersion release height

due to a higher physical stack height (about 50 meters) of the vessel. Cargo handling equipment and ship hotelling activities, on the other hand, occur in closer proximity to the affected communities and cargo handling equipment has a much lower dispersion release because of a relatively lower physical stack height (about 4-5 meters). CARB staff plans to have more detailed exposure assessments available in the future.

6. Port-Specific Impacts

Based on the methodology described above in section D-3, we estimated the non-cancer health effects, including premature death, hospital admissions, asthma and other lower respiratory symptoms, work loss days, and minor restricted activity days, for the Ports of Los Angeles (POLA) and Ports of Long Beach (POLB) and for five different years. The results for years 2005, 2010, and 2020 are summarized in Table A-16. Note that these results are derived from the POLA and POLB and cannot be applied to other ports. This is because that the non-cancer health effects depend on several factors: port activity pattern, emission spatial and temporal allocation, relations of the emission source versus receptor distance, the population density in the nearby communities, topographical feature in the ports and surrounding areas, and meteorological conditions. These results have been incorporated into Tables A-12 through A-15.

Table A-16 Non-Cancer Health Effects from Activities at the Ports of Los Angeles and Long Beach[1]

Health Outcome	2005	2010	2020
Premature Death	67 (18-120)	75 (20 – 130)	96 (30 – 170)
Hospital Admissions (respiratory causes)	14 (9 – 20)	16 (10- 22)	21 (13 – 29)
Hospital Admissions (cardiovascular causes)	27 (17-41)	30 (29-46)	38 (24 – 60)
Asthma and Other Lower Respiratory Symptoms	2,100 (780-3,300)	2,300 (880 – 3,700)	3,000 (1,100 – 4,800)
Acute Bronchitis	170 (-40 – 390)	190 (-150 – 430)	250 (-58 – 560)
Work Loss Days	12,000 (10,000 – 14,000)	14,000 (12,000 – 16,000)	18,000 (15,000 – 20,000)
Minor Restricted Activity Day	71,000 (58,000 – 84,000)	79,000 (64,000 – 94,000)	100,000 (83,000 – 120,000)

[1]Does not include the contributions from particle sulfate formed from SO_X emissions, which is being addressed with several ongoing emissions, measurement, and modeling studies. Range reflects uncertainty in health concentration-response functions, but not in emissions or exposure estimates. A negative value as a lower bound of the uncertainty range is not meant to imply that exposure to pollutants is beneficial; rather, it is a reflection of the adequacy of the data used to develop these uncertainty range estimates. Additional details on the methodology and the studies used in this analysis are given in earlier sections of the Appendix.

D. Economic Valuation of Health Effects

Table A-17 shows the value of health effects associated with goods movement within California. The estimates in this table result from multiplying the mean number of health effects cases, from Table A-12-a, by their undiscounted unit values, from Table A-8, and

discounting the value of future health effects at both 3% and 7% per year, rates recommended by U.S. EPA's guidance on social discounting (U.S. EPA, 2000).

Table A-17 Value of Statewide Health Effects of Ozone and PM Associated with Goods Movement in California (Millions of current value dollars)

Health Outcome	2005 Value [1]	2010 Val. @ 3%	2010 Val. @ 7%	2020 Val. @ 3%	2020 Val. @ 7%
Premature Death	$19,000	$15,000	$13,000	$9,400	$5,500
Hospital Admissions (respiratory causes)	$67	$55	$47	$39	$23
Hospital Admissions (cardiovascular causes)	$34	$27	$23	$19	$11
Asthma and Other Lower Respiratory Symptoms	$1.1	$0.89	$0.77	$0.54	$0.32
Acute Bronchitis	$2.2	$1.7	$1.4	$1.0	$0.60
Work Loss Days	$65	$53	$46	$37	$22
Minor Restricted Activity Day	$230	$190	$160	$120	$69
School Absence Days	$100	$84	$72	$63	$37

[1]Values are expressed in millions of 2005 dollars. 2005 values are undiscounted. 2010 and 2020 values are discounted at 3% and 7% per year.

Table A-17 shows the sensitivity of health effects values to the choice of social discount rates. Social discounting represents society's preference for present benefits over future benefits. The value of future health impacts discounted to the present becomes smaller, and signals a preference for immediate impacts, putting more emphasis on programs with earlier air pollution reductions. Lower rates discount the value of future health impacts less, resulting in values closer to present, undiscounted values. The range of discount rates in Table A-17 shows that a 7- percent discount rate signals a higher preference for present health impacts than a 3 percent rate. For example, the present value of premature deaths associated with goods movement emissions in 2020 is much lower when discounted at 7 percent, ($5.5 billion), than at 3 percent ($9.4 billion).

V. Discussion

A. Health Impacts Assessment

1. Statewide Impacts

The California Air Resources Board assessed the potential health effects associated with exposure to air pollutants arising from port-related goods movement activities (port, rail, and truck) in the State. This analysis focused on particulate matter and ozone because they represent the majority of risk associated with exposure to outdoor air pollution, and there have been sufficient studies performed to allow quantification of the health effects associated with emission sources.

We estimate that 2,400 premature deaths (720 – 4,100, 95% confidence interval (95%CI)) can be associated with goods movement emissions, annually on a statewide basis. To put these mortality numbers into perspective, attaining the California PM and ozone standards statewide would annually prevent about 9,000 premature deaths (3,100 – 15,000) based on 1999-2000 PM and 2001-2003 ozone monitoring data, or 4% of all deaths[1]. This is greater than the number of deaths (4,200 – 7,400) linked to second-hand smoke in the year 2000. In comparison, motor vehicle crashes caused 3,200 deaths and homicides were responsible for 2,000 deaths. Other health endpoints quantified are hospital admissions for respiratory causes, hospital admissions for cardiovascular causes, asthma and other lower respiratory symptoms, acute bronchitis, work loss days, minor restricted activity days and school absences, ranging from hundreds, to hundreds of thousands of cases, annually. We also projected the annual numbers of cases of death and disease for the years 2010 and 2020.

Since the majority of the economic impact arises from the estimated number of premature death, more detailed analysis of this health endpoint was conducted. For example, the number of premature deaths was calculated for each air basin (Table A-13). Our analysis showed about 50% of the premature deaths associated with goods movement occur in the SoCAB, while the San Diego County, San Francisco Bay Area, and San Joaquin Valley Air Basins collectively accounted for 27%. Moreover, for the SoCAB, goods movement-related health impacts account for a large portion of the total impact of ozone and PM pollution from all sources.

We also investigated the contribution of specific goods movement-related sources to air pollution problems. We found that the source of air emissions most responsible for estimated the health impacts is trucking, with ocean going ships, rail, and harbor craft as significant contributors (Table A-14). The relative ranking was similar for statewide estimates and for estimates of the health impacts in the major air basins.

The relative contribution of primary diesel PM, secondary PM (nitrate produced from the atmospheric conversion of goods movement-related NO_X emissions), and ozone to our health impacts estimates was also assessed. While exposure to either PM or ozone is a serious public health issue, the current health impact of these pollutants are not equal.

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[1] According to the Department of Health Services, there are about 235,000 annual deaths due to all causes in California (based on 2001-2003 data)

For example, statewide, it is estimated that ozone exposure above the proposed California eight- hour ozone standard contributes to approximately 630 premature deaths annually (CARB/OEHHA 2005b, Ostro et al. 2006). In contrast, exposure to PM2.5 above the California annual average standard can be associated with 8,200 premature deaths annually. In our goods movement assessment, we also found that the contribution of PM outweighs that of ozone by tenfold (Table A-15). Primary diesel PM is presently the major contributor to the total estimated premature deaths attributable to ports and goods movement, but, in 2020, secondary diesel PM (i.e., particle nitrate) becomes the most significant contributor as measures are already in place to be effective in controlling primary diesel PM emissions in the long run.

It is possible that this relatively large contribution of secondary PM can be mostly attributed to exposures in the SoCAB, which possesses the unique characteristic of a relatively high ambient nitrate concentration and a high population density.

Ambient ozone levels frequently exceed federal and state health protective standards, especially in Central and Southern California. Ports and related goods movement are major sources of the NO_X emissions that react in the atmosphere on warm, sunny days to form ozone. Ozone is a powerful oxidant that can damage the respiratory tract, cause lung inflammation, and irritation, which can lead to breathing difficulties. Statewide, it is estimated that ozone exposure, above the proposed California eight-hour ozone standard, contributed to approximately 630 premature deaths (CARB/OEHHA 2005b, Ostro et al. 2006). It is estimated (Table A-15) that goods movement contributes to approximately 240 premature deaths per year. These statewide numbers can be broken down by air basin to estimate the contribution of various sources to ozone health effects. For example, in the SoCAB, ozone air pollution contributed to approximately 300 additional instances of premature death, and it is estimated that goods movement contributes to approximately 71 premature deaths per year in the SoCAB. CARB staff will examine these and other air basin estimates in its mitigation plan.

Table A-17 shows the total valuation of the current health impacts associated with port-related goods movement and other port activities in California to be about $19 billion (in year 2005 dollars), with an uncertainty range of $6 billion to $36 billion.

2. Sensitivity Discussion

Several new epidemiology studies have recently been published which may also be relevant to the health impacts analysis. In November 2005, a study which analyzed PM exposure and premature death for the SoCAB was published (Jerrett et al. 2005). It found a 2.5 times higher estimate for premature death than the national study by Pope et al. (2002), but greater uncertainty. The 2.5-times higher result appears to be due to better exposure characterization techniques rather than higher toxicity of the PM mixture in Los Angeles. U.S. EPA has not adopted this study in its core health impacts analysis. Several additional studies have either just been published or will be in the next few months. CARB staff intends to review all of these studies and will solicit the advice of the study authors and other experts in the field and U.S. EPA to determine how to best incorporate these new results into our future assessments.

In addition, infant mortality is surfacing as an additional health endpoint in this type of analysis. We calculated a mean of 7 (3 – 11, 95% CI) infant deaths statewide from

exposure to current goods movement pollution sources using the Woodruff et al. (1997) study and a mean of 12 (-13 to 36, 95% CI) for the Woodruff et al. (2006) study. It is important to note that the Woodruff et al. (1997) study uses exposures from an earlier period and does not contain California data, while the Woodruff et al. (2006) study is specific to California and examines more current exposures.

For PM-related respiratory hospital admissions, using the Linn et al. (2000) study for age 30+ would lead to a lower estimate compared to our quantified estimate based on pooling Zanobetti and Schwartz (2003) and Moolgavkar (2000a; 2003a) for age 18+.

Based on Ostro et al. (2001), asthma exacerbations associated with goods movement emissions would be lower than total cases for asthma and other lower respiratory symptoms quantified in our analysis. To avoid double-counting, only estimates for asthma and other lower respiratory symptoms are presented.

Similarly, McConnell et al. (1999) could be used to estimate acute bronchitis and chronic phlegm among asthmatic children. However, because lower respiratory symptoms (including asthma-related symptoms), acute bronchitis, and school loss days are already being quantified, there are concerns of double-counting effects in children. As a result, the asthma-related effects among children are not treated separately.

3. Port-Specific Impacts

Results for port-specific impacts are presented in Table A-16. Below, we discuss two related assessments that address diesel PM health risks near ports and rail yards.

a) Diesel PM Health Risk Assessments

Goods movement related activities are a significant source of exposures to diesel PM. Approximately 70% of the potential cancer risk from toxic air contaminates in California is due to diesel PM. For diesel PM, the regional “background” risk in urban areas is about 500-800 potential cancers per million people over a 70-year period[1]. For areas in close proximity to major diesel sources, such as ports, rail yards and along major transportation corridors, the increase in potential cancer risk can exceed 500 potential cancers per million people over a 70-year exposure period, effectively doubling the risks of those exposed. Since the concentration of diesel PM in the air declines with distance from the source, risks decrease the farther one moves away from goods movement activity centers. However, even several miles away, the elevated cancer risk can still exceed 10 expected cancers per million people exposed.

The potential cancer risks are highly dependent on site specific variables such as the meteorological conditions, the types of activities occurring, the locations and emissions rates of the equipment, operating schedules and the actual location of where people live in relation to the goods movement operation. To better understand the potential health risks associated with living near a goods movement operation, CARB staff conducted

[1]The cancer risk from known carcinogens is expressed as the incremental number of potential cancers that could develop per million people exposed assuming the affected population is exposed to the carcinogen at a defined concentration over a presumed 70-year lifetime. The ratio of potential number of cancers per million people can also be interpreted as the incremental likelihood of an individual exposed to the carcinogen developing cancer from continuous exposure over a lifetime.

two key health risk assessments.[1] One was on a major port complex, and the other on a large rail yard. These health risk assessments were developed in cooperation with the owners and operators of those facilities, and using appropriate meteorological information and modeling techniques.

Below is a summary of the two studies, one for the Ports of Los Angeles and Long Beach located in Southern California, and the other for the J.R. Davis Rail Yard in Roseville, California.

b) Exposure Assessment Study for the Ports of Los Angeles and Long Beach

On October 3, 2005, CARB released the draft results from a diesel PM exposure assessment study for the Ports of Los Angeles and Long Beach. The purpose of the study was to enhance our understanding of the port-related diesel PM emission impacts by evaluating the relative contributions of the various diesel PM emission sources at the ports to the potential cancer risks to people living in communities near the ports. The study focused on the on-port property emissions from locomotives, on-road heavy-duty trucks, and cargo handling equipment used to move containerized and bulk cargo such as yard trucks, side-picks, rubber tire gantry cranes, and forklifts. The study also evaluated the at-berth and over-water emissions impacts from ocean-going vessel main and auxiliary engine emissions as well as commercial harbor craft such as passenger ferries and tugboats. For the ocean-going vessel emissions, the study evaluated the hotelling emissions, i.e., those emissions from vessel auxiliary engines while at berth, separately from the maneuvering and transiting emissions. While there are locomotive and on-road heavy-duty truck emissions associated with the movement of goods through the ports that occur off the port boundaries, these were not evaluated in this study.

The results of the risk assessment show a very large area impacted by the diesel PM emissions associated with the operations and activities of the Ports. Overall, the emissions from the Ports impact areas extending several miles from the Ports. The computer model estimates the risk in a 20-mile by 20-mile area (the study area), with about a 10 to 15 mile boundary around the Ports depending on the direction. The areas with the greatest impact outside of the Ports' boundaries have an estimated potential cancer risk of over 500 in a million and affect about 2,500 acres where 53,000 people live. The area where the risk is predicted to exceed 200 in a million is also very large, covering an area of about 29,000 acres where over 400,000 people live. At the edge of the modeling study area, referred to as the modeling receptor domain, the potential cancer risk was as high as 100 chances in a million in some areas. The affected land area where the predicted cancer risk is expected to be greater than 100 in a million is

[1]A risk assessment is a tool that is used to evaluate the potential for a chemical to cause cancer or other illness. A risk assessment used mathematical models to evaluate the health impacts from exposure to certain concentrations of chemical or toxic air pollutants released from a facility or found in the air. For cancer health effects, the risk is expressed as the number of chances in a population of a million people who might be expected to get cancer over a 70-year lifetime.

estimated to be about 93,500 acres in the study area. Impacts likely extend beyond the study area but were outside of the modeling receptor domain for this study.

The study revealed that cargo-handling equipment and ship hotelling activities are the largest contributors of toxic pollutants to neighboring communities. While ocean-going vessel transiting emissions contribute a substantial portion of the total port-related diesel PM, they did not produce a comparable cancer risk because these emissions are released off-shore and impact a very wide area.

c) Exposure Assessment Study for the J.R. Davis Rail Yard

In October 2004, the CARB released the results from the Roseville Rail Yard Study. The health risk assessment evaluated the impacts from the diesel PM emissions from diesel-fueled locomotives at the Union Pacific J.R. Davis Yard located in Roseville, California. The J.R. Davis Rail Yard serves as a classification, maintenance, and repair facility for Union Pacific Railroad. During the study period, approximately 31,000 locomotives visited the yard resulting in about 25 tons of diesel PM emissions per year. About 50% of the emissions were from moving locomotives, 45% from idling locomotives, and 5% due to locomotive testing. The results from the study showed that the diesel PM emissions from the Yard impacted a large area. Risk levels between 100 and 500 in a million occur over a 700 to 1600 acre area in which about 14,000 to 26,000 people live. Risk levels between 10 and 100 in a million occur over a 46,000 to 56,000 acre area in which about 140,000 to 155,000 people live.

B. Uncertainties and Limitations

There are a number of uncertainties involved in quantitatively estimating the health impacts associated with exposures to outdoor air pollution. Over time, some of these will be reduced as new research is conducted. However, some uncertainty will remain in any estimate. Below, some of the major uncertainties and limitations of the estimated health benefits presented in this report are briefly discussed.

1. Uncertainty Associated with Emissions Estimation

Emissions inventories are complex data sets that represent quantitative estimations of pollutant releases from stationary and mobile sources. These inventories evolve over time as data are updated. As a result, an emissions inventory presented at any given time represents a “snap shot” of the inventory at the time it was generated.

When compiling an emissions inventory, CARB staff assembled the best emissions data that are currently available. These estimates are subject to both variability and uncertainty. Examples of variability include using an average emission factor to represent emissions factors that change with time or other parameters; or representing activity with a single estimate, such as annual hours of equipment operation, when annual hours will vary over time. Examples of uncertainty include assuming an average emission factor from a limited number of vehicle source tests accurately reflects the true emission factor for a population of vehicles in a given area; or assuming a single load factor to represent the average of a population of equipment’s operating cycle, when the true average operating cycle is not well characterized.

CARB staff follows a rigorous quality control process during emissions inventory compilation which is designed to minimize error. At every stage of inventory development emissions estimates are evaluated for potential coding and transcription errors. Emissions inventory totals are compared against similar studies and inventories to ensure emissions estimates are reasonable.

2. Exposure Estimates and Populations

Use of the C-R function requires an input of the pollutant concentration to which the population is being exposed. For diesel PM, this calls for the population-weighted diesel PM concentration. For the calculations presented in this report we used basin-specific population-weighted average concentrations, which were estimated by CARB staff for the identification of diesel exhaust as an air toxic contaminant. The estimation procedure relied on many assumptions, the best available data sets at that time, and a variety of calculation techniques. In brief, the foundations of the estimates were results from three special studies – chemical mass balance (CMB) receptor modeling for the San Joaquin Valley (1988-89 data), the South Coast Air Basin (1986 data), and the San Jose area (1991-92 data). The CMB species considered in these studies were organic carbon and elemental carbon, or total carbon, and several elements, and the studies established overall motor vehicle contributions to PM10 at sampling locations (the base year was taken to be 1990). Diesel contributions to PM10 were estimated by scaling the CMB motor vehicle results with factors determined by a special PM10 emission inventory (constructed by CARB) that included separate estimates for diesel emissions. Then these diesel PM10 concentration estimates for sampling locations were used in interpolation algorithms to estimate regional concentrations; a linear rollback scaling was used to project the estimates forward in time to 1995, 2000 and 2010. Areas outside the special studies' regions were approximated by the San Joaquin Valley diesel PM10 estimates (which were scaled using local emission inventories). Finally, the spatial concentrations were averaged with population number weights to obtain a population weighted diesel PM10 estimate.

Despite the fact that a unique tracer for diesel particulate emissions has not been found, several recent receptor-based estimates of ambient diesel particulate concentrations, including that developed by CARB, show overall consistency in values. The results from such studies are outlined and compared below.

The CARB-funded Children's Health Study (CHS) contained a component in which source contributions to ambient particles were determined for the year 1995. In this work, Schauer et al. (2001) analyzed particulate matter collected at 12 sampling sites in the South Coast Air Basin for 96 organic compounds. A subset of these compounds was used in CMB receptor-based apportionment modeling studies. In contrast to the above CMB modeling for the special studies, this CMB modeling was able to directly estimate diesel particulate contributions to ambient PM (to achieve this separation, a diesel source profile and six other source profiles were utilized).

A third, more recent, CMB modeling study was conducted in the South Coast Air Basin: DOE/NREL's "Gasoline/Diesel PM Split Study." In this project, two preeminent practitioners of organic compound-based PM CMB source apportionment – University of Wisconsin, Madison (J. Schauer) and Desert Research Institute (E. Fujita) – collected

side-by-side mobile source samples (light and heavy-duty vehicle dynamometer tests) and ambient samples. Using these parallel samples, each group carried out independent chemical analyses, profile construction, and CMB modeling. Because of the many differences in sample collection and analysis techniques, profile construction methodologies, and CMB species selection and modeling, each group obtained different estimates for the contribution of diesel exhaust to ambient PM2.5. The relative contributions of gasoline and diesel exhaust to PM2.5 also differed: diesel contributed more than gasoline vehicle exhaust to PM2.5 in E. Fujita's analysis, and the opposite conclusion was found in J. Schauer's analysis.

Several estimates of diesel PM from the above studies are given in the table below. Direct comparisons for location and year are not possible. However, projected estimates from the CARB Diesel PM TAC study compare well in general with CHS's 1995 diesel PM mass estimates and with Gasoline/Diesel PM Split Study's estimates of diesel contributions to total carbon (which are likely close to mass contributions). The exception is J. Schauer's estimates of diesel PM2.5 for the Gasoline/Diesel PM Split Study, which is lower than both CARB's projected estimates and E. Fujita's parallel estimate (and his earlier CHS estimate). Further work is needed to clarify this discrepancy.

Table A-18 Estimated Diesel PM Concentrations.

Diesel PM concentration (μg/m^3)					
Study	**Location**	**1990**	**1995**	**2000**	**2010**
CARB Diesel PM TAC Id.	SoCAB statewide	3.6 (±1.4) 3.0 (±1.1)	2.7 2.2	2.4 1.8	2.4 1.7
CHS	Long Beach Riverside		2.9 (±.3)[1] 1.7 (±.2)[1]		
Gasoline/Diesel Split Study	(Schauer) (Fujita)			0.4-1.5[2] 1.2-3.4[2]	

[1]Average over the year
[2]L.A. North Main, concentration of total carbon from diesel exhaust (2001, summer)

To the extent that there is not a method for directly measuring outdoor diesel PM concentrations, the uncertainty behind primary diesel PM concentrations is unquantified in our analyses.

A related issue is whether small changes in diesel PM concentrations due to goods movement can have a measurable effect on health. It is important to emphasize that while a change may be small, it is an incremental change from a statewide population-weighted PM2.5 average concentration of 18.5 μg/m^3 (based on 1999/2000 data). For secondary diesel PM, particle nitrate monitoring data were used to interpolate and derive the basin-specific population-weighted concentrations. A sensitivity check using county-specific population-weighted concentrations revealed less than 5% change in the health impacts due to secondary sources. Due to insufficient information on particle sulfate, the health impacts associated with secondary diesel PM due to sulfate have not been quantified in this report.

For ozone, California has a monitoring network of approximately 175 monitors located throughout the State. In our ozone staff report (CARB 2005), hourly observations were input into the estimation of the health impacts of ozone exposures above the standard. Several scenarios of characterizing the ozone exposures were considered: averaging monitored values across each county, assigning portions of populations to monitored concentrations within each county, and interpolating exposures for each census tract. All three options led to very similar results

Nonetheless, there are likely uncertainties in the statewide ozone exposure assessment, and in whether the existing monitoring network provides representative estimates of exposure for the general population. We have attempted to reproduce the same relationship between ozone monitor readings and exposure as in the original epidemiological studies. Most of these studies use population-oriented, background, fixed site monitors, often aggregated to the county level. The available epidemiological studies have used multiple pollutant averaging times, and we have proposed conversion ratios for 1-hour to 8-hour and 24- hour ozone concentrations based on national estimates. A preliminary examination of the California monitoring data indicates that the ratios are similar to those found in the highly populated areas of the State. However, uncertainty is added to the estimated impacts of ozone exposure to the extent the converted concentration bases differ from monitored concentrations (CARB/OEHHA 2005b).

There exists some concern on quantifying the health effects due to exposures to outdoor air pollution while people spend much of their time indoors. We recognize this fact. However, the epidemiological studies considered in our review, which led to the chosen the concentration-response functions, found strong links between outdoor air pollution levels and adverse health effects. As more studies are developed to address indoor/outdoor exposures to air pollution, future health impact assessments will take into account the new results.

Related to the issue of exposure estimation is population. In this analysis, staff used population forecasts developed by the Department of Finance (years 2010, 2020) to estimate the health impacts. Without officially quantified uncertainty estimates, we did not incorporate this source of uncertainty in our calculations.

3. Concentration-Response Functions

A primary uncertainty is the choice of the specific studies and the associated concentration-response (C-R) functions used for quantification. Epidemiological studies used for these estimates have undergone extensive peer review and include sophisticated statistical models that account for the confounding effects of other pollutants, meteorology, and other factors. The C-R function used for quantification of death associated with PM exposures is based on a publication by Pope et al. (2002). Vital status and cause of death data were collected by the American Cancer Society as part of an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. The risk factor data for approximately 500,000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through 1998. Pope’s analysis updates the large data set analyzed in 1995 (Pope 1995) and re-analyzed in 2000 (Krewski 2000) with

additional follow-up time (doubling it to more than 16 years and tripling the number of deaths), substantially expands exposure data, including gaseous co-pollutant data and new PM2.5 data, improves control of occupational exposures, incorporates dietary variables that account for total fat consumption, and consumption of vegetables, citrus, and high-fiber grains, and uses recent advances in statistical modeling for incorporating random effects and non-parametric spatial smoothing components.

While there may be questions on whether C-R functions from the epidemiological studies are applicable to California, it should be noted that some of the cities considered by Pope *et al*. are in California. Also, numerous studies have shown that the mortality effects of PM in California are comparable to those found in other locations in the United States. Several new epidemiology studies have recently been published which may also be relevant to the health impacts analysis. In November 2005, a study which analyzed PM exposure and premature death was published (Jerrett et al. 2005). It found a 2.5 times higher estimate for premature death than the national study by Pope et al. (2002), but greater uncertainty. Several additional studies have either just been published or will be in the next few months. CARB staff intends to review all of these studies and will solicit the advice of the study authors and other experts in the field and U.S. EPA to determine how to best incorporate these new results into our future assessments.

In addition, many of the studies were conducted in areas having fairly low concentrations of ambient PM, with ranges in PM levels that covers California values. Thus, the extrapolation is within the range of the studies. Finally, the uncertainty in the C-R functions selected is reflected in the lower and upper estimates given in all the health impacts tables, which represent 95% confidence intervals. For premature death, this estimated error amounts to about a 50% difference from the mean value.

The C-R function used for quantification of death associated with ozone exposures is based on a review of all the published literature on the subject. As detailed in the CARB/OEHHA ozone standard staff report (CARB/OEHHA 2005b), the estimates for the effects of ozone on death reflect the range provided in several studies. Recently, three new meta-analyses conducted by three independent teams of researchers confirmed the validity of the chosen function (Levy 2005, Ito 2005, Bell 2005). Below, we detail some issues with choosing the C-R functions for ozone-related health impacts.

Potential confounding by daily variations in co-pollutants and weather is an analytical issue to be considered. With respect to co-pollutants, daily variations in ozone tends not to correlate highly with most other criteria pollutants (e.g., CO, NO_2, SO_2, PM10), but may be more correlated with secondary fine particulate matter (e.g., PM2.5) measured during the summer months. Assessing the independent health effects of two pollutants that are somewhat correlated over time is problematic. However, much can be learned from the classic approach of first estimating the effects of each pollutant individually, and then estimating their effects in a two-pollutant model. For this reason, we have emphasized use of ozone studies that have also controlled for PM.

The choice of the studies and concentration-response functions used for health impact assessment can affect the impact estimates. Because of differences, likely related to study location, subject population, study size and duration, and analytical methods, effect estimates differ somewhat between studies. We have addressed this issue by

emphasizing meta-analyses and multi-city studies, and also by presenting estimates derived from several studies. For ozone deaths, studies of short-term exposure and mortality have been replicated in many cities throughout the world, under a wide range of exposure conditions, climates and covarying pollutants. As a result, the evidence of an effect of ozone on premature mortality is compelling, especially with the recently published meta-analyses of the effect. Nevertheless, uncertainty remains about the actual magnitude of the effect and the appropriate confidence interval.

Finally, on the question of relative toxicity of diesel PM compared to PM2.5, in this assessment, staff assumed diesel PM is equally toxic as PM2.5. Without definitive evidence to include otherwise, this approach may underestimate the true effects of diesel PM exposures on adverse health effects.

4. Baseline Rates of Mortality and Morbidity

Mortality and morbidity baseline rates are entered into the C-R functions in order to calculate the estimates presented in this report, and there is uncertainty in these baseline rates. Often, one must assume a baseline incidence level to be consistent throughout the city or country of interest. In addition, incidence can change over time as health habits, income and other factors change. For this analysis, we used baseline rates that are used by U.S. EPA. Some of the rates were collected from Department of Health Services and Office of Statewide Health Planning and Development. It is expected that incidence rates may change over time. However, without any peer-reviewed information on projections of mortality and morbidity rates into the future, we opted to assume the current rates would remain and only adjusted future estimates for population shifts.

5. Health Effects from Sulfate Exposure

Emissions of sulfur oxides (SO_X) contribute to particle sulfate formation (and PM-related health effects) through complex chemical reactions and physical processes in the atmosphere. Stringent regulations on the sulfur content of motor fuels and stationary source controls have minimized SO_X emissions from most California sources. The largest uncontrolled fossil fuel sulfur source in California is the burning of residual oil as fuel in ocean-going vessels.

The December 2005 draft of this report did not include a quantitative health assessment of particle sulfate formed from goods movement-related emissions of SO_X. Any analysis is complicated by the fact that, in addition to sulfate formed from fossil fuel use in California, there are three other sources of atmospheric sulfate in California – natural “background” sulfate formed over the ocean by biologic activity, global “background” sulfate that is distributed throughout the Northern Hemisphere by the upper air westerly winds, and sulfate blown into Southern California from combustion in Mexico. New analyses of air quality and emissions data conducted in the intervening period indicate that uncontrolled SO_X emissions from ships increase the estimates of total goods movement-related health effects by about one quarter. However, this preliminary estimate contains several uncertainties, e.g., a considerable uncertainty associated with estimating ship emissions, and proper characterization of transport of transoceanic pollutants. Thus, a fully quantitative analysis must await the completion (by end of 2006) of research being jointly conducted by CARB staff, five university groups, the U.S. EPA,

and Environment Canada as part of a feasibility study for establishing a SO_X Emission Control Area (SECA) to reduce sulfur emissions from West Coast shipping. The research includes a refined inventory of ship activity and ship emissions, analysis of historical PM data from sites along the West Coast to look for evidence of ship emissions, development of new monitoring methods that can distinguish fossil fuel sulfate from that due to biologic activity in the ocean, and model development to allow simulation of sulfate formation and transport over the ocean and land areas of coastal California.

6. Unquantified Adverse Effects

An additional limitation in this analysis is that we did not quantify all possible health benefits that could be associated with reducing diesel PM and ozone exposure. Although the analysis illustrates that reduction in diesel PM and ozone exposure would confer health benefits to people living in California, we did not provide estimates for all endpoints for which there are C-R functions available. Unquantified health effects due to PM exposures include myocardial infarction (heart attack), chronic bronchitis, onset of asthma, and asthma attacks, as there is some overlap between these and the quantified effects such as lower respiratory symptoms and all respiratory and all cardiovascular hospitalizations. In addition, estimates of the effects of PM on premature births, and low birth weight, and reduced lung function growth in children are not presented. While these endpoints are significant in an assessment of the public health impacts of diesel exhaust emissions, there are currently few published investigations on these topics. Also, the results of the studies that are available are not entirely consistent. Nevertheless, there are some data supporting a relationship between PM exposure and these effects, and there is ongoing research in these areas that should help to clarify the role of diesel exhaust PM on these endpoints.

We recognize a multitude of endpoints that may contribute to impacting health. However, the weight of evidence to date was deemed insufficient to warrant quantification in our report. These include but are not limited to: psychosocial factors (stress), noise (including cardiovascular effects), light and its effects on sleep, major occupational issues including workplace exposures and injuries, traffic accidents and associated morbidity/mortality, other transportation related issues, and environmental consequences, quality of life, morbidity over extended periods of time, neurological disease, and developmental effects.

There is also evidence for other non-cancer health effects that are attributable to diesel exhaust PM exposure. For example, diesel PM apparently can act as an adjuvant in allergic responses and possibly asthma. However, additional research is needed at diesel exhaust concentrations that more closely approximate current ambient levels before the effect of diesel PM exposure on allergy and asthma rates is established. Also, because these endpoints have been investigated only in controlled exposure studies, population level C-R functions are not available for making estimates of the population-wide impacts of exposure.

Taken as a whole, the results of our limited analysis support the conclusion that reduction in emissions from Goods Movement will confer health benefits to the exposed population. However, since we did not make estimates for all possible endpoints, it is

likely that we have underestimated the health benefits in this analysis. Also, since we have been able to quantify all sources of uncertainty, the range behind our estimates is likely smaller than they should be.

7. Uncertainty Associated with Economic Valuation

The unit valuation for premature mortality, often referred to as the "value of a statistical life", is based on 26 studies (U.S. EPA, 1999). The estimates from these 26 studies fit a lognormal distribution with shape parameter, leading to an estimate of uncertainty. Similar data were available for Minor Restricted Activity Days. For the other health effects, we do not have a range in the unit valuation, so we were not able to calculate a quantitative estimate of the uncertainty in the unit valuation. Since the economic valuation of premature mortality, and uncertainty thereof, overwhelms the economic values of non-mortality effects, it was deemed appropriate to quantify the uncertainty associated with economic valuation behind mortality valuations only.

C. Ongoing Studies to Reduce Uncertainties

1. Emissions

There are a number of studies underway or planned for the near future which will improve our estimates of the emissions associated with ports and goods movement. For ocean-going ships, emission factors will be refined based on emission test data for propulsion and auxiliary engines. Emission testing of both bunker and marine diesel oil fired auxiliary engines is underway to provide better emission factors for ship auxiliary engines, based on type of fuel used. Emissions from ship boilers will be added into emissions inventory and information on anchorage emissions will be assessed for inclusion into emission inventory efforts. Emission testing of locomotives and ocean-going ships will be used as the basis for developing updates to size/speciation profiles for modeling efforts. For cargo handling yard trucks, emission testing of in-use vehicles equipped with diesel fueled off-road, on-road, and propane fueled engines are being performed to provide additional emission factor data. Data logging programs are underway to obtain better load factor information used in estimating emissions. CARB is participating with Starcrest Consulting Group, LLC programs to update emissions inventories for the Port of Long Beach and Los Angeles. Updated information from these inventories, such as equipment populations, activity, and load factors, will be used to refine CARB statewide emission inventories.

CARB is also working with the U.S. EPA, Environment Canada, and the Mexico National Institute of Ecology to assess the benefits of a SO_X Emission Control Area (SECA) designation. The overall goals of that work are to improve our understanding (i.e., reduce uncertainties) in the modeling of offshore transport and transportation of commercial marine vessels (CMV) emissions and to quantify the health and welfare impacts of CMV emissions using modeling and observation-based approaches. Several SECA projects are underway, including improved CMV emission inventories, air quality modeling efforts in the SoCAB and Central California, PM source apportionment, and ambient isotope analysis.

Work to improve emission estimates for other transportation sectors will also take place. Under the new 2005 Railroad Agreement, risk assessments will be performed over the

next 30 months at 16 rail yards throughout the state. CARB will receive detailed emission inventories (for both criteria pollutants and TACs) for all sources (mobile and stationary) at these facilities as part of this effort. The rail yards that will be included in this effort are identified in Attachment A of the Agreement, and generally represent the larger rail yards in the State. Another effort to improve the emission inventory for railroads will investigate the feasibility of using remote sensing technologies to measure emissions from locomotives. Assembly Bill 1222 requires CARB, in conjunction with the railroads, and the Sacramento Metropolitan and South Coast Air Quality Management Districts, to evaluate the feasibility of locomotive remote sensing. A report to the Legislature on the study will be prepared by December 31, 2006. Remote sensing, as it is being applied to locomotives, is a system that is designed to quantify in-use emissions as a locomotive passes a point along a track segment, and to ideally determine if that locomotive is operating within its emission certification levels. The intent would be to identify and tag for repair locomotives that have excessive emissions. The benefits of this program would be to reduce the number of "high polluting" locomotives in California service, but the anticipate emission reductions are unknown at this time as there is no estimate of what the population of high polluting locomotive baseline is. It is also unknown at this time if this technology will even work as described above, as it has not yet been demonstrated on locomotives.

Emissions from diesel trucks are a component of Goods Movement. Emissions associated with diesel engines are of great interest to CARB and for that reason, the Board co-funded an emissions test project, conducted under the auspices of the Coordinating Research Council (CRC). The project was recently completed. During this project, a total of 75 heavy-duty trucks (HDTs) were emissions tested over up to six test cycles. For a significant subset of these HDTs (about 30), two or three repeat tests of each test cycle were performed. In addition to mass emissions, a small subset also had chemical analyses performed, and a subset of these vehicles also had repeat emissions sampled for replicate chemical analyses. Analysis of these data will permit insights to be gained regarding the amount of variability or uncertainty associated with these emissions and chemistry data.

2. Exposure

Multiple studies are currently under way that will improve the characterization of emission sources related to Goods Movement and the associated the air quality impacts.

Regional air quality modeling is being conducted to address the 2007 Ozone SIP and the 2008 PM2.5 SIP. The best available emissions estimates from Goods Movement sources will be incorporated in these analyses. Under these SIP modeling projects, the impacts from these emissions can be evaluated on a regional basis throughout each of the SIP modeling domains.

Community Health Modeling is being conducted in the Wilmington region of Southern California using both regional and micro-scale modeling tools. These modeling studies include the best available emission estimates within and surrounding the Wilmington neighborhood, including the Ports of Los Angeles and Long Beach as well as emissions from trains and trucks. The dispersion of neighborhood-scale emissions within and

surrounding Wilmington will be simulated with a Gaussian plume dispersion model to evaluate near field impacts (i.e., resolved within a scale of hundreds of meters). The CalPuff air quality model will also be used to evaluate the impacts from sources, including Goods Movement sources, on areas further downwind from Wilmington (e.g., Los Angeles and Riverside). In addition, regional modeling of toxics will be conducted using the CAMx photochemical model within the SoCAB that surrounds Wilmington. These regional simulations account for the impacts of regional sources on air quality within the Wilmington neighborhood. A saturation monitoring study within Wilmington, including the use of passive monitoring techniques, is in the early planning stages and may provide a sufficient data set by which to assess model performance and micro-scale emissions inventory characterization.

As mentioned early, several SECA projects, including source apportionment and ambient measurements, are planned or underway to assess the impacts of ship emissions. The objective of these two projects is to quantify the contribution of ship emissions to ambient coastal PM using an advanced statistical technique (Positive Matrix Factorization) and a suite of instrumentation, including Aerosol time-of-flight mass spectrometers (ATOFMS) and isotope measurements, respectively. The outcome of these projects is expected to improve our exposure estimates attributed from ship emissions.

Studies on diesel PM emission sources in the Port of Los Angeles and the Port of Long Beach are underway. In addition, an analysis for diesel PM emissions from the port rail yard provides a good assessment of impacts near the rail yards. These studies represent a good first step in characterizing the magnitude of air quality impacts from these two major ports. Initial modeling has been conducted using a Gaussian plume dispersion model. This can be enhanced with a more advanced modeling tool, such as CalPuff (also to be used in the Wilmington study described earlier), to assess air quality impacts on larger, regional scale.

The Community Air Risk Evaluation (CARE) program was initiated by the Bay Area District in July 2004 and its goal is to evaluate health risk from air toxics in the nine Bay Area counties. The program includes enhanced air monitoring and analysis that will better determine the relative contribution of air pollution sources including vehicular and stationary emissions with an emphasis on diesel exhaust.

3. Health and Environmental Justice

Several on-going research studies in the SoCAB and the San Francisco Bay Area will provide more detailed information on the exposure and health effects of pollutants associated with goods movement. These projects include epidemiologic investigations of the potential health effects of particle pollution on vulnerable subjects such as the elderly, those at risk for cardiovascular disease, and children; and a series of projects and studies aimed at understanding the differential effects of air pollution exposure that may be experienced by economically disadvantaged populations living in communities surrounding goods movement facilities—specifically, port facilities or railroads.

CARB is co-sponsoring a study, along with the National Institute of Environmental Health Sciences and the South Coast Air Quality Management District, to determine how exposures to ultrafine and fine particles may impact the health of the elderly living

near traffic in Los Angeles. Investigators from the University of California at Irvine and Los Angeles as well as from the University of Southern California are monitoring heart function as well as biological markers of injury in elderly participants. Air quality measurements are being made both inside and outside the retirement homes under study. The elemental carbon content of local air is of special concern.

A study relating asthma to traffic-related pollution in Los Angeles neighborhoods will conduct NO_X and NO_2 monitoring at 200 locations within the Los Angeles (CARB 2005c). In the Los Angeles Family and Neighborhood Survey (L.A. FANS) study domain Land Use Regression models will be used to predict traffic pollutant (NO_X, NO and NO_2) exposures for all of the LA FANS subjects. These will be used to evaluate associations between traffic pollutant exposures and lung function and asthma (prevalence, exacerbation and possibly incidence) in children ages 0-17 years. This study will also use geostatistical models to estimate regional background concentrations of O_3 and $PM_{2.5}$ to evaluate whether concentrations of these more regionally distributed background pollutants confound or modify the effects of exposure (lung function and asthma) to the more heterogeneously distributed traffic-related pollutants (NO_X, NO, and NO_2). This study will provide information on respiratory impacts of motor vehicle emissions in a low socioeconomic status population and will aid in the development of air pollution exposure models that could be used in future epidemiological studies in L.A. County.

The “Teachers Cohort Study” (CARB 2005d) has the unique opportunity to use an existing dataset, the California Teachers’ cohort, established by the Northern California Cancer Center and the California Department of Health Services. This cohort includes 133,479 current and former female public school teachers and administrators recruited in 1995. Investigators have followed this population for incidence of disease and mortality. The information gathered will allow the investigators to determine whether long-term exposure to PM (PM10 and PM2.5) or gaseous pollutants is associated with cardiovascular and cardiopulmonary disease incidence or mortality. Investigators will also determine whether exposure to traffic emissions, measured by residential proximity to busy roads, is related to cardiovascular disease incidence or mortality.

In order to assess community impacts of goods movement—the CARB has several projects underway that will build on recently completed emissions inventory and modeling studies conducted in the Wilmington port area. The primary studies are: *Investigation and Characterization of Pollution Concentrations Gradients in Wilmington, CA Using a Mobile Platform* (CARB 2005e), and, *Environmental Justice Saturation Monitoring of Selected Pollutants in Wilmington* (CARB 2005e).

The overall objective of the first study is to generate a vehicle-related pollutant gradient grid for Wilmington. The project will acquire a non-polluting vehicle and outfit it with a set of real-time instruments capable of measuring key variables and pollutants of interest. These pollutants include utrafine particles, PM2.5, CO and CO_2, oxides of nitrogen and black carbon. The main study phase of the project will conduct mobile platform measurements in the warm and cool seasons in and around Wilmington and investigate the identified pollution gradients as a function of traffic volume and composition, meteorological factors and weekday versus weekend influences. This information will be used to identify suitable locations for fixed site, passive monitors in the second study conducted by the Desert Research Institute (DRI). This DRI

“saturation monitoring”study will investigate the previously identified pollution gradients in the Wilmington area and examine how such gradients are affected by key variables. Investigators will also obtain data relevant to resolving the relative importance of local point sources versus traffic-generated emissions versus transported background pollution. This study will also test the use of passive monitors for conducting field measurements. The pollutants to be measured will include, O_3, NO, NO_2, NO_X, SO_2, BTEX (benzene, toluene, ethylbenzene, xylenes), formaldehyde, acrolein and odor-causing sulfides. In the initial phase of this study the precision, accuracy, sampling rates and validity of passive sampling methods will be tested in the laboratory using a flow through chamber with known pollutant concentrations. Combined, these studies have as their objectives: to assess the Wilmington community’s air quality concerns and identify “hot spots”; develop and test methods to validate existing air emissions inventory and pollutant concentration modeling, and, to develop tools for community-scale monitoring of pollutants for identification of exposure gradients.

Two recently approved research studies taking place in the Los Angeles area will provide additional information for assessing exposure to utrafine particle pollution: *Fine-Scale Spatial and Temporal Variability of Particle Number Concentrations within Communities and in the Vicinity of Freeway Sound Walls* and *Ultrafine Particle Concentrations in Schools and Homes (*CARB 2005g)*.*

In the San Francisco Bay region CARB is sponsoring an investigation to determine whether socioeconomic variables are related to differential air pollution exposures. This study: *Air Pollution and Environmental Justice: Integrating Indicators of Cumulative Impact and Socioeconomic Vulnerability into Regulatory Decision Making* (CARB 2005i) has, as one of its primary objectives, to provide CARB staff with a “concrete tool” to integrate cumulative impact and risk measures with community vulnerability factors (socioeconomic measures). The study area for this project is the San Francisco East Bay, primarily the highway 880 corridor. This environmental justice study will also conduct a state-wide analysis of patterns of racial and ethnic disparities in cancer and other health risks associated with outdoor air pollution.

The project will integrate a wide range of data from federal, state, and air district sources, as well as a local-scale study to (a) address methodological challenges in assessing cumulative exposure, (b) develop and test a dual model which accounts for environmental and socio-economic conditions, (c) incorporate analysis of spatial auto-correlation to improve predictive power and experiment with differing scales of analysis, (d) incorporate community meetings and community-based participatory research in order to enhance community confidence, and (e) develop screening measures that can be used to guide regulatory action and community outreach. The local-scale study will incorporate community-based researchers utilizing geo-positioning devices to identify local air toxics emitters. A screening tool will be developed to identify communities that may be vulnerable due to SES and environmental conditions.

Many of the known biological responses associated with air pollution exposures could potentially alter an individual’s risk of getting a disease or influence the way an existing disease progresses. For example, even though the evidence that air pollution causes asthma is only beginning to emerge (McConnell et al*.* 2002), air pollution is known to induce asthmatic episodes in people with the disease. Repeated episodes of asthma

may damage or alter the respiratory tract of asthmatics, leading to worsening of the disease and a poorer quality of life. The Fresno Asthmatic Children's Environment Study (FACES) was designed to evaluate observations of elevated childhood asthma in Fresno. Fresno was selected because it is the largest population center in the San Joaquin Valley, with high 24-hour-average PM2.5 (160 $\mu g/m^3$) and PM10 (199 $\mu g/m^3$) concentrations and the second and third highest asthma hospitalization rates in California for black and Hispanic children, respectively. Health scientists have established that asthma sufferers have more breathing problems when PM is high and that children exhibit more asthma problems than adults do. Investigators at the University of California at Berkeley, the California Department of Health Services, private consultants, and the CARB developed an epidemiologic field investigation to determine how young children known to have asthma are affected by various environmental and lifestyle factors on a day to day and longer term basis. FACES includes 44% Hispanic, 14% black, 2% Asian, and 19% low-income families (less than $15,000 household income) among the approximately 300 participants. The study is anticipated to continue until 2007.

The Children's Health Study (CHS), which began in 1992, is a long-term epidemiologic study of the health effects of children's chronic exposures to southern California air pollution. About 5500 children in 12 communities have been enrolled in the study; two-thirds of them were enrolled as fourth-graders. The CHS includes 28% Hispanic, 5% black, and 5% Asian among its participants. Data on the children's health, their exposures to air pollution, and many factors that affect their responses to air pollution are gathered annually. Concentrations of pollutants have been measured in each community throughout the study and for brief periods in schools and some homes. In addition, each child's lung function is tested every spring. Annual questionnaires ask about the children's respiratory symptoms and diseases, such as chronic cough and asthma; level of physical activity; time spent outdoors; and many other factors known to influence children's responses to air pollution, such as parental smoking and mold and pets in the household.

4. Economics

Information on the health benefits of regulatory programs is necessary for accurate economic assessment. Currently, several adverse health outcomes associated with exposure to air pollution have been demonstrated. However, the economic benefits of reducing many adverse health outcomes have not been characterized. In response, the CARB is actively engaged in economic research that will improve its ability to accurately quantify the health benefits of reducing exposure to outdoor air pollution.

The last comprehensive assessment of health benefits of air pollution reductions in California was completed in 1986 and is outdated. Although South Coast and San Francisco Bay Area districts have quantified health benefits for their plans to meet air quality standards, many of the underlying health benefits studies that these analyses are based upon are more than a decade old. In addition, there are significant gaps in the economics literature that have not yet been addressed. Recent work funded by CARB to develop new estimates of economic value for reducing hospitalizations provides useful new information for such assessments, but there are several important remaining gaps in the literature.

Recent health effects research points toward air pollutants as risk factors for the onset of several chronic respiratory and cardiovascular illnesses. These include cardiovascular disease, asthma, and permanent lung function decrements. Willingness-to-pay (WTP) estimates are available in the economics literature only for reducing risks of onset of chronic bronchitis (Viscusi et al. 1991).

One CARB-supported study, "Economic Value of Reducing Cardiovascular Disease Morbidity Associated with Air Pollution" will make an important contribution to better quantifying the health benefits of air pollution control in California, because there are no WTP estimates, or even very good COI (cost-of-Illness) estimates, for lifetime cardiovascular disease (CVD) morbidity. The study team will design, implement and analyze a WTP survey that develops a monetary estimate of individual WTP to reduce the risk of developing cardiovascular disease.

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