APPENDIX A

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

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Table of Contents

Executive Summary

The California Air Resources Board (CARB) staff assessed the potential health effects associated with exposure to air pollutants arising from port-related goods movement (i.e., ships, ports, trains, trucks) and other port activities (e.g., commercial fishing vessels) 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 port-related goods movement, and provides an economic valuation of these health effects. Because of the uncertain nature of several key inputs and methodologies, these results are a first estimate that will be refined over time.

Background

Port-related emission sources, which are mostly diesel engines, emit PM directly (i.e., diesel PM) and form additional PM (i.e., particle nitrates, particle sulfates) through chemical reactions and physical processes in the atmosphere involving emitted nitrogen oxides (NO_X) and sulfur oxides (SO_X). Emissions of NO_X and reactive organic gasses (ROG) 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, asthma attacks, 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) with an uncertainty range of 3,000 to 15,000. 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 $4 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 $57 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 port-related goods movement was estimated from scaling factors (based on measurements and air quality modeling) linking air basin-wide emission inventories

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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 previous estimates. The U.S. EPA also uses this study (e.g., see http://www.epa.gov/interstateairquality/pdfs/finaltech08.pdf).

of diesel PM, NO$_X$, and ROG to outdoor levels of PM components (diesel exhaust, particle nitrates) and ozone. A similar analysis for particle sulfates formed from SO$_X$ emissions was not possible and a quantitative estimate must await technical analyses being conducted by CARB, five university groups, Environment Canada, and the U.S. Environmental Protection Agency (EPA) and its contractors, due next summer.

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 value of eliminating these adverse health effects, due mostly to avoided premature deaths but also to savings in health care expenditures, is also shown. Particle nitrate accounts for 60% of the risk. Since it takes several hours to form this pollutant from NO$_X$ emission sources, risks are more uniformly distributed over an air basin than from diesel PM (35%), which is highest for those living closest to the sources. The South Coast Air Basin dominates the risk (70%), 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.

Table A-1 Annual (2005) Statewide PM and Ozone Health Effects Associated with Port-related Goods Movement and Other Port Activities in California[1]				
Health Outcome	Cases per Year	Uncertainty Range[2] (Cases per Year)	Valuation (million)	Uncertainty Range[3] (Valuation - million)
Premature Death	750	260 to 1,300	$6,200	$2,100 to 12,000
Hospital Admissions (respiratory causes)	290	170 to 410	$10	$6 to 14
Asthma Attacks	14,600	3,600 to 26,000	$1	$0.1 to 1.9
Work Loss Days	130,000	110,000 to 150,000	$23	$19 to 26
Minor Restricted Activity Days	880,000	630,000 to 1,100,000	$53	$25 to 110
School Absence Days	330,000	85,000 to 610,000	$28	$7 to 53
TOTAL VALUATION	N/A	N/A	$6,300	$2,200 to 12,000

[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 concentration-response functions, but not in emissions or exposure estimates.

[3]Range reflects statistically combined uncertainty in concentration-response functions and economic values, but not in emissions or exposure estimates.

Projecting future population and goods movement emissions growth and control (for already adopted measures) to the year 2020 results in 920 (320 to 1,600 uncertainty range) deaths per year, and an estimated annual value (in 2005 dollars) of $3.1 to 5.5 ($1.0 to 11) billion. The year 2020 mitigation strategies presented in the main report are expected to result in a reduction of 500 (180 to 890) deaths annually, with an estimated value of $1.7 to 3.0 ($0.6 to 5.8) billion. Results from estimates of cumulative health impacts and an economic valuation from 2005 to 2020 will be given in the next version of this assessment.

Peer 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 this assessment and have agreed to review this document in parallel with the public review. Their comments will be presented and, to the extent possible, incorporated into the next version of this assessment.

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 incidences of hospitalizations for exacerbation of heart disease, chronic lung diseases (i.e., asthma and chronic obstructive pulmonary disease), and acute lung diseases (i.e., pneumonia and acute bronchitis). In addition, estimates of the effects of PM on infant mortality, premature births, low birth weight, and reduced lung function growth in children are not presented. While these outcomes are 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. 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 SO_X Emission Control Area (SECA) designation for North America will improve estimates for particle nitrate, particle sulfate, and ozone during 2006. Over the next 30 months, CARB 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, nitrates, sulfates) 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-related emission categories. Of these, 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 operate at California ports and transport international 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 400 tons per day of NO_X are emitted from ports and international goods movement activities in California. NO_X emissions from ports and international goods movement lead directly to formation of ozone PM and represent about 10 percent of the total statewide NO_X emissions inventory. Sixty tons per day of SO_X were generated by ports and international 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 18 tons per day of PM and represented about 20% of the statewide diesel particulate inventory.

Table A-2 2001 Statewide Pollutant Emissions by Goods Movement Source Type[1]
(Tons per Day)

Pollutant	Ships	Harbor Craft	Cargo Handling Equipment	Trains	Trucks and TRU	Total
ROG	3	9	3	5	14	**34**
Diesel PM	8	4	1	2	3	**18**
SO$_X$	59	1	<1	3	2	**65**
NO$_X$	94	86	21	77	129	**407**

[1]Transportation Refrigeration Units (TRUs) were not considered in the health impacts assessment because staff determined the fraction of TRU emissions related to goods movement could not be accurately evaluated in time for release of this report, and will be addressed in future assessments.

Over the next several decades, the amount of goods imported into or moved through California is projected to increase dramatically. This will result in increased goods movement through all ports in California, but most of this increase is expected to be borne by the Ports of Los Angeles, Long Beach, and Oakland. As imports increase, more ships will enter the ports, more cargo handling equipment will move imported goods, and more trucks and trains will transport goods to their final destinations. This growth will have a major impact on southern California and the State as a whole.

CARB estimates growth in each category of the ports and international goods movement emissions inventory. Growth estimates are based on expected growth in economic and equipment-specific factors relevant to each source category, which are affected by the expected growth in imported goods over time. Figure A-1 provides goods movement emissions estimates by pollutant and by year for 2001-2025. By 2025, diesel PM emissions are projected to double and SO$_X$ emissions are projected to triple. NO$_X$ emissions are projected to increase more than 10 percent by 2025, primarily in areas that are currently not in attainment with air quality standards.

Figure A-1 Statewide Ports and International Goods Movement Emissions

Appendix A-11
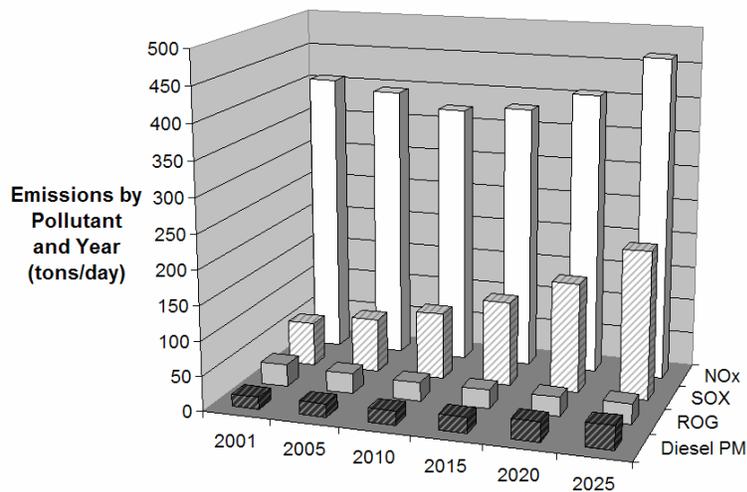
California has four major goods movement corridors: (1) the Los Angeles-Inland Empire Region, (2) the Bay Area Region, (3) the San Diego / Border Region, and (4), the Central Valley Region. Regions like Los Angeles and the Bay Area are major centers of goods movement because they contain the largest ports in California. In particular, the Los Angeles 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 Central Valley is a major corridor 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 four regions.

Table A-3 2001 Ports and International Goods Movement Related Emissions Released Over Land by Corridor Region (tons/day)

Region	ROG	Diesel particulate	NO_X	SO_X
South Coast	13	4	130	8
San Francisco	4	2	50	3
Central Valley	4	2	60	1
San Diego / Imperial	2	1	20	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 draft 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. Non-cancer health effects and economic losses were estimated to include 29 premature deaths, 750 asthma attacks, and over 6,500 days of lost work each year, although these have been updated with new peer-reviewed studies and more recent concentration-response functions, an extended modeling domain, and additional pollutants for the statewide goods movement risk assessment. 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.

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 (Lloyd and Cackette 2000). PM 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. This discussion of pollutants of concern for goods movement focuses primarily on PM and ozone. These are the two pollutants for which there is sufficient evidence of the health effects, including estimations of premature mortality, thereby permitting calculations of the adverse impacts. The primary studies relied upon for estimating the health effects of particulate matter use ambient PM2.5 concentration estimates as the measure of exposure. A more general discussion of the health effects of direct diesel emissions, or diesel PM, and associated PM-related pollutants such as sulfates and PAHs provides some background for understanding the toxicity of diesel PM relative to other hazardous air pollutants.

One of the goals of this analysis is to estimate the economic impacts associated with health impacts related to goods movement. From the economic standpoint, premature mortality from PM2.5 and ozone is by far the greatest concern. Additional health endpoints with potentially high economic valuation that were assessed are:

- o Hospital admissions for respiratory diseases from ozone
- o Asthma attacks from PM
- o Work loss days from PM
- o Minor restricted activity days from PM and ozone
- o School absence days from ozone

Although it is possible to link many cancer and non-cancer health effects directly to changes in pollutant concentration, there are additional health effects from PM, ozone, and other pollutants that cannot yet be easily quantified, or for which a comprehensive evaluation must be undertaken to understand their associations with pollutant concentrations. In general, the unquantifiable impacts that have been identified are associated with much smaller effect sizes and therefore have a lesser economic impact. This does not, however, lessen their importance and additional resources must be acquired to adequately characterize these health and community impacts in order to fully evaluate the effects of increased goods movement. Identified, but as yet unquantifiable health effects, are listed in Table A-4 along with the health endpoints that were examined in this report. It is important to note that some of these health effects (i.e., adverse birth outcomes, immune effects, atherosclerosis) are measured at current ambient levels of PM.

Table A-4 Summary of the Health Effects of PM and Ozone

Effect	Identified		Quantified	
	PM	Ozone	PM	Ozone
Premature Death				
All-cause mortality	X	X	X	X
Cardiopulmonary[2]	X	X		
Coronary heart disease[2]	X	X		
Lung cancer[2]	X	X		
Respiratory Effects				
Asthma attacks	X		X	
New cases of asthma[3]	X	X		
Increased respiratory symptoms[4]	X	X		
Chronic bronchitis[4]	X			
Increased hospitalization for respiratory disease		X		X
Reduced lung capacity (adults)[5]	X	X		
Decreased lung function in children[5]		X		

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[2] This health endpoint is included in the all-cause mortality estimate.
[3] New cases of asthma related to ozone, see: McConnell *et al.* 2002; PAHs (PM) linked to asthma in newborns (Miller *et al.*, 2004); NO_2 linked to new asthma in Gauderman *et al.*, 2005.
[4] Some portion of these effects may be captured by the estimate of increased hospitalization for respiratory disease.
[5] There is insufficient epidemiologic evidence to quantify these effects.

Effect	Identified		Quantified	
	PM	Ozone	PM	Ozone
Cardiovascular Disease (CVD)				
Increased hospitalization for CVD[6]	X			
Underlying CVD (atherosclerosis)[5]	X			
Other Effects				
Cumulative health impacts[4]	X			
Birth outcomes[6]	X	X		
Infant mortality[6]	X			
Minor restricted activity days	X	X	X	X
Neurotoxicity[5]	X			
School absences		X		X
Work loss days	X		X	

1. Particulate Matter

Airborne particles less then 0.1 µm in aerodynamic diameter are often referred to as ultrafines. Particles between 0.1 µm 2.5 µm in aerodynamic diameter are classified as fines, and particles 2.5 µm to 10 µm are considered the "coarse fraction" of PM. PM is monitored throughout the state based on its size distribution: PM2.5 or fine PM (particles less than 2.5 µm in diameter) or PM10 (particles less than 10 µm in diameter). Direct diesel emissions are generally discussed in terms of gaseous and solid or particulate matter (PM) phases.

The particles in diesel emissions are very small (90% are less than 1 µm by mass) and have hundreds of chemicals adsorbed onto their surface, many of which are known or suspected mutagens (capable of causing gene mutations) or carcinogens. 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). The exhaust of diesel engines has been found to contain 15 substances listed by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, or as a probably human carcinogen. In 1998, CARB identified diesel PM as a toxic air contaminant (TAC). A health risk assessment was completed by the OEHHA (1998). This document characterized the potential for diesel exhaust to affect human health and was largely based on epidemiologic studies associating airborne particles with health risks.

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[6] Estimates could be generated after extensive review of the epidemiologic literature.

The health effects of PM can be divided into short-term (acute) effects or long-term (chronic) non-cancer effects. Mortality is considered both a short-term effect and a long-term effect and a considerable number of studies have demonstrated an increase in premature death associated with both time variables. These mortality studies have generally attributed deaths to either all-cause, cardiopulmonary disease, or lung cancer. Recent evidence attributes a greater portion of these deaths to cardiovascular disease. The other short-term effects of PM include acute respiratory symptoms (aggravated cough or difficult or painful breathing). Other chronic effects include respiratory symptoms such as bronchitis and decreased lung function, aggravation of asthma, and neurotoxic effects.

a) Premature Death

Numerous epidemiologic studies have investigated the role of PM in premature death due to cardiopulmonary disease and lung cancer. These studies make it possible to attribute estimated mortality to changes in pollutant concentration. Pope et al. (1995) examined mortality and long-term exposure to ambient PM2.5 in the largest of these studies conducted between 1982 and 1989. National ambient air pollution data in 151 U.S. metropolitan areas was linked to individual risk factors and vital status data for over 500,000 adult members of an American Cancer Society (ACS) cohort. The relationship between air pollution (sulfates and PM2.5) to all-cause, lung cancer, and cardiopulmonary mortality were examined using multivariate analyses which controlled for smoking, education, and other risk factors. A 17% increased risk for all-cause mortality was found for a 24.5 µg/m^3 PM2.5 concentration difference between the most polluted city versus the least polluted (Pope et al. 1995). Krewski et al. (2000) performed a reanalysis of this Pope et al. (1995) study using the annual mean PM2.5 concentration rather than the median. The annual mean is more affected by high PM values than the median, and if high PM concentrations are more important in causing premature mortality, then this value is preferred. Relative risks for mortality determined for this study were also calculated based on the difference in the mean concentrations between the least and most polluted cities. An increased risk of 12% for all-cause mortality was found for a 24.5 µg/m^3 difference in PM2.5 (Krewski et al. 2000). The estimates of mortality from this study have been used as the coefficient for the concentration-response function in previous analyses to determine the potential health effects of PM2.5 pollution in California populations (CARB 2005a).

Pope et al. (2002) updated information on the original ACS cohort. This study doubled the follow-up time to more than 16 years and tripled the number of deaths; it also substantially expanded the exposure data, improved the control of occupational exposures, incorporated other lifestyle factors, and used recent advances in statistical analysis. A 6%, 8% and 13% increased risk for all-cause, cardiopulmonary and lung cancer mortality, respectively, was identified for each 10-µg/m^3 difference in fine particle concentration (Pope et al. 2002). Measures of coarse particle fraction were not associated with increased risk of mortality.

A very recent analysis done by Jerrett et al. (2005) using data from the California residents of the same ACS cohort analyzed by Pope et al. (1995, 2000) and Krewski et al. (2000) found much higher relative risks for all three mortality measures based on a 10-µg/m^3 difference in fine particle concentration; all-cause mortality was estimated at

17%, cardiopulmonary at 12%, and lung cancer at 44%, after adjusting for 44 covariates. These estimates are nearly three times larger than the Pope et al. (2002) estimates and provide new evidence on the specificity of health effects, which correspond convincingly with new findings related to hypothesized pathophysiological pathways linking ambient PM2.5 to cardiovascular disease mortality. Jerrett et al. (2005) arrived at an estimate of 39% increased risk for ischemic heart disease per 10 µg/m^3 PM2.5 concentration after adjustment for all covariates. Pope et al. (2004) also performed additional statistical analysis on the original ACS cohort and found that long-term PM2.5 exposures were more strongly associated with ischemic heart disease (22% increased risk per 10 µg/m^3 PM2.5), dysrhythmias, heart failure, and cardiac arrest.

The Jerrett et al. (2005) study does suggest that intraurban exposure gradients may be associated with higher mortality estimates than previously supposed and 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). These new estimates, once confirmed, may be particularly relevant to areas experiencing higher exposures due to goods movement. Measures of exposure to ambient PM2.5 in the Jerrett et al. (2005) study were very different than those used previously by Pope et al. (1995, 2002); incorporating new concepts in spatial analysis to estimate individual exposure. Further studies to confirm the results of this study are warranted.

While diesel PM is not directly measured in any of these mortality studies, an examination of various sources of particulate matter was examined in relationship to the mortality estimates made in a study by Dockery et al. (1993), known as the Six-Cities study. This study by Laden et al. (2000) used the elemental composition of size-fractionated particles to identify several distinct source-related fractions of particles: mobile source, coal-combustion, and soil and crustal matter. These fractions were then associated with the daily mortality estimates to produce a meta-analysis of the overall relative risks for each source fraction. Mobile sources were associated with the strongest increase in daily mortality with an increase of 3.4% observed for each 10 µg/m^3 increase in the mean of the mobile source factor. Furthermore, the increased mortality was found to be associated with ischemic heart disease; there was a 2% increase in daily mortality from ischemic heart disease found in association with each 10 µg/m^3 increase in the mean of the mobile source factor. Coal combustion was associated with a 1.1% increase in mortality, while there were no associations found for crustal material (Laden et al. 2000).

Several epidemiologic studies have examined the effects of other chemical-specific constituents of PM, including sulfates, nitrates and metals, and their associations with premature mortality. For example, in a study in Santa Clara County, Fairley (1999) examined the impacts of nitrates, sulfates, and COH (coefficient of haze). The latter is highly correlated with elemental carbon, and is likely to be a good marker of pollution from motor vehicles (especially diesel exhaust) and of wood smoke. All of these constituents of PM2.5 were associated with all-cause mortality, while nitrates were also associated with cardiovascular mortality. These findings were consistent with those in the Netherlands, where associations were reported for sulfates, nitrates, and black

smoke (Hoek et al. 2000). In a study in Buffalo, Gwynn et al. (2000) reported effects on total mortality for COH, sulfates and hydrogen ion, a measure of aerosol acidity. Lippmann et al. (2000) did not find associations of mortality with sulfate or hydrogen ion in Detroit, although only limited data for these pollutants were available. In their study of the eight largest Canadian cities, Burnett et al. (1997) examined the impact of 47 separate elements within PM2.5. Among the constituents in the fine PM fraction, sulfates, zinc, nickel, and iron were all associated with mortality, as was COH.

Relatively few studies on the human health effects of ambient ultrafine particles have been completed or confirmed. Wichmann et al. (2000) examined the effects of PM2.5 mass as well as ultrafine particles (0.01 to 0.1 µm) for the small German city of Erfurt. The number rather than the mass of ultrafine particles was used as the exposure measure. For this study, three different size classes of ultrafines were measured, including 0.01 to 0.03 µm, 0.03 to 0.05 µm, and 0.05 to 0.1 µm. The authors reported that both PM2.5 mass and several measures of ultrafines were associated with daily mortality.

b) Cardiovascular Disease

The evidence associating particle pollution to cardiovascular disease was recently summarized by Brook et al. (2004). Much of this evidence points to a pulmonary-systemic oxidative stress pathway as the biologic underpinning for the short- and long-term cardiovascular effects of PM. Particles may also act directly on the cardiovascular system through systemic absorption via the pulmonary epithelium. Gases, soluble constituents of PM2.5 (transition metals), and ultrafine particles are generally thought to follow the latter pathway and represent a plausible explanation for the occurrence of rapid or short-term effects such as increased myocardial infarction (heart attacks), as seen in time-series studies. Thrombotic events have also been shown to be triggered by ultrafine particles (Schultz *et al*., 2005).

Less acute, chronic and indirect cardiovascular effects such as atherosclerosis may occur via the pulmonary oxidative stress/inflammation pathway (Brook et al. 2004). Oxidative stress occurs after exposure to diesel exhaust particles (Brown et al. 2000, Shukla et al. 2000) and ambient PM2.5 (Sorensen et al. 2003). Personal exposure to PM2.5 is also associated with increased levels of markers for lipid and protein oxidation in human blood (Sorensen et al. 2003). There is significant evidence that the mechanisms underlying atherosclerosis, the strongest risk factor for heart disease, involve inflammatory responses and oxidative stress triggered by PM pollution (Li *et al*. 2003, Libby *et al*. 2002, Dick *et al*. 2003, Stone 2004). Fine particulate matter has also recently been associated with increased atherosclerosis in humans (Kunzli *et al.* 2004), and in animals (Suwa *et al.* 2002).

Exposure to PM increases fibrinogen, a major factor in blood viscosity. Blood viscosity has been associated with the severity of cardiovascular disease and may increase with ambient levels of total suspended particles or sulfur dioxide (SO_2). Fibrinogen, an inflammatory factor, is also an important independent risk factor for myocardial infarction and stroke. Epidemiologic data associate blood coagulation with particle pollution; and enhanced platelet activation and thrombosis formation are associated with exposure to diesel exhaust and ultrafine particles (Brook et al. 2004).

c)   Lung Cancer

The PM mortality studies discussed above include estimations of premature deaths associated with lung cancer. The original Pope et al. (1995) study found a 36% increase in risk for lung cancer associated with a 19.9 µg/m^3 difference in sulfate concentrations between the most polluted and least polluted cities. A much smaller estimate of 0.03% was arrived at for changes in PM2.5 concentration equal to 24.5 µg/m^3, with 95% confidence intervals suggesting the risk could be non-significant. In an update of this study, adjusted relative risks indicated a 14% increased risk (estimated range 4 to 23%) for lung cancer mortality (Pope et al. 2002). Fairley (1999) found a statistically significant 15% excess risk for respiratory mortality in association with sulfates in Santa Clara County, which is very similar to the estimate of Pope et al. (1995) estimates. PM2.5 estimated excess risk (non-significant) was 13% (confidence estimates not provided). Jerrett et al. (2005) found much higher excess risk for lung cancer in California residents of the SoCAB. These investigators identified a 60% increased risk for lung cancer; however, after adjusting for 44 covariates the estimate fell to 44% and the 95% confidence intervals surrounding this estimate were wide, indicating the risk could be non-significant in this smaller cohort.

Increases in lung cancer have been identified in most studies of groups occupationally exposed to diesel exhaust. PAHs may play a primary role in the etiology of lung cancer associated with PM. PAHs are adsorbed to the surface of PM and a number of these compounds are listed as carcinogens (CARB 1997). 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). Based on these studies and an estimated ambient concentration of diesel PM for which most Californians are exposed (1.54 µg/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).

d)   Acute and Chronic Effects

Several hundred epidemiologic studies have reported associations between various measures of PM and a range of health outcomes. The health outcomes associated with PM include hospitalization for cardiovascular or respiratory disease, emergency room and urgent care visits, asthma exacerbation, acute and chronic bronchitis, restriction in activity, work loss, school absenteeism, respiratory symptoms, and decrements in lung function. The most recent evidence associated with direct diesel PM is discussed below.

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. In addition, it has been shown in numerous studies that diesel exhaust particulate matter can enhance allergic asthma (Diaz-Sanchez 1999, 2000). The role diesel PM plays in the increasing prevalence of asthma and other allergic respiratory diseases is thought to be associated with its immunologic effects. Diesel exhaust exposure can result in measurable increases in antibody production and

other immune responses, especially when exposure is accompanied by known allergens (OEHHA 1998).

Many studies have looked at the enhancement of the allergic response following intranasal instillation of diesel exhaust particles that may cause people to become allergic to airborne substances that they would not otherwise be allergic to (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). Other products of combustion, including PAHs and dioxins, affect the immune system. Prenatal exposure to PAHs has been found to result in increased respiratory symptoms and probable asthma in children from an urban cohort (Miller *et al*. 2004). The levels of exposure to substances that can affect the immune system currently cannot be quantified. Short-term exposure directly to diesel exhaust has also been shown to enhance airway responsiveness (Nordenhall *et al.* 2001). Urban particulate is associated with asthma exacerbation in numerous studies (Ostro *et al*. 1995, 2001, Delfino *et al*. 2002). These immunologic effects can have a very significant health impact due to the large number of individuals in urban areas with respiratory allergies and asthma. To-date the only respiratory impact that can be quantitatively linked to PM concentrations is number of asthma attacks.

Concerns are emerging about potential neurotoxicity from ultrafine particulate matter. It has been known for some time that very fine particles can cross membranes readily, including nervous system tissue. Recent studies on inhaled ultrafine particles indicate widespread distribution following exposure including into the brain cells (Oberdoerster *et al*. 2005). Oxidative stress was induced in the brain of fish exposed to nanoparticles in water (Oberdoerster 2004). Mice exposed to concentrated airborne particulate matter had elevated inflammatory markers in the brain compared to controls (Campbell *et al*. 2005). Thus, there is a real possibility of neurotoxicity from ultrafine particles.

2. Ozone

Ozone is regulated in California as a criteria air pollutant. In April of 2005, 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 determined as 0.09 ppm. Ozone is a powerful oxidant that can damage the respiratory tract, causing inflammation and irritation.

The inflammatory effect of ozone may be responsible for the links between exposure and increases in premature death. In a recent study completed by Chen *et al*. (2005), two-pollutant statistical models that included ozone or SO_2 concentrations and PM2.5, found increased risk for fatal coronary heart disease in women. Ozone has been found to increase the permeability of lung epithelials (Bloomberg *et al*. 2003) — thus increasing susceptibility to particulate matter. This finding adds to the growing evidence that initiation of pulmonary and systemic oxidative stress and inflammation by components of the different PM particles may be the primary pathway for increased cardiovascular effects (Brook *et al*. 2004). Daily time-series studies of counts of all-

cause mortality have been reviewed over several years. Several meta-analyses (Anderson 2004, Levy 2001, Stieb 2003) have been used to arrive at an estimate of the affect on premature death due to ozone exposure. These studies are discussed in the methodology section below.

Hospital admissions for respiratory disease have been linked to ozone concentrations in daily time-series studies of hospital discharge diagnoses related to circulatory or respiratory diseases. Burnett *et al.* (2001) investigated respiratory hospitalizations in children under two years of age and found a 7.8% increase per 10-ppb change in five-day moving averages of 1-hour daily maximum concentrations. Emergency room visits for pediatric asthma have been studied by numerous investigators (Tolbert *et al.* 2000, Friedman *et al.* 2001, Jaffe *et al.* 2003, Romieu *et al.* 1995, Stieb *et al.* 1996). Restricted activity day estimates for adults are derived from a sample of an adult working population. More details on these studies and the point estimates and confidence intervals used for the concentration response functions can be found below and in the Ozone Standard Review Report (CARB 2005b).

Ozone can induce respiratory symptoms such as coughing, chest tightness, shortness of breath, and exacerbation of asthma symptoms. The greatest risk from ozone is to those who are more active outdoors during smoggy periods, such as children, athletes, and outdoor workers. Exposure to levels of ozone above the current national ambient air quality standard can lead to lung inflammation and lung tissue damage, and a reduction in the amount of air inhaled into the lungs (CARB 2005b). Recent evidence has, for the first time, linked the onset of asthma to exposure to elevated ozone levels in exercising children (McConnell 2002). To date, there is insufficient epidemiologic evidence to quantify the effect of ozone on new cases of asthma.

3. 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. 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. 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). 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

Given more time and resources, modeling approaches using CALPUFF and/or CMAQ to estimate particulate matter (PM) and ozone concentrations associated with all goods movement sources would be appropriate. However, given the short time frame to generate health and economic impact estimates, modeling is not an option except for the ports of Los Angeles and Long Beach, which have already been completed. Thus, this assessment employs exposure and health risk methodology for diesel PM and particle nitrates (Lloyd and Cackette, 2001), modified to a region-by-region approach, with the addition of similar methodologies for particle sulfates which did not succeed and ozone. Health endpoints used in the PM (CARB and OEHHA 2002) and ozone (CARB and OEHHA 2005) standard reports will be included, and annual impacts for 2005, 20010, 2015 and 2020 were calculated. An economic valuation of the health impacts was performed using the same methods employed for airborne toxic control measures (ATCMs) by CARB.

To correct for potential inconsistencies between exposure and emissions where emissions are not distributed uniformly in urban areas, adjustment factors for diesel PM emissions sources located in the outer continental shelf. This correction is assumed not to be necessary for secondary pollutant precursors (VOC, NO_X, and SO_X)

Since the health and economic impacts estimates were developed have large uncertainties, 5th and 95th percentile confidence bounds based on an integrated analysis of uncertainties in human health concentration-response functions and the economic values are provided. While including uncertainty due to emissions and exposure estimated is desirable, a quantitative assessment is not available and qualitative descriptions are provided.

A. Air Pollutant Emissions from Specific Port-related Sources

CARB maintains a comprehensive statewide emissions inventory that is used to assess the relative importance of air pollutant sources, and as a planning tool to gauge the effectiveness of air pollutant emission reduction control strategies. Goods movement emissions are generated by mobile sources. CARB emissions inventories provide emissions by source type and county, not by industry or economic sector. The inventory was not designed to estimate the fraction of statewide or regional emissions related to vehicles and equipment engaged in moving goods. As a result, we used statewide inventories as a starting point and benchmark for estimating emissions related to goods movement. Developing an emissions inventory in this case required analyzing statewide inventories as well as other recently developed data sources in order to improve our understanding of emissions generated by sources engaged in ports and international goods movement. Compiling this inventory required looking specifically at each emissions source category including ocean-going ships, harbor craft, cargo handling equipment, trains, and heavy-duty trucks. The following sections provide a brief overview of how these inventories were calculated.

There are a number of efforts underway to further improve the emissions inventory and this information will be incorporated in planning and implementation processes as it

becomes available. Aspects of the inventory such as the number, location, age, activity patterns, growth, and control of the engines used in various source categories will continue to be reviewed and improved in future revisions to the goods movement inventory and this document.

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, roll-on/roll-off vessels that move imported automobiles from Asia, and others. Some vessel types, like container ships, directly move imported goods into the State. Other vessel types, like passenger vessels, 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.

The ocean-going ship category is defined by size; the category includes all ship 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 acts 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.

CARB has 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. The 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 hotelling emissions generated by auxiliary engines as a ship is docked at port.

For this analysis, emissions were calculated on a statewide basis for each port in California. Emissions are calculated for hotelling 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 report. 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.

Growth of ocean-going vessel emissions are a major concern for this category. For this inventory CARB staff worked with experts at the University of Delaware to compile data on the 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[7], which agree with CARB growth projections based on main engine size. As a result, growth rate estimates for 2025 used in the report are consistent with the No Net Increase report.

As with all emissions inventories, the ocean-going ship inventory provided in this report is a snapshot of a larger inventory effort that is evolving over time. Emissions estimates can always be improved. This is especially true for categories such as ships where information is limited.

2. Harbor Craft

Harbor craft are commercial boats that operate generally within harbors and bays, or near the coast, or are smaller ships that support a commercial or public purpose. The harbor craft category includes many vessels operated by the U.S. Coast Guard, fishing vessels, tug boats and other types of ships. Vessel types in the goods movement inventory include:

- Crew and Supply - Ships used for carrying personnel and supplies to and from off-shore and in-harbor locations.
- Pilot Vessels – Ships used to guide ocean-going vessels into and out of a port or harbor.
- Towboat/Pushboat - Ships used to tow/push barges and pontoons. The hull of these vessels is usually rectangular in plan and has little freeboard.
- Tug Boats - Ships used for the towing and pushing of ocean-going ships or other floating structures such as barges.
- Other - Ships used in various commercial operations that do not fit into any other category.
- Work Boat - Ships used to perform duties such as fire/rescue, law enforcement, hydrographic surveys, spill/response research, training, and construction.
- Ferry/Excursion Vessel - Vessels used for public use in the transportation of persons or property.
- Commercial Charter Fishing Vessel - Vessels available for hire by the general public and used for the search and collection of fish for the purpose of personal consumption.
- Commercial Fishing Vessel - Vessels used in the search and collection of fish for the purpose of sale at market.

CARB staff recently developed an improved emissions inventory for the harbor craft category. The emission methodology uses the statewide population of ships, in conjunction with information about the size and activity of propulsion engines by ship 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

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

electrical systems and can also provide additional power to unique, essential vessel equipment (i.e. refrigeration units) during the normal day-to-day operation of the vessel.

Growth in harbor craft emissions was assessed by vessel category. Growth in emissions generated by tug boats was assumed proportional to growth in the number of visits to each port by ocean-going ships in each year. No growth was assumed in other harbor craft ship types unless location specific information was provided by local authorities.

The commercial harbor craft inventory in this report, like the ocean-going ship inventory, is a snapshot of a larger inventory effort which is evolving over time. CARB staff are continuing to evaluate inventory assumptions in this category.

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 emissions inventory representing cargo handling equipment that covers the following types of machines:

- Cranes – Because most large container handling cranes at ports are electrified, this category generally covers mobile cranes, often referred to as rubber tire gantry cranes, that are used to move, stack, and unstack containers at ports.
- Forklifts – Forklifts are used to move cargo, truck chassis, and other equipment for short distances.
- Container Handling Equipment – Containers are handled using specific types of vehicles similar to forklifts called side picks, top picks, and reach stackers.
- General Industrial Equipment – This category covers various types of equipment including rail-car movers and heavy duty off-highway trucks.
- Sweepers / Scrubbers – These vehicles are used to clean-up areas after bulk materials have been moved.
- Excavators – Excavators are a specific type of equipment designed for handling heavy dry bulk materials.
- Bulk Handling Equipment- This category covers tractors, loaders, and backhoes that are used to handle bulk materials.
- Yard Trucks – Yard trucks or "hostlers" are used to move containers within or between terminals.

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.

4. Trains

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 represents

goods that are imported into and through California from overseas, while the balance represents domestic freight imported or exported to California, and freight generated and consumed within California.

CARB's inventory of emissions from locomotives was first developed in 1991 and has been updated periodically. The inventory uses a relatively simple methodology that accounts for generalized locomotive activity patterns over broad geographical regions. The inventory covers two types of train locomotives. Line-haul locomotives are large locomotives that are used to move trains over long distances. Switchers are locomotives used to transport smaller trains within a rail yard or over short distances. Switching engines are much smaller, and often older, than locomotives used for line-hauls. Line-haul locomotives operate in rail yards as they travel through to their final destination.

CARB staff started with our standard statewide locomotive inventory in order to develop an inventory of emissions from locomotives engaged in the movement of internationally imported and exported goods. To develop a goods movement inventory we first reviewed available literature and information to estimate a percentage of overall train activity in each region of California that is related to imported and exported goods. This fraction ranged from 25% in many regions of California to 40% in the Los Angeles region and about 35% in the Bay Area and Central Valley.

Next we developed emissions estimates for locomotive activity specifically within ports and rail yards. For ports, we used locomotive emissions estimates developed by the Ports of Los Angeles and Long Beach, and scaled these emissions to develop estimates for other ports based on port-specific total tonnage of freight throughput. All port emissions were assumed related to internationally imported and exported goods. For off-port rail yards, we used locomotive emissions estimates for the Roseville Rail Yard that were developed previously by CARB, and scaled these emissions to develop estimates for other rail yards based on our best estimate of the number of locomotives and railcars passing through each rail yard on an annual basis. These emissions estimates are based upon the best data currently available to CARB staff. As the State's major rail carriers submit additional data required through the Statewide Rail Memorandum of Understanding with major rail carriers, these estimates will improve.

5. Heavy-duty Trucks

Heavy-duty trucks are an integral and important component of California's goods movement transportation system. Most goods, whether imported into California from overseas, imported into California from Canada, Mexico, or other states in the U.S., generated and consumed within California, or generated and exported from California are moved by a truck at some time during their transport in California. Emissions generated by the movement of international imported goods is only a fraction of the total emissions generated by heavy-duty trucks in California.

Emissions from heavy duty trucks are estimated in California by a complex model, called EMFAC, which is designed to take into account many factors that affect emissions, from driving patterns and vehicle miles traveled, to engine age, technology, and controls. The EMFAC model provides emissions estimates by vehicle class and by

county. It does not provide emissions estimates for a specific industry or sector of the economy, such as international goods movement.

We used additional data sources and assumptions to estimate truck emissions generated during ports and international goods movement. First we estimated total truck emissions within each port and rail yard in California, and second, we estimated the fraction of heavy-duty truck emissions by air basin that can be attributed to goods movement. Inventory estimates representing trucks associated with goods movement should be considered draft in this document. These estimates represent the best information currently available to CARB staff.

To estimate truck emissions at ports and rail yards we used emissions data representing the Ports of Los Angeles and Long Beach that were published in 2004. These estimates included both idling and movement emissions within port boundaries and within individual marine terminals. We used combined emissions from the Ports of Los Angeles and Long Beach, and scaled these emissions to develop estimates for other ports based on total annual tonnage throughput at each port[8].

We used combined emissions from the Ports of Los Angeles and Long Beach, and scaled these emissions to develop estimates for each intermodal rail yard in California using the total number of on-site dedicated yard trucks. The number of yard trucks was estimated by CARB staff at each rail yard in California based on a survey of cargo handling equipment that was part of the basis of the cargo handling equipment inventory described above. Overall, heavy-duty truck emissions at intermodal rail yards were a small fraction of emissions at major ports in California.

These estimates represent the best data currently available to CARB. We expect that by working with port and rail yard operators in the future more refined emissions estimates can be generated.

We estimated the fraction of port-related goods movement truck emissions in each air basin based upon data from the Southern California Association of Governments (SCAG) and reports generated by SCAG and other local transportation agencies in southern California. In southern California, SCAG generates estimates of total miles traveled by heavy-duty trucks within their jurisdiction (the Los Angeles and Inland Empire) regions. SCAG maintains a heavy duty truck travel demand model that estimates vehicle miles traveled as well as the number of trips that are generated by the ports of Los Angeles and Long Beach, as shown in Table A-5.

Table A-5 Heavy Duty Trucks Involved in Ports and International Goods Movement: Daily Trips, Vehicle Miles Traveled, and Estimated Trip Length Generated by the Ports of Los Angeles and Long Beach by Year

Year	Trips	VMT	Average Trip Length
2000	39,500	1,463,670	37.1
2010	50,800	1,866,550	36.7

[8] American Association of Port Authorities (2005). Port Industry Statistics: 2003 US Port Cargo Tonnage Rankings. http://www.aapa-ports.org/industryinfo/statistics.htm

| 2025 | 105,540 | 3,459,040 | 32.8 |

These data were used to estimate the fraction of heavy duty truck emissions in the SCAG region that are related to direct trips to and from the ports of Los Angeles and Long Beach.

Using technical reports generated by transportation agencies in the Los Angeles region, we estimated an additional amount of heavy duty truck miles traveled as a result of secondary truck trips generated by a fraction of trucks that are expected to travel to a distribution center for either local or long-haul additional truck trips[9]. We assumed 38% of local primary truck trips result in three additional secondary trips by smaller trucks. These additional local trips were assumed to have the same length as primary truck trips. We assumed 21% of local primary truck trips terminate in a distribution center, where the contents of a 40 foot container are transloaded to a 53 foot container. Because a 40 foot container is about two-thirds the size of a 53 foot container, we assumed two-thirds additional transload long-haul trips are generated. Of these secondary, transload, long haul trips, we assumed 27% terminated outside of the SoCAB in other areas of California, 20% terminated in Arizona, 6% terminated in Canada or Illinois, with the rest terminating in other national destinations[10]. We assumed a trip length associated with each termination point to estimate VMT by termination point. All primary trips, and all long-haul trips were assumed to be performed by heavy-heavy duty trucks.

VMT was summarized by year and compared to total VMT by vehicle class for the SoCAB in the EMFAC model. This ratio was then applied to emissions to estimate emissions related to trucks engaged in goods movement.

To estimate the fraction of goods movement emissions outside of the SoCAB, we scaled primary and secondary local VMT in the SoCAB to other air basins based on total tonnage throughput at each port. If no port was located in an air basin, no VMT was assigned. For the port of Oakland, scaled primary trips estimated using SCAG trip estimates scaled by tonnage throughput at the Port of Oakland were used to estimate secondary truck trips based on the same transload long-haul percentage (21%) as assumed for the SoCAB. Thirty percent of trips were assumed to travel north, and 70% were assumed to travel east. A trip length was assumed for each of these trips.

Secondary trips generated by the Ports of Oakland, Los Angeles, and Long Beach were tracked for both expected destinations in a given air basin in California, and through traffic as long-haul trips travel to their final destination. A trip length was assumed for each of these trips to estimate secondary VMT. Total goods movement VMT by vehicle class was divided by the air basin VMT total by vehicle class. This ratio was then applied to estimate emissions related to trucks engaged in goods movement.

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[9] (ACTA, 2004), as cited in Jones and Stokes (2004), Port of Los Angeles, Portwide Rail Synopsis

[10] Metropolitan Transportation Authority (2004). DRAFT Compendium of Freight Information for the Greater Los Angeles Metropolitan Area

Table A-6 presents the fraction of truck emissions related to international import and export goods movement assumed for selected air basins in California. As is shown, a significant fraction of total emissions in the SoCAB are assumed related to goods movement. Because the Port of Oakland is smaller than the Ports of Los Angeles and Long Beach, the fraction of goods-movement related truck emissions is lower than in the SoCAB. Heavy-duty truck fractions are high in the Central Valley (Sacramento and San Joaquin Valleys) because of through traffic VMT generated by the Ports of Los Angeles, Long Beach, and Oakland.

Table A-6 Percentage of Truck Emissions Generated by Ports and International Goods Movement by Year, Air Basin, and Truck Type

Air Basin	2001 Light-Heavy and Medium-Heavy Duty Trucks	2001 Heavy-Heavy Duty Trucks	2025 Light-Heavy and Medium-Heavy Duty Trucks	2025 Heavy-Heavy Duty Trucks
South Coast	15	20	30	40
San Francisco Bay Area	1	7	2	15
San Diego	0	4	1	7
San Joaquin Valley	0	25	0	35
Sacramento Valley	0	20	0	40

B. Adjustment Factors 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 fraction of off-shore diesel PM emissions never reaches the shore, depending on wind direction and overwater deposition rates.

To address the differing impact of off-shore sources, CARB staff developed a statewide ship emissions impact adjustment factor of 0.1 for off-shore diesel PM emissions. In calculating the impact of off-shore emissions, the mass of directly emitted diesel PM associated with off-shore marine sources was multiplied by 0.1 and the resulting emissions were assigned to the appropriate coastal county. No adjustment was made for secondary PM formation from NO_X and SO_X emissions, since these pollutants require at least several hours to form particle nitrate and sulfate. For the same reason, off-shore sources of NO_X and VOC that contribute to ozone formation were also not adjusted. These latter assumptions probably overestimate the impact of offshore emissions, as there will be some losses due to offshore winds and overwater

deposition, although 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 exposure adjustment factor for diesel PM was derived from dispersion modeling results for the Ports of Los Angels and Long Beach (CARB 2005a). The population-weighted diesel PM concentrations from on-shore emission sources within the urban region were about 10 times the population-weighted concentration from off-shore emission from ship main propulsion engines. Another way of looking at this is that about 0.1 tons of diesel PM released on-shore would result in an equivalent population-weighted diesel PM concentration to 1 ton of diesel PM released off-shore in the shipping lanes.

Since the ships in San Diego and San Francisco Bays are surrounded by urbanized areas, the adjustment factor was increased to 0.25. This was considered conservative (i.e., health protective). In San Diego Bay, the area around the port is not as heavily populated as other parts of San Diego County. For San Francisco Bay, the average wind speed was about four times higher than the average wind speed of Los Angeles and San Diego, which would cause greater dispersion, although this is partially offset by the greater frequency of on-shore flow, over 80% of the time (compared to 50% for the other two areas) The impact of ship emissions in these areas will be refined using region-specific modeling studies, as has been performed for the Ports of Los Angeles and Long Beach. Again, the adjustment factors were used only for directly emitted diesel PM.

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, 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. Nitrates and Sulfates

This section provides information on the population-weighted exposure calculation of annual geometric mean for nitrate and sulfate to which people in different parts of California are potentially exposed. The term "potentially" is used because we realize that 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 sulfates, 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. However, with Geostatistical Analyst 9.0 software staff was able to modify the power used to 2.5 in order to optimize the interpolated estimations. In addition for the weighting factor, a minimum of 10 monitoring stations were used even if those sites were beyond the search radius. 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 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

Most airborne sulfate in California is due to anthropogenic sulfur emissions, but apportioning exposure to sulfur sources must take into account "background" sulfate from the two major sources of background sulfate in California - biogenic sulfate generated over the ocean, and global transport of natural and anthropogenic sulfate in elevated layers of the atmosphere.

A rough estimate of the statewide distribution of background sulfate was constructed based on limited measurements from remote sites that are isolated from exposure to urban or industrial pollution, and results of model simulations of the global sulfur processing in the atmosphere.

Unequivocal measurements of background sulfate are limited to a few weeks of data from three sites in northern California. Oceanic sulfate data come from Trinidad Head in Humboldt County, and global transport sulfate data come from Trinity Alps and Mount Lassen. Comparison with several years of routine monitoring data permitted estimation of the average annual concentration of background sulfate at these sites.

Computations of annual average background sulfate in the rest of the State were based on rough estimates of the effects of site-specific meteorology and terrain on inputs from the ocean and upper air.

The statewide estimates were reviewed for consistency with reported sulfate air quality data and published global sulfate model results, and adjusted if necessary.

Finally, the background estimates were subtracted from ambient data to approximate site-specific anthropogenic sulfate concentrations. Although there is considerable uncertainty in the background estimates, ambient concentrations at most urban sites in California are several times background, so that the impact of this uncertainty on statewide sulfate population exposure is believed to be small.

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 µg/m^3 and 0.08 µg/m^3 for nitrates and sulfates calculations, respectively.

In this report, staff did not estimate the health impacts from exposures to sulfates for two reasons. First, staff has determined that there is a need for a better understanding of the total contribution from background. Second, the monitored data and emission inventory data need to be reconciled. Most airborne sulfates in California are due to anthropogenic sulfur emissions, but a portion comes from sulfate "background". The two major sources of background sulfates in California are biogenic sulfates generated over the ocean, and global transport of natural and anthropogenic sulfates in elevated layers of the atmosphere. A rough estimate of the statewide distribution of background sulfates was constructed using the best currently available information. Even with the subtraction of the estimated background, a large discrepancy between monitored data and emission inventory data remained. Staff attempted to determine how much sulfates would be expected to be measured by the monitoring network based on the emission inventory. The total sulfates emission inventory for the state was applied to the SoCAB, and a conservative mixing layer of 100 meters was applied. The total amount due to the emission inventory was only one-tenth of the measured value with the background removed. Because of this large discrepancy, staff has determined that there is a need for a better understanding of the total contribution from background together with a reconciling of the monitoring data and the emissions inventory before a health impacts assessment from sulfates exposures can be completed.

3. Ozone

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.

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 2002, 2005b) to determine concentration-response functions for eight different 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

- o Premature Deaths
- o Asthma Attacks
- o Work Loss Days
- o Minor Restricted Activity Days

Ozone

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

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 the 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:

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

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 nitrates formed from goods movement-related NO_X must be calculated to derive secondary diesel PM estimates; similarly, diesel PM formed from goods movement-related SO_X must also be estimated. Details on how each of the pollutant concentrations was derived are provided above and in the 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 function to the exposed population for each basin;
3. Associate the health impacts with the related emission inventory in the base year (diesel PM, NO_X and SO_X for primary diesel PM, PM nitrates and PM sulfates, 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.

a) Premature Death

For premature death due to diesel PM2.5, the study by Pope et al. (2002), updating the original mortality estimates of the original ACS cohort study for all-cause, cardiopulmonary and lung cancer mortality was used to derive the concentration-response function. For this study a 6% increased risk for all-cause mortality was identified for each 10 µg/m^3 difference in fine particle concentration (Pope et al. 2002). The US EPA is currently using Pope et al. (2002) results for estimating premature deaths from PM exposures (U.S. EPA 2005).

The log-linear approach to calculating diesel PM mortality in this health impact assessment is consistent with the exposure response relationships observed by Pope et al. (2002). The log relative risks for all-cause, cardiopulmonary, and lung cancer mortality increased across the exposure gradient for fine PM, and goodness-of-fit tests found that the associations were not significantly different from linear associations (Pope et al. 2002). Furthermore, this relationship did not appear to have a discernible lower "safe" threshold (Brook et al. 2004). This linear relationship between excess mortality risk and PM is demonstrated in Figure II-1, which plots the relative risks associated with all-cause mortality and long-term PM exposure in the Harvard Six-Cities study (Dockery et al. 1993). A number of recent studies have demonstrated the linear association between particulate matter pollution and cardiovascular disease. Chen et al. (2005) found a concentration-response function for fatal coronary heart disease in California women participants of the Adventist Health Study on the Health Effects of Smog (AHSMOG). Using a cumulative monthly average pollution estimate and dividing the females in the AHSMOG sub-cohort into three different groups: those exposed to low concentrations of PM2.5, and those exposed to median or high concentrations; the investigators found that the risk levels increased—with the greatest risk seen in a two-pollutant model with ozone at concentrations exceeding 38 µg/m^3 PM2.5 (Chen et al. 2005).

A California-specific study of the same mortality endpoints in relation to ambient PM2.5 has recently been published. This study (Jerrett et al. 2005), employs many methodological advances and uses the latest techniques in spatial analysis with the intent of reducing exposure misclassification. Several arguments are put forth by Jerrett et al. (2005) to explain the larger effect estimates found in this analysis. These include: underlying differences in the subcohort; differing rates of decline in ambient PM2.5 concentration from one metropolitan area to another (in the ACS study); different exposure sources; meteorological or topographic differences; and, larger exposure measurement error due to heterogeneous changes in air pollution levels during follow-up. The authors provide well-developed arguments against any of these factors having a significant impact on the estimates. However, given the number of potential areas for differences to occur, and the variability of all of these parameters in different regions throughout the state, staff and peer reviewers felt it was premature to use these new estimates to calculate statewide mortality estimates. It seems reasonable not to use these estimates before confirmatory studies can be performed in different metropolitan regions.

Figure A-2: Mortality Relative Risk and Long-Term PM Exposure in the Six Cities Study*
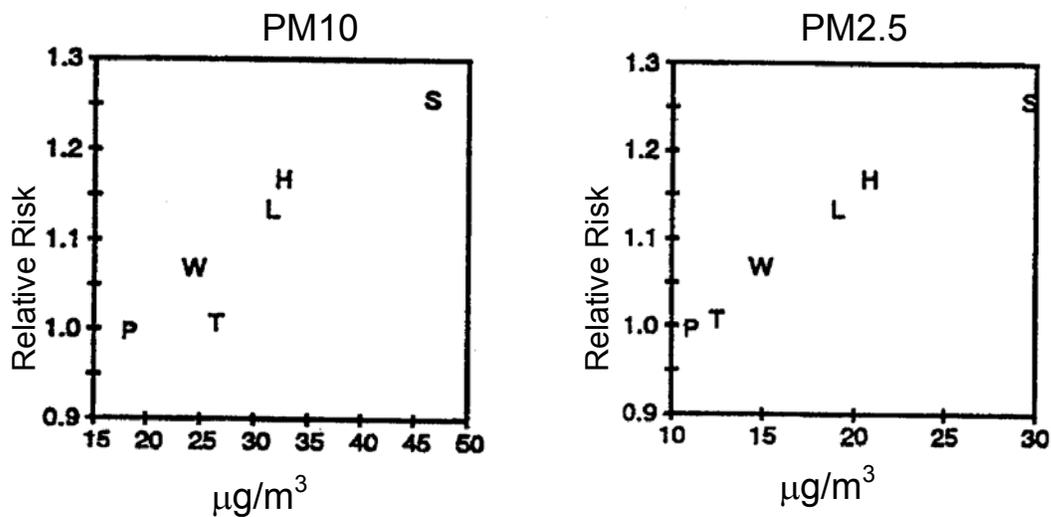
*Figure adapted from US EPA 1996, AQ Criteria for PM, Vol. III, p. 12-167:

H= Kingston-Harriman TN; L=St. Louis MO; P=Portage WI; S=Steubenville, OH; T=Topeka KS; W=Watertown MA

b) Asthma Attacks

A study by Whittemore and Korn (1980) was used to develop the concentration respons function for the relationship between air pollution and asthma attacks. Children and adults living in six southern California communities were followed for three 34-week periods in 1972-1975. In a two-pollutant model with ozone, daily levels of both TSP and ozone were significantly related to reported asthma attacks. The coefficient in the

concentration-response function is based on total suspended particulate concentration estimates.

c) Work Loss Days

Work loss days and restricted activity days were estimated using data from a study by Ostro (1987) of adults living in major metropolitan areas. Ostro estimated the impact of PM2.5 on the incidence of work-loss days, restricted activity days, and respiratory-related restricted activity days in a national sample of a urban adult working population (ages 18-65). A weighted average of the coefficients from each year of the study (1976-1981) were pooled to develop the coefficient for the concentration response function.

d) Minor Restricted Activity Days

The coefficient for this concentration-response function was derived from Ostro and Rothschild (1989). These investigators estimated the impact of PM2.5 on the incidence of minor restricted activity days and respiratory-related restricted activity days in a national sample of an urban adult working population (ages 18-65).

2. Ozone

For health effects due to goods movement-related ozone concentrations, staff followed the same basic procedure outlined in the Review of the Ozone Standards (CARB 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 2001-2003 averages above the newly approved State 8-hour standard of 0.070 ppm. As detailed in the Ozone Standard Staff Report (2005), 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 the reactive organic gas (ROG) emissions inventory and the NO_X emissions to determine two sets of health impact factors. These factors are then applied to the Goods Movement inventories of ROG and NO_X to determine the health impacts. The average of the two results is then considered to be the ozone-related health impacts of Goods Movement. 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 2005b).

a) Premature Death

To develop the concentration response function for premature mortality, several meta-analyses[11] of daily (short-term) exposure to ozone and premature mortality were used to determine a probable range of estimates. A central estimate of 1% per 10-ppb change in 24-hour ozone was based on several meta-analyses, including those by Anderson (2004), Levy (2001), and Stieb (2003). A low estimate of 0.5% per 10-ppb change in 24-hour ozone was based on Bell's analysis of NMMAPS (National Mortality Morbidity Air

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[11] Meta-analyses are statistical summaries of a range of estimates from epidemiologic studies with similar methodologies.

Pollution Study) (Bell 2004). A high estimate of 1.5% was based on Thurston and Ito (2001) and Gryparis' European study (Gryparis 2004) of the summer-only effect.

Three recent meta-analyses of short-term mortality and ozone exposure were recently published. These studies confirmed the results of the analyses used for this health endpoint concentration-response function (Levy et al. 2005, Bell et al. 2005, Ito et al. 2005).

b) Hospital Admissions

An estimate of the relationship between ozone pollutant changes and hospital admissions for respiratory disease was based on daily time-series studies of hospital discharge diagnoses related to circulatory or respiratory diseases by Thurston and Ito (1999). These authors used a random effects model based on three studies from North America for various age groups.

c) Minor Restricted Activity Days

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).

d) School Absences

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.

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, asthma attacks, 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 percent 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 mi x 20 mi) into an area of 40 mi x 30 mi in the north direction and another area of 20 mi x 20 mi 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 mi (east-west) and 50 mi (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 mi x 50 mi.

E. Economic Valuation

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 (or $8.2 million in 2005 dollars) for a 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 5 contingent valuation studies and 21 wage-risk studies, which span the range 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.

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 about how much they place on risk avoidance. Today the referendum format is shown to be more effective, in which the survey suggests a specific dollar amount and asks the respondents whether they would be willing to pay that much for a decreased probability of realizing a specific and 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 the 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. They simply averaged the 26 studies to get a value of 4.8 million in 1990 dollars. A recent review (Viscusi and Aldy 2003) discusses some of the studies and provides the level of risk reduction used in the study. From that we can infer the compensating wage.

Table A-7 Annual 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	1988	Wage-risk	3.6		

Authors	Year	Type of Estimate	Valuation (millions 1990$)	Annual risk reduction	Implied compensating wage (1990$/year)
Girard					
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

Note that typical studies (those getting a result close to the mean of $4.8 million in 1990 dollars) involve a mortality risk of 1/10,000 or something close to that level. The risk premium is several hundred dollars per year.

As real income increases, people may be willing to pay more to prevent premature death (U.S. EPA, 2003, pages 9-17 and 9-18). U.S. EPA further adjusted the 1990 value by a factor of 1.27 for a 2020 income level. Assuming that real income grew at a constant rate from 1990 and will continue at the same rate until 2020, we adjusted the value of avoiding a premature death (increasing it at a rate of 0.8% per year) for each year. We then updated the value to 2005 dollars. After these adjustments, the value is $8.2 million in 2005, $8.6 million in 2010 and $9.3 million in 2020, all expressed in 2005 dollars.

The U.S. EPA also uses the WTP methodology for some of the non-fatal health endpoints, including minor restricted activity days and acute asthma attacks. For school absence days, the U.S. EPA uses an estimate of the parent's lost wages (U.S. EPA, 2004). The CARB calculated the cost of hospital admission for acute respiratory problems as the direct cost of illness plus associated costs such as loss of time for work, recreation and household production, as well as non-pecuniary losses such as pain, suffering and inconvenience. (CARB, 2003). The CARB estimates the valuation of a work loss day as the lost wages for the employee.

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 are a function of income level, so they change over time.

Table A-8 Values for Health Effects Per Case of Mortality, Hospital Admissions, and Minor Illnesses (in 2005 Dollars)

Health Endpoint	2005	2010	2020	References
Mortality				
Premature death[1] ($ million)	8.2	8.6	9.3	US EPA (2003), 9-27
Hospital Admissions				
Acute Respiratory	34,000	34,000	34,000	CARB (2003), 63
Minor Illnesses				
Minor restricted activity day (MRAD)	61	61	63	US EPA (2004), 9-159
Work loss day	178	178	178	2002 California wage data, US Department of Labor
Asthma – acute (per symptom day)	50	51	52	US EPA (2004), 9-158
School absence day	87	87	87	US EPA (2004), 9-159

[1]These are adjusted by income for the respective years because they are based on willingness to pay (WTP) studies. The other health endpoint valuations are based on cost-of-illness (COI) studies and do not need an income adjustment.

F. Uncertainty Calculations

The health impacts were estimated with an uncertainty range that reflected the uncertainty behind the concentration-response functions used. The per-case economic valuations also come with standard errors for three of the health endpoints – premature death, asthma attacks, and MRADs. The total economic valuation entails multiplying the health impacts (number of cases) by the per-case valuations. To propagate the uncertainty in cases and valuation together, staff estimated the lower bound and the upper bound of the product as follows.

Since the premature death per-case valuations follow a lognormal distribution, staff calculated the ratio of the mean and the lower bound for cases and per-case valuation, and combined them to get the lower bound of the product. Similarly, staff calculated the ratio of the mean and the upper bound for cases and per-case valuation, and combined them to get the upper bound of the product.

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 international 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 particulate 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 also excludes all emissions from transportation refrigeration units. Overall, the use of dispersion adjustment factors led to a 50% reduction in diesel PM in 2001 that was not emitted in the SoCAB, and a 70% reduction for diesel PM in 2020 that was not emitted in the SoCAB.

Table A-9 Dispersion-Adjusted Goods Movement Emissions Inventory

Pollutant	2001	2005	2010	2015	2020
Diesel PM	6	5.5	5.5	6	6.5
NO$_X$	400	390	370	380	410
ROG	30	28	26	26	27
SO$_X$	60	80	95	120	160

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 attributed to the outer continental shelf air basin, which is defined as outside of three miles from shore, were discounted by 90%. Overall, the use of dispersion adjustment factors led to a 10% reduction in diesel PM in 2001, and a 30% reduction in 2010.

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

Pollutant	2001	2005	2010	2015	2020
Diesel PM	70	70	60	50	45
NO$_X$	3700	3200	2700	2200	2000
ROG	5200	4700	4400	4200	4200
SO$_X$	320	260	280	320	360

[1]Statewide inventory does not contain natural windblown dusts and does contain biogenic emissions. This table includes emissions from the SoCAB.

B. Exposure Estimates

This section provides information on the exposure estimates used in the analysis of the health impacts. The modeled results of the population-weighted exposures of the Annual Geometric Mean for nitrate (NO3) and sulfate (SO4) are estimated concentrations to which people in different parts of California are potentially exposed. The term "potentially" is used because we realize that daily activity patterns influence a person's exposure to outdoor levels of the air pollutants. The exposure estimates for NO3 and SO4 are based solely on "outdoor" data, as measured by the Statewide Routine Monitoring Network and additional special monitoring networks such as IMPROVE and Children's Health Study. For Diesel Particulate Mater (DPM), CARB staff used a combination of receptor modeling techniques, emissions and monitoring data to estimate DPM concentrations because there is not a method for directly measuring outdoor diesel PM. Unlike nitrates, sulfates and DPM, the analysis of ozone-related health impacts relied on daily measured values rather than an annual average. Here, we present the peak indicator of 1-hour concentrations in each basin. It is a highly precise estimate of the highest concentration expected to occur once per year, on average. Overall, the modeled exposures results presented here provide an integrated regional perspective rather than an indication of exposure at any individual location.

Table A-11 provides a summary of the calculation of exposure estimates required for the health impacts assessment. The methodology and years used for the exposure estimates for DPM, O3, NO3 and SO4 are different for each pollutant. The details are explained in the methodology section of this report.

Table A-11 Summary of the Exposure Estimates by Air Basin.

	1998	1998	2000	2003
AIR BASIN	NO_3^{1} $(\mu g/m^3)$	SO^{42} $(\mu g/m^3)$	DPM^3 $(\mu g/m^3)$	O_3^{4} (ppm)
Great Basin Valleys	0.86	0.79	0.10	0.084
Lake County	0.59	0.55	0.20	0.071
Lake Tahoe	0.12	0.12	0.40	0.081
Mojave Desert	2.60	1.18	0.40	0.117
Mountain Counties	0.81	0.70	0.40	0.122
North Central Coast	0.85	0.63	0.80	0.089
North Coast	0.40	0.36	0.80	0.068
Northeast Plateau	0.39	0.34	0.70	0.072
Sacramento Valley	0.85	0.74	1.20	0.111
Salton Sea	1.82	1.60	1.50	0.119
San Diego	2.21	2.08	1.40	0.101
San Francisco Bay	0.84	0.61	1.60	0.098
San Joaquin Valley	1.36	1.31	1.30	0.122

South Central Coast	1.28	1.39	1.10	0.103
South Coast	*3.58*	1.84	2.40	0.146
CALIFORNIA	2.25	1.40	1.80	N/A

[1] NO$_3$ exposure based on inverse-distance-weighted and population-weighted annual geometric means for nitrates.

[2] SO$_4$ exposure based inverse-distance-weighted and population-weighted annual geometric mean for sulfates. Although they're presented here, sulfates were not part of our health impacts assessment in this report.

[3] DPM is derived from receptor modeling results, emissions and monitoring data.

[4] Ozone 1-hour peak indicator is based on 2001-2003 data and provides the basis for our 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 2005).

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 is a summary of the combined statewide health effects from PM and ozone exposure linked with goods movement. We estimate that 750 premature deaths (260-1,300, 95% confidence interval (95%CI)) can be associated with goods movement emissions, annually on a statewide basis. Valuations are in year 2005 dollars.

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 over 60% 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 18%. Moreover, for the SoCAB, goods movement-related health impacts account for about 11% of the total impact of ozone and PM pollution from all sources.

Table A-12 Statewide PM and Ozone Health Effects Associated with International Goods Movement and Economic Valuation (95% confidence limits in parentheses)

Health Outcome	2005 Number of outcomes	Valuation ($million)	2010 Number of outcomes	Valuation ($million)	2020 Number of outcomes	Valuation ($million)
Premature Death	750	$6,200	760	$4,700 to 5,700	920	$3,100 to 5,500
	(260-1,300)	($2,100-12,000)	(270-1,300)	($1,600-11,000)	(320-1,600)	($1,000-11,000)
Hospital Admissions (repiratory causes)	290	$10	290	$7 to 8	320	$4 to 7
	(170-410)	($6-14)	(160-400)	($4-12)	(180-450)	($2-10)
Asthma Attack	15,000	$1	15,000	$1 to 1	18,000	$0 to 1
	(3,600-26,000)	($0-2)	(3,600-26,000)	($0-2)	(4,400-32,000)	($0-2)
Work Loss Days	130,000	$23	130,000	$16 to 20	160,000	$10 to 18
	(110,000-150,000)	($19-26)	(110,000-150,000)	($14-23)	(130,000-180,000)	($9-21)
Minor Restricted Activity Day	880,000	$53	880,000	$38 to 46	1,100,000	$23 to 41
	(630,000-1,100,000)	($25-110)	(640,000-1,100,000)	($18-90)	(770,000-1,300,000)	($11-80)
School Absence Day	330,000	$28	320,000	$20 to 24	350,000	$11 to 20
	(85,000-610,000)	($7-53)	(82,000-580,000)	($5-44)	(92,000-650,000)	($3-36)
TOTAL	N/A	$6,300	N/A	$4,800 to 5,800	N/A	$3,100 to 5,600
		($2,200-12,000)		($1,700-11,000)		($1,000-11,000)

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

Year Air Basin	2005 Mean Deaths	95% confidence Limits	2010 Mean Deaths	95% confidence Limits	2020 Mean Deaths	95% confidence Limits
GBV	<1	<1	<1	<1	<1	<1
LC	<1	<1	<1	<1	<1	<1
LT	<1	<1	<1	<1	<1	(<1-1)
MC	2	(2-3)	2	(1-3)	1	(<1-2)
MD	15	(6-25)	13	(5-21)	13	(5-23)
NC	<1	(1-2)	<1	(<1-1)	<1	(<1-1)
NCC	3	(1-5)	3	(<1-4)	3	(1-5)

Appendix A-51

NEP	<1	(<1-1)	<1	(<1-1)	<1	(<1-1)
SC	530	(190-930)	530	(180-930)	580	(200-1,000)
SCC	38	(13-67)	46	(16-81)	71	(25-120)
SD	44	(15-77)	52	(18-92)	100	(35-180)
SF	67	(23-120)	74	(25-130)	100	(35-180)
SJV	27	(10-47)	23	(8-40)	23	(8-40)
SS	12	(4-20)	11	(4-19)	12	(4-20)
SV	15	(5-26)	13	(5-23)	13	(5-24)
Total	750		760		920	

[1] Values are rounded.

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 vessels (Hotelling Activities Associated with Ships, Maneuvering and Transit Associated with Ships) 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 International Goods Movement: Contributions of Source Categories (95% confidence limits in parentheses)

	2005	2010	2020
Source Category	Number of deaths	Number of deaths	Number of deaths
Commercial Harbor Craft	110	110	120
	(39-190)	(39-200)	(43-220)
Cargo Handling Equipment and Dredgers	43	38	17
	(15-76)	(13-67)	(6-29)
Hotelling Activities Associated with Ships	65	90	160
	(23-110)	(31-160)	(55-280)
Maneuvering and Transit Associated with Ships	110	140	240
	(38-190)	(48-240)	(85-430)
Rail	99	83	89
	(35-170)	(30-150)	(32-160)
Truck	250	210	170

	(89-450)	(74-370)	(60-310)
SoCAB Ports (modeled)	74	90	130
	(25-130)	(31-160)	(43-220)
STATEWIDE TOTAL	750	760	920
	(260-1,300)	(270-1,300)	(320-1,600)

4. Pollutant-Specific Impacts

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. n our goods movement assessment, we found that the contribution of PM outweighs that of ozone by several fold (Table A-15). However, when the relative contributions of primary and secondary PM are examined, the secondary PM appears to be the major contributor, accounting for more than 60% of the total estimated annual premature deaths.

Table A-15 Mortality Effects Associated with International Goods Movement: Pollutant Contributions (95% confidence limits in parentheses)

	2005	2010	2020
Pollutant	Number of deaths	Number of deaths	Number of deaths
Primary Diesel PM	260	270	370
	(88-460)	(94-490)	(130-650)
Secondary Diesel PM			
(Nitrates)	450	450	510
	(150-800)	(150-790)	(180-900)
Ozone	44	42	48
	(22-66)	(21-64)	(24-72)
Statewide Total	750	760	920
	(260-1,300)	(270-1,300)	(320-1,600)

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 later in 2005.

6. Port-specific Impacts

Based on the methodology described above in section E, we estimated the non-cancer health effects, including premature death, asthma attacks, 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 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 for POLA and POLB

Health Effects	Range	2001	2005	2010	2015	2020
Premature Deaths	Mean	56	74	90	100	130
(Age 30+)	Lower	19	25	31	34	43
	Upper	100	130	160	180	220
Asthma Attacks	Mean	1,200	1,500	1,900	2,100	2,600
(All ages)	Lower	280	370	450	500	630
	Upper	2,000	2,700	3,300	3,700	4,600
Days of Work Loss	Mean	10,000	14,000	16,000	18,000	23,000
(Age 18-65)	Lower	8,800	12,000	14,000	16,000	20,000
	Upper	12,000	16,000	19,000	21,000	27,000
Minor Restricted Activity Days	Mean	54,000	72,000	87,000	97,000	120,000
(Age 18-65)	Lower	44,000	58,000	70,000	78,000	98,000
	Upper	65,000	86,000	100,000	120,000	140,000

D. Economic Valuation

1. Value of Avoided Health Effects

Table A-17 shows the statewide valuation of health effects associated with goods movement within California. The values reported in this table result from multiplying mean number of health effects cases reported in Table A-12 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).

Table A-17 Statewide Health Effects of Ozone, Nitrates and Diesel PM Associated with Goods Movement in California (Valuations in millions of 2005 dollars)

	2005 Cases	2005 Val.[1]	2010 Cases	2010 Val. @ 3%	2010 Val. @ 7%	2020 Cases	2020 Val. @ 3%	2020 Val. @ 7%
Premature Death	750	$6,200	760	$5,700	$4,700	920	$5,500	$3,100
Hospital Admissions (repiratory causes)	290	$10	290	$8	$7	320	$7	$4
Asthma Attack	15,000	$0.7	15,000	$0.6	$0.5	18,100	$0.6	$0.3
Work Loss Days	130,000	$23	130,000	$20	$16	160,000	$18	$10
Minor Restricted Activity Day	880,000	$53	880,000	$46	$38	1,060,000	$41	$23
School Absence Day	330,000	$28	320,000	$24	$20	350,000	$20	$11

[1]Val. – Valuation in millions of 2005 dollars. @ 3% - discounted at 3% per year, @ 7% - discounted at 7% per year.

Between 2005 and 2020, the cases increase for all health endpoints. At the same time, the valuations discounted to the present year of 2005 decrease. Discounting is a way to represent preferences between the future benefits and the present. Future valuations discounted to the present become smaller farther in the future signaling a preference for the present and thus putting more emphasis on programs with earlier air pollution reductions. For example, the premature deaths for 2005 are less than 2020 (750 vs 920, respectively), but have a higher value ($6.2 vs. $5.5 billion discounted at 3 percent) implying early controls are preferable to those in the far future. Lower discount rates signal that the future benefits are important and should not be weighed much less than benefits attainable in the present. The range of discount rates used in the Table A-17 shows that, for decision making, the future benefits discounted at 3 percent signal a higher preference for the future than the 7 percent rate, because the valuation is higher at 3 percent ($5.5 billion), than 7 percent ($3.1 billion).

Table A-17 shows the sensitivity to the choice of discount rate. Discount rates are a way to represent preferences between the future and the present benefits. Lower discount rates tend to value the future benefits closer to the present ones. The 3% discount rate, used in the above table, implies that, in decision making, the future benefits are more important than when a 7% discount rate is used.

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 750 premature deaths (260-1,300, 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), or 4% of all deaths. 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, asthma attacks, 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, which show modest increases statewide.

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 over 60% 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 18%. Moreover, for the SoCAB, goods movement-related health impacts account for about 11% 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 vessels 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. For example, statewide, it is estimated that ozone exposure above the proposed California eight- hour ozone standard contributes to approximately 630 premature deaths annually. 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 several fold (Table A-15). However, when the relative contributions of primary and secondary PM are examined, the secondary PM appears to be the major contributor, accounting for more than 60% of the total estimated annual premature deaths. 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 600 premature deaths. It is estimated (Table A-15) that goods movement contributes to approximately 44 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. CARB will examine these and other air basin estimates in its mitigation plan.

Table A-16 shows the total valuation of the current health impacts associated with port-related goods movement and other port activities in California to be about $6.3 billion (in year 2005 dollars), with an uncertainty range of $2.2 billion to $12 billion.

2. Port-specific Impacts

Results for port-specific impacts are presented in Table A-16. Below, we discuss 2 related assessments that address diesel PM health risks near ports and railyards.

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[12]. 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.

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[12]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.

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 two key health risk assessments.[13] One 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,

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[13]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.

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 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 estimations of pollutant released from stationary and mobile sources. These inventories evolve over time as data are updated to reflect the most recent information available. 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.

The goods movement emissions inventory developed for this report contains estimates for a wide variety of categories, including ocean-going ships, commercial harbor craft, harbor dredging equipment, cargo handling equipment, trucks, locomotives, and transportation refrigeration units. Emissions estimates representing each of these categories were developed using relatively complex estimation techniques. This goods movement emissions inventory is still draft and is undergoing review. It is anticipated that refinements will be made, and that the inventory will be updated in the final Goods Movement Emissions Reduction Plan.

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). These 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 Children's Health Study (CHS) contained a component in which source contributions to ambient particles were determined for the year 1995. In this work, J. Schauer 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 ($\mu g/m^3$)					
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 $\mu g/m^3$. For secondary diesel PM, nitrates 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 sulfates, the health impacts associated with secondary diesel PM due to sulfates 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 2005).

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. 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 ozone standard staff report (2005), 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.

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.

5. 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. For example, we did not estimate incidences of hospitalizations for exacerbation of heart disease, chronic lung diseases (i.e., asthma and chronic obstructive pulmonary disease), or acute lung diseases (i.e., pneumonia and acute bronchitis).

In addition, estimates of the effects of PM on infant mortality, premature births, and low birth weight 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.

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.

6. 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 and asthma attacks. For the other health effects, we do not have a range in the unit valuation, so we are not able to calculate a quantitative estimate of the uncertainty in the unit valuation. Thus for three of the health endpoints - work loss days, hospitalization, and school absences - we did not quantify uncertainty.

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 international 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 Research Division 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 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 µg/m^3) and PM10 (199 µg/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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VII. Supplement

A. Nitrates and Sulfates

1. Calculation of Nitrates Population-weighted Exposures

In this report, staff modified the methodology used in year 2000 to address PM nitrates exposures in California. Staff updated the data base by adding monitoring data and improved the calculations to make the methodology more robust and replicable. In addition to the Statewide Routine Monitoring Network used in the previous work, staff included data from the special monitoring networks, IMPROVE and Children's Health Study (CHS), which were not available in 2000. (See map 1 – 3 for NO3 annual geometric mean and site location of Routine, IMPROVE, and CHS networks). The IMPROVE network provided additional information in the rural areas, while the CHS added more data to Southern California. Both the previous and the current methodologies were based on the Inverse Distance Weighting method. Both methods assigned weight to each monitor's annual geometric mean as a function of its distance from the point in space (for example, the centroid within each census tract) within the state, using an inverse distance weighting function (1/distance to a power). However, the power assigned to the distance was different in each method. The current methodology used a power of 2.5 in order to optimize the interpolations, whereas the previous methodology used a power of 2.0 (for distance squared). Further, the current methodology uses a minimum of 10 monitoring stations and up to a total of 15 in weighting the results to estimate the concentration at each census tract. In comparison, the previous methodology only used sites within a 50-kilometer radius, regardless of how many may fall within the fixed radius. After the interpolations were completed, the values were assigned to the affected populations within each census tract and averaged to obtain the population-weighted exposures. The previous methodology associated the interpolated concentrations to 1990 census populations while the current method uses year 2000 census. These differences account for a change in the statewide population-weighted exposures of approximately 0.45 µg/m^3 (2.25 µg/m^3 compared to previously derived value of 1.8 µg/m^3).

MAP 1. California

Redwood NP
0.13

Redding-Health Dept Roof
0.32

Lassen Volcanic NP
0.06

Red Bluff-Riverside Drive
0.66

Chico-Manzanita Avenue
0.32

Willows-E Laurel Street
0.68

Bliss SP-IPMP
0.08

Roseville-N Sunrise Blvd
0.83

Sacramento-T Street
1.08

Santa Rosa-5th Street
0.58

Napa-Jefferson Avenue
0.51

Point Reyes National
0.39

Vallejo-304 Tuolumne Street
0.56

Stockton-Hazelton Street
1.54

San Rafael
0.73

Concord-2175 Treat Blvd

Modesto-Lorled
1.34

San Francisco-Arkansas Street

Livermore-Old 1st Street
0.95

Fremont-Chapel Way

Redwood City
0.82

1.10

San Jose-Tully Road
1.04

Yosemite NP
0.14

Fresno-1st Street/Clovis-N Villa Avenue
21

Pinnacles NM
0.4

Sequoia NP
0.52

Hanford-S Irwin Street
1.59

Visalia-N Church Street
1.52

Corcoran-Patterson Avenue / Corcoran-Van Dorsten Avenue
1.85   1.08

China Lake-Powerline Road
0.39

Atascadero
1.34

Oildale-3311 Manor Street

Bakersfield-5558 California Avenue   53
1.70

San Luis Obispo-Marsh Street
0.77

Taft College
1.19

Mojave-923 Poole Street
0.61

Santa Maria-Santa Maria-Library
1.15 0.36

Lompoc
0.67

Lancaster
1.88

Santa Clarita-County Fire Station
1.14

San Gabriel

Crestline

Lake Arrowhead
1.99

El Rio-Rio Mesa School Rd
1.80

Burbank-W Palm Avenue 3.26 Azusa
0.99

Los Angeles-North Main Street
3.41

Redlands-Dearborn

San Bernardino-4th Street / Rubidoux / Banning-Alessandro
3.63

Hawthorne

North Hollywood
5.11

Perris
2.40 2.41

Indio-Jackson Street
1.53

2.17

Long Beach
3.01

Lake Elsinore 3.02

North Long Beach

El Toro
2.52   2.83

Oceanside-Mission Avenue
1.65

Brawley-Main Street
1.44

El Centro-9th Street
1.28

Calexico-Ethel Street
1.77

Alpine
.46

El Cajon-Redwood Avenue

Tijuana-TT

Tijuana-Las Palmas

Tijuana-La Mesa
3.49

Rosarito-Tijuana-Colef
3.00

NITRATES
1998

MAP 2. Central CA

Chico-Manzanita Avenue
0.52

Willows-E Laurel Street
0.68

Bliss SP (TRPA)
0.68

Roseville-N Sunrise Blvd
0.83

Sacramento-T Street
1.08

Santa Rosa-5th Street
0.58

Napa-Jefferson Avenue
0.51

Point Reyes National
0.39

Vallejo-304 Tuolumne Street
0.62

Bethel Island Road
0.76

Stockton-Wagner-Holt School
1.29

San Rafael
0.56

Concord-2975 Treat Blvd
0.73

Stockton-Hazelton Street
1.54

San Francisco-Arkansas Street
0.81

Livermore-Old 1st Street
0.95

Modesto-I Street
1.34

Yosemite NP
0.14

Fremont-Chapel Way
1.10

Redwood City
0.82

San Jose-Tully Road
1.04

Clovis-Villa Avenue
Fresno-1st Street 1.21

Pinnacles NM
0.44

Sequoia NP
0.52

Hanford-S Irwin Street
1.59

Visalia-N Church Street
1.52

Corcoran-Patterson Avenue Corcoran-Van Dorsten Avenue
1.85 1.08

Atascadero
1.34

Oildale-3311 Manor Street
1.53

Bakersfield-5558 California Avenue
1.70

San Luis Obispo-Marsh Street
0.77

Taft College
1.19

**NITRATES
1998**

Appendix A-83

MAP 2. South CA

Cochran-Patterson Avenue
1.35

China Lake-Powerline Road
0.39

Oildale-3311 Manor Street
1.53

Bakersfield-5558 California Avenue
1.70

...College
1.19

Mojave-923 Poole Street
0.61

Lancaster
1.88

Santa Clarita-County Fire Station
1.54

San Gabriel
0.99

Crestline
1.74

Lake Arrowhead
1.99

Sim Valley-Cochran Street
1.14

El Rio-Rio Mesa-School #0
1.40

Burbank-W Palm Avenue
3.26

Azusa Glendora
1.17

Upland Fontana-Arrow Highway San Bernardino-4th Street

Los Angeles-North Main Street
3.63

Ontario-Airport
.16

Redlands-Dearborn
4.34

Mira Loma Riverside-Rubidoux
3.44 Riverside 7.11

Hawthorne
3.35

Norco-Norconian
5.11

Riverside
7.19

Banning Airport 2.40
Banning-Alessandro

Palm Springs-Fire Station
1.53

Long Beach North Long Beach
4.09 3.12

Anaheim-Harbor Blvd
3.01

Perris
3.02

Indio-Jackson Street
2.17

El Toro
2.52

Lake Elsinore
2.83

Oceanside-Mission Avenue
1.65

Alpine
1.46

El Cajon-Redwood Avenue
1.45

Tijuana-ITT

Tijuana-Las Playas
3.09 Tijuana-La Mesa
2.47 3.49

Rosarito 2.43
Tijuana-Chef
3.00

NITRATES
1998

Appendix A-84

Redwood NP
0.36 0.36

Redding-Health Dept Roof
0.1

Lassen Volcanic NP
0.25 0.248

Red Bluff-Riverside Drive
0.1 0.866

Chico-Manzanita Avenue
0.1 0.769

Willows-E Laurel Street
0.1 0.754

Bliss SP (TRPA)
0.21 0.291

Roseville-N Sunrise Blvd
0.2 0.462

Sacramento-T Street
0.2 1.030

Santa Rosa-5th Street
0.3

Napa-Jefferson Avenue
0.3 0.847

Point Reyes National
San Rafael **0.4**

Vallejo-304 Tuolumne Street

Bethel Island Road
0.3

Stockton-Hazelton Street
0.2 1.382

Concord-2975 Treat Blvd
0.3

Livermore-Old 1st Street
0.3

Modesto-I Street
0.1 1.457

Yosemite NP
0.26 0.403

Redwood City
0.4 1.882
0.6

Fremont-Chapel Way

San Jose-Tully Road
0.3 0.983

Pinnacles NM
0.21 0.661

Fresno-1st Street
0.1

Clovis-N Villa Avenue
0.1

Sequoia NP 0.572
0.26

Hanford-S Irwin Street
0.1

Visalia-N Church Street
0.1 1.611

Corcoran-Patterson Avenue
0.1

Corcoran-Van Dorsten Avenue
0.1

China Lake-Powerline Road
0.1 0.932

Atascadero
0.21 0.681

San Luis Obispo-Marsh Street
0.25 1.243

Oildale-3311 Manor Street 1.809

Bakersfield-5558 California Avenue
0.1

Taft College
0.1 1.664

Mojave-923 Poole Street
0.1 1.172

Santa Maria-Santa Maria-Library
0.21 0.25

Lancaster
0.09 0.916

Lompoc
0.21 0.852

Santa Clarita-County Fire Station

El Rio-Rio Mesa School #2
1.996

Burbank-W Palm Avenue
Los Angeles-North Main Street

San Gabriel
0.26

Azusa
0.4

Upland
0.3

Crestline
0.4

Lake Arrowhead

Chino-Airport Redlands-Dearborn
Riverside-Rubidoux Airport Banning-Allesandro

Hawthorne
0.4

Norco-Norco
0.4 2.547

Perris
0.2 1.4

Indio-Jackson Street
0.2 2.006

Palm Springs-Fire Station
0.2

Long Beach
North Long Beach

El Toro

Lake Elsinore
0.17 1.494

Oceanside-Mission Avenue
0.6 2.410

Alpine 1.285
0.26

El Cajon-Redwood Avenue

Brawley-Main Street
0.2 1.741

El Centro-9th Street
0.2

Calexico-Ethel Street
0.2 2.117

Tijuana-TTT
Tijuana-La Mesa
Tijuana-Center of Health
0.25

Rosarito
Tijuana-Colef
0.4 3.179

SULFATES
1998

Background (Bold) and Monitored Data (Underlined)

Redding-Health Dept Roof
0.1 0.532

Lassen Volcanic NP
0.25 0.248

Red Bluff-Riverside Drive
0.1 0.866

Chico-Manzanita Avenue
0.1 0.769

Willows-E Laurel Street
0.1 0.754

Bliss SP (TRPA)
0.21 0.297

Roseville-N Sunrise Blvd
0.2 0.957

Sacramento-T Street
0.2 1.030

Santa Rosa-5th Street
0.3 0.753

Napa-Jefferson Avenue
0.3 0.847

Point Reyes National
0.51

Vallejo-304 Tuolumne Street
0.75 1.055

Bethel Island Road

Stockton-Wagner-Holt School

Stockton-Hazelton Street
0.2 1.382

San Rafael
0.4

Concord-2975 Treat Blvd
0.3 1.109

San Francisco-Arkansas Street
0.75 1.355

Livermore-Old 1st Street
0.3 1.006

Modesto-I Street
0.1 1.457

Yosemite NP
0.26 0.403

Fremont-Chapel Way
0.4 1.082

Redwood City
0.6 1.601

San Jose-Tully Road
0.3 0.983

Fresno-1st Street   Clovis-N Villa Avenue
0.1

Pinnacles NM
0.21 0.661

Sequoia NP
0.26 0.572

Hanford-S Irwin Street   Visalia-N Church Street
0.1 1.622   **0.1** 1.511

Corcoran-Patterson Avenue   Corcoran-Van Dorsten Avenue
0.1   **0.1**

China Lake-Powerline Road
0.1 0.932

Atascadero
0.21 0.681

Oildale-3311 Manor Street
0.1 1.809

San Luis Obispo-Marsh Street
0.25 1.243

Bakersfield-5558 California Avenue
0.1 1.832

Taft-College
0.1 1.664

Mojave-923 Poole Street
0.1 1.172

SULFATES
1998

Santa Maria-Santa Maria-Library
0.21 **0.25**

Lancaster
0.09 0.916

Background (Bold) and Monitored Data (Underlined)

Davis-N Villa Avenue
0.1

Sequoia NP
0.26 0.572

Hanford-S Irwin Street
0.1 1.622

Visalia-N Church Street
0.1 1.611

Corcoran-Patterson Avenue
0.1 Corcoran-Van Dorsten Avenue

China Lake-Powerline Road
0.1 0.932

Oildale-3311 Manor Street
0.1 1.809

Bakersfield-5558 California Avenue
0.1 1.832

Taft College
0.1 1.664

Mojave-923 Poole Street
0.1 1.172

Lancaster
0.09 0.916

Santa Clarita-County Fire Station
0.25 1.557

San Gabriel
0.26 0.613

Crestline
0.3 1.182

Simi Valley-Cochran Street
0.25 1.708

El Rio-Rio Mesa School #2
0.25 1.996

Burbank-W Palm Avenue
0.4 2.169

Azusa Glendora
0.3 0.26

Lake Arrowhead
0.924

0.3

Upland
0.26 Ontario-Airport
San Bernardino-4th Street **0.3**
Redlands-Dearborn
0.3 1.927

Los Angeles-North Main Street
0.4 2.573

Mira Loma
0.26 Riverside-Rubidoux
Riverside **0.2**
0.17

Banning Airport
Banning-Alesandro **0.2**
Palm Springs-Fire Station
0.2 1.518

Hawthorne
0.6 2.863

Norco-Norco
0.2

Anaheim-Harbor Blvd
0.4

Long Beach North Long Beach
0.6 0.51

Perris
0.2 1.725

Indio-Jackson Street
0.2 2.006

Lake Elsinore
0.17 1.484

El Toro
0.4 2.354

Oceanside-Mission Avenue
0.6 2.410

Brawley-Main Street
0.2 1.741

Alpine
0.26 1.285

El Centro-9th Street
0.2 1.787

El Cajon-Redwood Avenue
0.25 1.961

Calexico-Ethel Street
0.2 2.117

Tijuana-ITT
Tijuana-Las Playas **0.2** Tijuana-La Mesa
0.4 0.25 3.154

Rosarito
0.4 Tijuana-Colef
0.4 3.179

SULFATES
1998

Background (Bold) and Monitored Data (Underlined)

Appendix A-87

2. Methodology of Analyses of Nitrate and Sulfate Population-weighted Exposure

a) Introduction

Population-weighted exposure is the link between ambient pollutant concentrations and pollutant concentration-response functions that permits computation of public health impacts. Population-weighted exposure is the sum of potential individual exposures computed as the product of community population and community pollutant concentration. Long term health effects for particles containing sulfate (SO_4=) and/or nitrate (NO_3-) were computed based on the annual geometric means of measured concentrations of these ions, adjusted to mass assuming that ammonium nitrate (NH_4NO_3) and ammonium sulfate (($NH_4)_2 SO_4$) are the particulate chemical species.

This calculation is termed "potential" exposure because daily activity patterns influence an individual's actual exposure. For example, being inside a building will decrease a person's exposure to outdoor nitrate and sulfate concentrations, while a person who is outdoors may experience highly localized concentrations that are different from the community averages used in this study. Readers should bear in mind that the exposures presented here were computed to develop integrated regional values, and may not reflect all the local factors that would need to be considered to evaluate exposure at a particular location.

This exposure analysis is based solely on "outdoor" nitrate and sulfate data, as measured by the CARB and local Districts in the Statewide Routine Monitoring Network, supplemented by data from special monitoring networks such as the Federal Interagency Monitoring for Protected Visual Environments (IMPROVE) network and the Children's Health Study (CHS) monitoring program.

b) PM in California

Airborne particulate matter (PM) is not a single pollutant, but rather a mixture of primary and secondary particles. A large variety of emission source types, both natural and man-made, contribute to atmospheric levels of PM. Particles vary widely in size, shape, and chemical composition, and may contain inorganic ions, metallic compounds, elemental carbon (EC), organic carbon (OC), and mineral compounds from the earth's crust. PM changes as it ages in the atmosphere as directly emit PM ("primary" particles), becomes coated with the low-vapor-pressure products of atmospheric chemical reactions ("secondary" PM). Secondary PM typically contains compounds of ammonia (NH_3), oxides of sulfur (SO_X) and nitrogen (NO_X), and partially oxidized organic compounds (OC).

Generally, atmospheric PM can be divided into two distinct size classes - fine (<2.5 µm) and coarse (>2.5 µm). Fine and coarse particles differ in formation mechanisms, chemical composition, sources, and exposure relationships.

Fine PM is derived from combustion residue that has volatilized and then condensed to form primary PM, or from precursor gases reacting in the atmosphere to form secondary PM. Fine particles typically are comprised of sulfate, nitrate, ammonium, elemental carbon, organic compounds, and a variety of trace materials usually generated as combustion "fly ash."

Coarse particles, in contrast, are formed by crushing, grinding, and abrasion of surfaces, which breaks large pieces of material into smaller pieces. These particles are then suspended by wind or by activities such as construction, mining, vehicle traffic, and agriculture.

The spatial distribution of various PM sources, combined with diurnal and seasonal variations in meteorological conditions, cause the size, composition, and concentration of particulate matter to vary in space and time.

Sulfate

Sulfur dioxide (SO_2) 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 sulfates are also directly emitted from combustion or from industrial processes, but usually 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. The dominant form of sulfate in PM in California is ammonium sulfate (($NH_4)_2 SO_4$). Sulfate concentrations in the SoCAB are much greater than other areas of California. Historically, reduction in fuel sulfur content has lead to reductions in ambient SO_2 concentrations and less particulate sulfate formation.

Nitrate

In urban areas of California, nitrate represents a larger fraction of PM mass compared to the rest of the nation due to the State's widespread use of low-sulfur fuels for both 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 ammonium nitrate (NH_4NO_3).

In coastal areas, gas phase acids can react with sea salt by reaction of nitric acid (HNO_3) with sea salt particles ($NaCl$), producing stable particulate sodium nitrate ($NaNO_3$) accompanied by liberation of gaseous hydrochloric acid (HCl). This reaction is a principal source of coarse nitrate, and plays an important role in atmospheric chemistry because it is a permanent sink for gas-phase nitrogen oxide species.

Geometric Mean Mass

Particle concentration data commonly exhibit a skewed frequency distribution, with many low values and a few very high ones. For this reason it is standard practice to treat these data as log-normally distributed, and thus annual concentration statistics are reported as a geometric mean, which provides a better representation of "typical" concentrations than would an arithmetic mean.

3. Calculation of Nitrate and Sulfate Population-weighted Exposures

Concentrations of many air pollutants, including nitrate and sulfate, 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 geographically resolved. Population counts by census tract group block (typically a few thousand people) provide a convenient source of highly resolved population data.

Densely populated areas have many census tract group blocks, while sparsely populated areas have very few.

In order to compute a population-weighted exposure, the scattered measurements of PM must be converted to a form that allows assigning annual PM concentrations to all populated areas of the State. This was done using the Inverse Distance Weighting method implemented in the *Geostatistical Analyst 9.0* software package to interpolate PM concentrations down to the census block level. The nitrate and sulfate annual geometric mean values and population counts were associated by census tract group block and merged to assemble a spatially resolved population-weighted exposure estimate.

The interpolation procedure for assigning nitrate and sulfate concentrations to a census tract group block computed a weighted-average of the concentrations measured at 10 or more neighboring monitors. The weight assigned to each monitor was a function of its distance from the point being estimated, using an inverse distance weighting function of $1/d^{2.5}$. Using a weighting exponent of 2.5 forced the estimates to be strongly weighted to the closest monitors. For most points a minimum of 10 monitoring stations were used, with up to 15 used for some locations. Geographical barriers such as mountain ranges that may impede the movement of emissions and pollutants were not considered in the exposure calculations. While this may cause some rural estimates to be less accurate, this omission had little impact on the overall results since strongly weighted local monitors were available to drive the estimation for most of the State's population.

4. Nitrate and Sulfate Monitored Data

The PM nitrate and sulfate data used for the exposure calculation were gathered from a variety of routine and special monitoring program databases. Ambient data from 1998 were used because that year provided maximum spatial resolution for combined routine monitoring network and special study PM data. 1998 is considered representative of present air quality because major SO_X and NO_X source emissions have not changed significantly in recent years.

The PM data that were used in this study generally met EPA's minimum data completeness criterion (11 of 15 samples per calendar quarter or no more than 25% missing data). Three different data sets for 1998 were used to provide the ambient nitrate and sulfate concentrations.

- Size Selective Inlet (SSI) high volume sampler PM10 data. In 1998 the SSI sampling network consisted of 91 sites collecting PM10 and operating on a one-in-six day sampling schedule. Data completeness screening reduced the number of sites used in this study to 60. Compositional analysis of SSI filters provides the mass of nitrate and sulfate ions.

- Children's Health Study Two Week Sampler (TWS) PM2.5 data. 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 the study required robust but not highly time-resolved data, the TWS provides continuous sample collection reported as two-week average fine particle concentrations. The two-week sampling frequency provides 26 samples per site per year and is sufficient to determine seasonal as well as

annual mean concentrations. Because the TWS provides an integrated two-week measurement, and thus lacks the spikes that characterize short-term PM data, reported annual arithmetic means for TWS data were used without recalculation.

- Interagency Monitoring of Protected Visual Environments (IMPROVE) program data. The Federal IMPROVE program monitoring sites are located in federally protected Class 1 areas and are outside of urban areas. Data from 11 California sites operating in 1998 were used in this study.

- The California Dichotomous Sampler ("dichot") network data. The dichot sampler uses a low-volume PM10 inlet followed by a virtual impactor which separates the particles into two airstreams, one containing the PM2.5 (fine) fraction and the other the PM10-2.5 (coarse) fraction, with each collected on its own filter. The sum of PM2.5 and PM10-2.5 provides a measure of PM10. Samples were usually collected from midnight to midnight every sixth day.

- South Coast Air Quality Management District's (SCAQMD) Particulate Technical Enhancement Program (PTEP) data. The PTEP program operated at six sites (downtown Los Angeles, Anaheim, Diamond Bar, Rubidoux, Fontana, and San Nicolas Island) in southern California in 1995, collecting separate PM10 and PM2.5 samples. These data were used to fill gaps in the 1998 record and to assess PM2.5 / PM10 relationships.

<u>Combining PM10 and PM2.5 Nitrate and Sulfate Data</u>

The concentrations used in this study are a mixture of both PM10 and PM2.5 data. 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, ratios of annual PM10 to PM2.5 sulfate were computed from data from the PTEP data. Ratios of annual geometric mean PM2.5 sulfate to PM10 sulfate at these sites were in the range of 0.8 to 0.9. A similar relationship between PM10 nitrate and PM2.5 nitrate has also been observed at urban locations elsewhere in California. In order to maximize spatial coverage, because the probable error is small, and because site-specific correction factors were not available for most sites, PM10 and PM2.5 sulfate data were used in this study without adjusting for which size cut was reported at each monitoring site.

<u>Computing Sulfate and Nitrate PM Mass</u>

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.* anion, cation, and associated tightly bound water). The ammonium cation (NH_4^+) is the major cation for nitrate and sulfate ions in California, so mass was calculated assuming only ammonium nitrate and sulfate were present in the samples.

There is considerable uncertainty regarding the amount of water associated with ammonium nitrate and ammonium sulfate, but, since these compounds are fully saturated when inhaled into the moist conditions within the lung, no water correction was applied. For this study, the mass associated with only the ammonium, nitrate, and sulfate ions was computed by multiplying the nitrate values by the ratio of the molecular weight of the ammonium salt to the molecular weight of nitrate (1.29) or sulfate (1.38).

5. Background Estimation for PM Sulfate and Nitrate

Sulfate

Most airborne sulfate in California is due to anthropogenic sulfur emissions, but apportioning exposure to sulfur sources must take into account "background" sulfate from the two major sources unrelated to local combustion in California - biogenic sulfate generated over the ocean, and global transport of natural and anthropogenic sulfate in elevated layers of the atmosphere.

A rough estimate of the statewide distribution of background sulfate was constructed based on limited measurements from remote sites that are isolated from exposure to urban or industrial pollution, and reviewing results of model simulations of global sulfur processing in the atmosphere.

Unequivocal measurements of background sulfate are limited to a few weeks of data from sites in northern California. Oceanic sulfate data reviewed for this study come from Trinidad Head in Humboldt County, and global transport sulfate data come from Trinity Alps and Mount Lassen. Comparison with several years of routine monitoring data permitted estimation of the average annual concentration of background sulfate at these sites and extrapolation to other remote sites.

Computations of annual average background sulfate in the rest of the State were based on rough estimates of the effects of site-specific meteorology and terrain on inputs from the ocean and upper air.

The statewide estimates were reviewed for consistency with reported sulfate air quality data and published global sulfate model results, and adjusted if necessary.

Finally, the background estimates were subtracted from ambient data to approximate site-specific anthropogenic sulfate concentrations. Although there is considerable uncertainty in the background estimates, ambient concentrations at most urban sites in California are several times background, so that the impact of this uncertainty on statewide sulfate population exposure is believed to be small.

Nitrate

PM nitrate is generated from local emissions by a reversible chemical reaction that is dependent on temperature, relative humidity, and the concentrations of the precursor gases (ammonia and nitric acid). Long range transport of nitrate is generally weak because dispersion, heating, or drying of the air mass will cause ammonium nitrate to break down and return ist components to the gas phase. Small amounts of non-volatile nitrate can form by reaction of nitric acid with soil or sea salt, but limited measurements suggest that "background" concentrations are very low (generally less than 0.1 $\mu g/m^3$). For this reason, no effort was made to adjust measured nitrate values for a background contribution.

6. Uncertainty in Exposure Estimates

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 greatly accelerated 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.

Finally, there is uncertainty in these estimates of the secondary fraction of PM2.5 mass. For example, there was limited ambient speciated data in many areas, particularly rural areas. 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 methods provide reasonable estimates of the contribution of secondary PM in most of the heavily populated air basins, but the numbers reported here are not as precise as would be generated by a focused field and modeling program designed around the questions addressed in this study.

7. Data Sources and Uncertainty for Annual Geometric Means and Estimation of Background Concentrations

<u>Measurement Methods</u>

Routine monitoring for sulfate and nitrate in particles utilizes filter sampling and aqueous extraction for ion chromatographic analysis (IC).

This method is highly reliable for sulfate, which, once in particle form is chemically stable. In addition, virtually all sulfur in California PM10 and PM2.5 samples is in the form of sulfate, so that comparison of elemental sulfur analyses with sulfate ion analyses (SO_4= mass = S * 3) provides a "built-in" cross-check on the measurements.

Nitrate IC measurement quality is comparably to that for sulfate, but there are possible sources of sampling error in nitrate data. Particle ammonium nitrate is not chemically stable, but exists in equilibrium with the surrounding air. This equilibrium depends on gaseous concentrations of ammonia and nitric acid, and is also influenced by humidity and temperature. As a typical 24-hour filter sample is collected, these conditions can change, and thus the amount of nitrate on a filter can change, most often as previously collected particle nitrate returns to the gas phase and is lost from the filter, but it is also possible to add artifact nitrate as gas phase precursors react with material on the filter. Standard practice to control for nitrate loss or gain is to place a "denuder" upstream of the filter to remove gas-phase nitric acid from the air stream (preventing positive artifact), and a nylon "backup" filter behind the sampling filter, where volatilized nitric acid will chemically react with the nylon and be collected for measurement.

The sulfate and nitrate measurements from the IMPROVE network are typical of PM10 and PM2.5 filter measurements used in the current study. The IMPROVE Quality Assurance Plan's (IMPROVE, 2002) measurement objectives for these compounds are listed in the following table.

| IMPROVE Measurement Quality Objectives				
Method	Parameters	Precision*	Accuracy	MQL
PIXE	Elements S to Mn	±5%	±5%	1 - 4 ng/m3
IC	NO3, SO4, NH4	±5%	±5%	10 - 30 ng/m3

Uncertainty

The 5% uncertainty for an individual sample translates to less than 0.5% in computation of an annual mean for 78 samples (75% completeness in a typical 104 sample-day IMPROVE year). Applying the same measurement quality and completeness criterion to networks reporting only one-in-six day sampling (48 sample minimum) gives an uncertainty just under 1%.
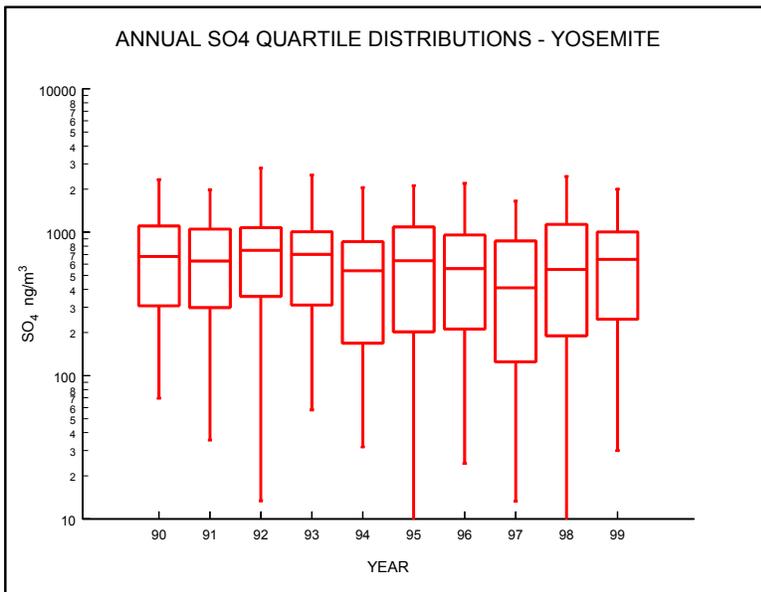
The largest uncertainty in using the annual mean of either sulfate or nitrate comes from interannual variability. As an example, IMPROVE annual distributions of SO_4 and NO3 data at Yosemite for the decade of the 1990s are shown in the following graphs.

ANNUAL SO4 QUARTILE DISTRIBUTIONS - YOSEMITE

ANNUAL NO3 QUARTILE DISTRIBUTIONS - YOSEMITE
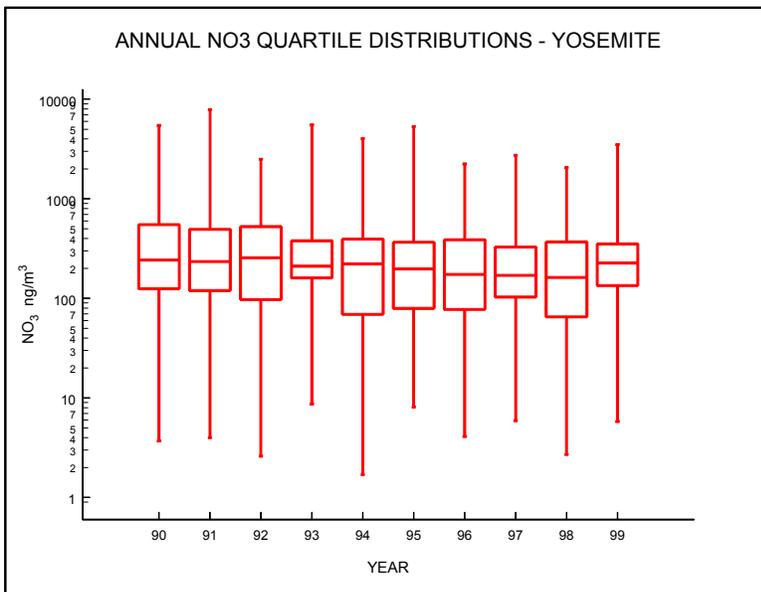
Since the Yosemite site is not located in an urban area, these data reflect the large impact of

meteorology on transport patterns and secondary particle formation. At an urban site, this kind of variation would be added to changes due to local activity around the site.

PM10 and PM2.5 Relative Composition

In order to get the greatest possible spatial coverage of chemically speciated PM data for this analysis, sulfate and nitrate data from both PM10 and PM2.5 measurements were used in the present study. Mixing data from differently size- limited sampling imposes some uncertainty in the analysis. The logic of this is based on the generalization that secondary PM species are concentrated in the fine (<2.5 μm) size fraction, so that collection of PM10, rather than PM2.5, should not significantly change the collected mass of these species. This assumption is supported by paired PM2.5 and PM10 sulfate data from the 1995 PTEP study as shown in the following table.

	San Nicolas Is.	Anaheim	Downtown LA	Diamond Bar	Fontana	Rubidoux	Mainland AVG	Land / Sea
PM 10								
NO3-	1.54	9.4	11.55	11.53	15.52	19.35	11.2	7.3
SO4=	1.96	4.54	5.19	4.24	3.92	4.39	3.7	1.9
PM 2.5								
NO3-	0.68	6	8.47	8.35	11	16.12	8.3	12.2
SO4=	1.4	3.79	4.63	3.88	3.66	3.53	3.2	2.3
RATIO PM2.5 / PM10								
NO3-	0.442	0.638	0.733	0.724	0.709	0.833	0.728	1.6
SO4=	0.714	0.835	0.892	0.915	0.934	0.804	0.876	1.2

These data demonstrate that, except in the immediate vicinity of the ocean, both sulfate and nitrate are preferentially formed in the fine particle phase. This is especially the case for urban sulfate, which averages 87% fine. Note also the reversal of this relationship for NO3 at San Nicolas Island, indicative of nitric acid reaction within coarse, wet sea salt particles - a sink not available away from the coast.

Background Estimation

Background for sulfate and nitrate were estimated based on a combination of reported analyses of source contributions to ambient concentrations at remote locations and reasonable assumptions based on sulfate and nitrate chemistry in the atmosphere.

Sulfate

The principal published PM background observations in California for global-scale transport are analyses of historical data from Mt. Lassen, CA and Crater Lake, OR, (VanCuren, 2003) and results from a short-term intensive field program in northwestern California (VanCuren *et al.,* 2005). These results were confirmed by comparison with published global sulfate model results (Chin and Jacob, 1996; Chin *et al,* 1996, Koch *et al.*, 1999). Oceanic sulfate concentrations were interpreted from unpublished analyses of particle sampling data from Trinidad Head (ITCT-2K2) and Point Reyes, CA (IMPROVE), and published data from San Nicolas Island, CA (Kim, Teffera, and Zeldin, 2000) and compared to global model results (Chin and Jacob, 1996; Chin *et al,* 1996, Koch *et al.*, 1999).

High altitude monitoring sites in the Sierra-Cascade range are regularly exposed to free tropospheric air flowing off the northern Pacific Ocean. During the prolonged dry summer season this air is marked by the presence of Asian dust which provides an unequivocal marker for this air mass. Analysis of 10 years of IMPROVE data from Mt. Lassen and Crater Lake (VanCuren, 2003) shows that samples associated with Asian dust have a typical PM2.5 sulfate concentration near 330 ng/m^3. Analysis of highly time resolved aerosol data from a 6-week period in spring 2002 (VanCuren et $al.$, 2005) used chemical markers to identify periods of downslope flow which delivered unmodified free tropospheric air to particle samplers; these showed a mean PM10 sulfate concentration of 408 ng/m^3, of which about 88% (360 ng/m^3) were below 2.5 μm in diameter. Model estimates of surface sulfate in this region were 400-1700 ng/m^3. Based on these observations we estimate annual average tropospheric "background" sulfate over California is about 300 – 500 ng/m^3. Adjusting for upslope winds, inversions, and other factors, we estimate annual average background sulfate at high altitude surface sites to be around 300 ng/m^3.

The largest natural source of sulfate aerosol is heterogeneous oxidation of biogenic SO_2 and methyl sulfonic acid (MSA) produced by photosynthesis in the upper layers of the ocean. Sulfate levels produce by these processes are controlled by sunlight, nutrient supply, and atmospheric conversion rates oxidizing sulfur compounds to sulfate. Measurements at Trinidad Head showed mean PM10 sulfate to be about 1.6 µg/m^3, with about 1.1 µg/m^3 PM2.5. This is comparable to the observed annual sulfate burden at Point Reyes (1.1 µg/m^3 PM2.5) Coastal sites some distance inland report concentrations about half of the beach zone (Redwood National Park, CA annual burden about 0.6 µg/m^3 PM2.5. Based on these data, beach line background sulfate concentrations were estimates as 1 µg/m^3 PM2.5, and inland sites adjusted downward to reflect increased mixing height with distance from the ocean, the effects of sea-breeze / land breeze flow, and seasonal variations.

Nitrate

The volatile nature of nitrate makes it generally short lived, and thus there is little "background" nitrate in the free troposphere. Some stable nitrate is formed by reaction of nitric acid with mineral dust (Gong et $al.$, 2003), and there is a small amount formed in the marine boundary layer by reaction of natural nitric acid with sea salt (in the shore zone in populated areas, most of this reaction is driven by NO_X emissions from anthropogenic sources).

There is little information on global nitrate except as generated by specialized transport models (Gong et $al.$, 2003). Published PM background observations in California for global-scale transport (VanCuren, 2003) show tropospheric background nitrate to be on the order of 0.1 – 0.2 µg/m^3 PM2.5. Nitrate measurements at Trinidad Head associated with strong on-shore winds (to suppress local NO_X emission effects) are less than 0.1 µg/m^3 PM2.5. These values are comparable to those reported in transport models (Gong et $al.$, 2003).

Based on these observations, nitrate values used in this study were not corrected for nitrate from global transport and oceanic processes.

8. References:

Chin, M., and D. Jacob, Anthropogenic and natural contributions to tropospheric sulfate: a global model analysis, *J. Geophys. Res.* 101(D13), 1996.

Chin, M., D. Jacob, G. Gardner, M. Foreman-Fowler, Pl Spiro, A global three-dimensional model of tropospheric sulfate, *J. Geophys. Res.* 101(D13), 1996.

Koch D., D. Jacob, I. Tegen, D. Rind, M. Chin, Tropospheric sulfur simulation and sulfate direct radiative forcing in the Goddard Institute for Space Studies general circulation model. *J. Geophys. Res*. 104(D19), 1999.

Gong, S. L., X. Y. Zhang, T. L. Zhao, I. G. McKendry, D. A. Jaffe, and N. M. Lu, Characterization of soil dust aerosol in China and its transport and distribution during 2001 ACE-Asia: 2. Model simulation and validation, *J. Geophys. Res.,* 108(D9), 2003.

VanCuren, R., Asian aerosols in North America: Extracting the chemical composition and mass concentration of the Asian continental aerosol plume from long-term aerosol records in the western United States, *J. Geophys. Res.*, 108(D20), 2003.

VanCuren, R., and T. Cahill, Asian aerosols in North America: Frequency and concentration of fine dust, *J. Geophys. Res.* 107(D24), 2002.

B. Calculation Protocol

Below, we provide the SAS program used to calculate health impacts associated with exposures to DPM. Similar programs for calculating health impacts associated with exposures to nitrates or ozone are also available.

```
/* goods mvt plan
   primary diesel pm using ab info (Pope 2002 for death)
   file effect_dpm_v4_112805.sas                11/28/05, h.t  */

libname gmp 'C:\My Documents\CARB\GoodsMovtPlan\PrimaryDPM';
libname gm 'C:\My Documents\CARB\GoodsMovtPlan';

/**** PART A: primary DPM, year 2005   *************************/

/*  Section A.1: year 2005, premature death */

/* STEP 1: get emissions data & health effects to calculate factors */

/* import health effects */
PROC IMPORT OUT= WORK.effect1
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\PrimaryDPM\1 LT
mortality diesel PM by basin Pope.xls"
            DBMS=EXCEL2000 REPLACE;
     GETNAMES=YES;
RUN;
/* import all emissions
PROC IMPORT OUT= WORK.allems
            DATAFILE= "C:\My
Documents\CARB\GoodsMovtPlan\GM_ALL_EMS_112805.xls"
            DBMS=EXCEL2000 REPLACE;
     GETNAMES=YES;
RUN;
*/
/* calculate ab emissions */
data allems;
set gm.gm_all_ems_adj_112805;
run;

proc sort data=allems out=temp100;
by ab poln;
run;
proc univariate data=temp100 noprint;
by ab poln;
var ems2000 ems2005 ems2010 ems2015 ems2020 ems2025;
output out=allems_ab sum=allems2000 allems2005 allems2010 allems2015
allems2020 allems2025;
run;

/* STEP2: calculate factors */

/* get ab factors for health effect 1: mortality */
data allems_ab_dpm;
set allems_ab;
if poln='DPM';
run;
proc sort data=effect1 out=temp101;
```

```sas
by ab;
run;
data temp102;
set temp101;
if ab='SFB' then ab='SF';
run;
data temp103;
merge temp102 allems_ab_dpm;
by ab;
run;

data factors1;
set temp103;
factor1_lower=allems2000*365/effect_lower;
factor1_mean=allems2000*365/effect_mean;
factor1_upper=allems2000*365/effect_upper;
drop allems2005 allems2010 allems2015 allems2020 allems2025;
run;
/* STEP 3: get population for each year */

PROC IMPORT OUT= WORK.pop_all
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\pop by coabdis
1995-2050.dbf"
            DBMS=DBF REPLACE;
     GETDELETED=NO;
RUN;


/* calculate ab populations */
proc sort data=pop_all out=temp120;
by ab ;
run;
proc univariate data=temp120 noprint;
by ab ;
var p2000 p2005 p2010 p2015 p2020 p2025;
output out=pop_ab sum=p2000 p2005 p2010 p2015 p2020 p2025;
run;

/* STEP 4: get emissions due to GM */
/*PROC IMPORT OUT= WORK.gmems
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\GM_EMS_111805.xls"
            DBMS=EXCEL2000 REPLACE;
     GETNAMES=YES;
RUN;*/
data gmems;
set gm.gm_ems_adj_112805;
run;

/* calculate ab emissions */
proc sort data=gmems out=temp110;
by ab type poln;
run;
proc univariate data=temp110 noprint;
by ab type poln;
var ems2000 ems2005 ems2010 ems2015 ems2020 ems2025;
output out=gmems_ab sum=ems2000 ems2005 ems2010 ems2015 ems2020 ems2025;
run;
```

Appendix A-99

```
data gmems_ab_dpm;
set gmems_ab;
if poln='DPM';
run;


/* merge GM emissions w/ populations and factors */

data combine_1;
merge gmems_ab_dpm pop_ab factors1;
by ab;
run;

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_1;
gm_effect_2005=(ems2005*365/factor1_mean)*(p2005/p2000);
gm_effect_2005_lower=(ems2005*365/factor1_lower)*(p2005/p2000);
gm_effect_2005_upper=(ems2005*365/factor1_upper)*(p2005/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC     ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;        /* use ab1 here if want only major 5 basins */
run;
proc univariate data=temp500 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;
data gm_effect_type_ab_1;
set gm_effect_type_ab;
year=2005;
endpoint='1_death_pope';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;
```

Appendix A-100

```sas
/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
output out=gm_effect_sw sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_1
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2005.xls"
            DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2005.xls"
            DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2005.xls"
            DBMS=EXCEL REPLACE;
                 SHEET="statewide";
RUN;

PROC EXPORT DATA= factors1
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2005.xls"
            DBMS=EXCEL REPLACE;
                 SHEET="factors";
RUN;

/******  Section A.2: Year 2005, asthma attacks ***********/

/* import health effects */
PROC IMPORT OUT= WORK.effect2
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\PrimaryDPM\11
Asthma diesel PM by basin.xls"
            DBMS=EXCEL2000 REPLACE;
     GETNAMES=YES;
RUN;

/* STEP 2: calculate factors */

/* get ab factors for health effect */
data allems_ab_dpm;
set allems_ab;
if poln='DPM';
run;
proc sort data=effect2 out=temp101;
by ab;
run;
data temp102;
set temp101;
```

Appendix A-101

```sas
if ab='SFB' then ab='SF';
run;
data temp103;
merge temp102 allems_ab_dpm;
by ab;
run;

data factors2;
set temp103;
factor1_lower=allems2000*365/effect_lower;
factor1_mean=allems2000*365/effect_mean;
factor1_upper=allems2000*365/effect_upper;
drop allems2005 allems2010 allems2015 allems2020 allems2025;
run;
/* STEP 3,4: use population & GM emissions already created for basins */

/* merge GM emissions w/ populations and factors */

data combine_2;
merge gmems_ab_dpm pop_ab factors2;
by ab;
run;

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_2;
gm_effect_2005=(ems2005*365/factor1_mean)*(p2005/p2000);
gm_effect_2005_lower=(ems2005*365/factor1_lower)*(p2005/p2000);
gm_effect_2005_upper=(ems2005*365/factor1_upper)*(p2005/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;
data gm_effect_type_ab_2;
set gm_effect_type_ab;
year=2005;
endpoint='2_asthma';
poln='DPM';
run;
```

```
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
output out=gm_effect_sw sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_2
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2005.xls"
            DBMS=EXCEL REPLACE;
            SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2005.xls"
            DBMS=EXCEL REPLACE;
            SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2005.xls"
            DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors2
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2005.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/****** Section A.3: Year 2005, WLD ***********/

/* import health effects */
PROC IMPORT OUT= WORK.effect3
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\PrimaryDPM\12 WLD
diesel PM by basin.xls"
            DBMS=EXCEL2000 REPLACE;
    GETNAMES=YES;
RUN;

/* STEP 2: calculate factors */
```

```
/* get ab factors for health effect */
data allems_ab_dpm;
set allems_ab;
if poln='DPM';
run;
proc sort data=effect3 out=temp101;
by ab;
run;
data temp102;
set temp101;
if ab='SFB' then ab='SF';
run;
data temp103;
merge temp102 allems_ab_dpm;
by ab;
run;

data factors3;
set temp103;
factor1_lower=allems2000*365/effect_lower;
factor1_mean=allems2000*365/effect_mean;
factor1_upper=allems2000*365/effect_upper;
drop allems2005 allems2010 allems2015 allems2020 allems2025;
run;
/* STEP 3,4: use population & GM emissions already created for basins */

/* merge GM emissions w/ populations and factors */

data combine_3;
merge gmems_ab_dpm pop_ab factors3;
by ab;
run;

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_3;
gm_effect_2005=(ems2005*365/factor1_mean)*(p2005/p2000);
gm_effect_2005_lower=(ems2005*365/factor1_lower)*(p2005/p2000);
gm_effect_2005_upper=(ems2005*365/factor1_upper)*(p2005/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC     ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
```

```sas
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;
data gm_effect_type_ab_3;
set gm_effect_type_ab;
year=2005;
endpoint='3_WLD';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
output out=gm_effect_sw sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_3
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2005.xls"
            DBMS=EXCEL REPLACE;
            SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2005.xls"
            DBMS=EXCEL REPLACE;
            SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2005.xls"
            DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors3
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2005.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;
```

```
/******  Section A.4: Year 2005, MRAD ***********/

/* import health effects */
PROC IMPORT OUT= WORK.effect4
            DATAFILE= "C:\My Documents\CARB\GoodsMovtPlan\PrimaryDPM\13 MRAD
diesel PM by basin.xls"
            DBMS=EXCEL2000 REPLACE;
     GETNAMES=YES;
RUN;

/* STEP 2: calculate factors */

/* get ab factors for health effect */
data allems_ab_dpm;
set allems_ab;
if poln='DPM';
run;
proc sort data=effect4 out=temp101;
by ab;
run;
data temp102;
set temp101;
if ab='SFB' then ab='SF';
run;
data temp103;
merge temp102 allems_ab_dpm;
by ab;
run;

data factors4;
set temp103;
factor1_lower=allems2000*365/effect_lower;
factor1_mean=allems2000*365/effect_mean;
factor1_upper=allems2000*365/effect_upper;
drop allems2005 allems2010 allems2015 allems2020 allems2025;
run;
/* STEP 3,4: use population & GM emissions already created for basins */

/* merge GM emissions w/ populations and factors */

data combine_4;
merge gmems_ab_dpm pop_ab factors4;
by ab;
run;

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_4;
gm_effect_2005=(ems2005*365/factor1_mean)*(p2005/p2000);
gm_effect_2005_lower=(ems2005*365/factor1_lower)*(p2005/p2000);
gm_effect_2005_upper=(ems2005*365/factor1_upper)*(p2005/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
```

```
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;
data gm_effect_type_ab_4;
set gm_effect_type_ab;
year=2005;
endpoint='4_MRAD';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2005_lower gm_effect_2005 gm_effect_2005_upper;
output out=gm_effect_sw sum=gm_effect_2005_lower gm_effect_2005
gm_effect_2005_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_4
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2005.xls"
           DBMS=EXCEL REPLACE;
           SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2005.xls"
           DBMS=EXCEL REPLACE;
           SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2005.xls"
```

Appendix A-107

```
                DBMS=EXCEL REPLACE;
                     SHEET="statewide";
RUN;

PROC EXPORT DATA= factors4
             OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2005.xls"
             DBMS=EXCEL REPLACE;
                     SHEET="factors";
RUN;

/**** PART B: primary DPM, year 2010   ***************/

/*  Section B.1: year 2010, premature death */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_1;
gm_effect_2010=(ems2010*365/factor1_mean)*(p2010/p2000);
gm_effect_2010_lower=(ems2010*365/factor1_lower)*(p2010/p2000);
gm_effect_2010_upper=(ems2010*365/factor1_upper)*(p2010/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC     ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;
data gm_effect_type_ab_1b;
set gm_effect_type_ab;
year=2010;
endpoint='1_death_pope';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab;
```

Appendix A-108

```sas
        output out=gm_effect_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
output out=gm_effect_sw sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_1b
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2010.xls"
            DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2010.xls"
            DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2010.xls"
            DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors1
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2010.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/*  Section B.2: year 2010, asthma attacks */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_2;
gm_effect_2010=(ems2010*365/factor1_mean)*(p2010/p2000);
gm_effect_2010_lower=(ems2010*365/factor1_lower)*(p2010/p2000);
gm_effect_2010_upper=(ems2010*365/factor1_upper)*(p2010/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
```

```sas
     else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;
data gm_effect_type_ab_2b;
set gm_effect_type_ab;
year=2010;
endpoint='2_asthma';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
output out=gm_effect_sw sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_2b
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2010.xls"
           DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;
```

Appendix A-110

```
PROC EXPORT DATA= factors2
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2010.xls"
            DBMS=EXCEL REPLACE;
                 SHEET="factors";
RUN;


/*  Section B.3: year 2010, WLD */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_3;
gm_effect_2010=(ems2010*365/factor1_mean)*(p2010/p2000);
gm_effect_2010_lower=(ems2010*365/factor1_lower)*(p2010/p2000);
gm_effect_2010_upper=(ems2010*365/factor1_upper)*(p2010/p2000);
run;


/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;


proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;
data gm_effect_type_ab_3b;
set gm_effect_type_ab;
year=2010;
endpoint='3_WLD';
poln='DPM';
run;

/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
```

```sas
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
output out=gm_effect_sw sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;


/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_3b
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;


PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2010.xls"
           DBMS=EXCEL REPLACE;
               SHEET="statewide";
RUN;


PROC EXPORT DATA= factors3
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2010.xls"
           DBMS=EXCEL REPLACE;
               SHEET="factors";
RUN;


/*  Section B.4: year 2010, MRAD  */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine 4;
gm_effect_2010=(ems2010*365/factor1_mean)*(p2010/p2000);
gm_effect_2010_lower=(ems2010*365/factor1_lower)*(p2010/p2000);
gm_effect_2010_upper=(ems2010*365/factor1_upper)*(p2010/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
```

```
proc univariate data=temp500 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;
data gm_effect_type_ab_4b;
set gm_effect_type_ab;
year=2010;
endpoint='4_MRAD';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2010_lower gm_effect_2010 gm_effect_2010_upper;
output out=gm_effect_sw sum=gm_effect_2010_lower gm_effect_2010
gm_effect_2010_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_4b
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2010.xls"
           DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2010.xls"
           DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors4
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2010.xls"
           DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;
```

```
/**** PART C: primary DPM, year 2020   ****************/

/*  Section C.1: year 2020, premature death */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_1;
gm_effect_2020=(ems2020*365/factor1_mean)*(p2020/p2000);
gm_effect_2020_lower=(ems2020*365/factor1_lower)*(p2020/p2000);
gm_effect_2020_upper=(ems2020*365/factor1_upper)*(p2020/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;
data gm_effect_type_ab_1c;
set gm_effect_type_ab;
year=2020;
endpoint='1_death_pope';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
output out=gm_effect_sw sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;
```

```
/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_1c
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2020.xls"
            DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2020.xls"
            DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2020.xls"
            DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors1
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_death_2020.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/*  Section C.2: year 2020, asthma attacks */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_2;
gm_effect_2020=(ems2020*365/factor1_mean)*(p2020/p2000);
gm_effect_2020_lower=(ems2020*365/factor1_lower)*(p2020/p2000);
gm_effect_2020_upper=(ems2020*365/factor1_upper)*(p2020/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB  ';
else if ab='SD' then ab1='3_SD   ';
else if ab='SJV' then ab1='4_SJV  ';
else if ab='SV' then ab1='5_SV   ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
```

```sas
run;
data gm_effect_type_ab_2c;
set gm_effect_type_ab;
year=2020;
endpoint='2_asthma';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
output out=gm_effect_sw sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_2c
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2020.xls"
           DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2020.xls"
           DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2020.xls"
           DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors2
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_asthma_2020.xls"
           DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/*  Section C.3: year 2020, WLD */

/* STEP 5: calculate GM health impacts by category */
```

```
data effect_ab;
set combine_3;
gm_effect_2020=(ems2020*365/factor1_mean)*(p2020/p2000);
gm_effect_2020_lower=(ems2020*365/factor1_lower)*(p2020/p2000);
gm_effect_2020_upper=(ems2020*365/factor1_upper)*(p2020/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC     ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;
data gm_effect_type_ab_3c;
set gm_effect_type_ab;
year=2020;
endpoint='3_WLD';
poln='DPM';
run;
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
output out=gm_effect_sw sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_3c
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2020.xls"
           DBMS=EXCEL REPLACE;
    SHEET="type_ab";
RUN;
```

Appendix A-117

```
PROC EXPORT DATA= gm_effect_ab
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2020.xls"
            DBMS=EXCEL REPLACE;
      SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2020.xls"
            DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors3
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_wld_2020.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/*  Section C.4: year 2020, MRAD  */

/* STEP 5: calculate GM health impacts by category */

data effect_ab;
set combine_4;
gm_effect_2020=(ems2020*365/factor1_mean)*(p2020/p2000);
gm_effect_2020_lower=(ems2020*365/factor1_lower)*(p2020/p2000);
gm_effect_2020_upper=(ems2020*365/factor1_upper)*(p2020/p2000);
run;

/* sum by category by ab */
data effect_ab; set effect_ab;
if ab='SC' then ab1='1_SC    ';
else if ab='SF' then ab1='2_SFB   ';
else if ab='SD' then ab1='3_SD    ';
else if ab='SJV' then ab1='4_SJV   ';
else if ab='SV' then ab1='5_SV    ';
else ab1='6_Others';
run;

proc sort data=effect_ab out=temp500;
by ab type;
run;
proc univariate data=temp500 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab type;
output out=gm_effect_type_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;
data gm_effect_type_ab_4c;
set gm_effect_type_ab;
year=2020;
endpoint='4_MRAD';
poln='DPM';
run;
```

```
/* sum across categories for each ab */
proc sort data=effect_ab out=temp510;
by ab;
run;
proc univariate data=temp510 noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
by ab;
output out=gm_effect_ab sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* sum across basins for statewide totals */
proc univariate data=gm_effect_ab noprint;
var gm_effect_2020_lower gm_effect_2020 gm_effect_2020_upper;
output out=gm_effect_sw sum=gm_effect_2020_lower gm_effect_2020
gm_effect_2020_upper;
run;

/* STEP 6:  export files */
PROC EXPORT DATA= gm_effect_type_ab_4c
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2020.xls"
           DBMS=EXCEL REPLACE;
     SHEET="type_ab";
RUN;

PROC EXPORT DATA= gm_effect_ab
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2020.xls"
           DBMS=EXCEL REPLACE;
     SHEET="ab";
RUN;
PROC EXPORT DATA= gm_effect_sw
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2020.xls"
           DBMS=EXCEL REPLACE;
                SHEET="statewide";
RUN;

PROC EXPORT DATA= factors4
           OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_mrad_2020.xls"
           DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

/*  PART D: FINAL OUTPUTS   */

/* One composite table */

data results_all;
set gm_effect_type_ab_1(rename=(gm_effect_2005=mean
gm_effect_2005_lower=lower gm_effect_2005_upper=upper))
gm_effect_type_ab_2(rename=(gm_effect_2005=mean gm_effect_2005_lower=lower
gm_effect_2005_upper=upper))
gm_effect_type_ab_3(rename=(gm_effect_2005=mean gm_effect_2005_lower=lower
gm_effect_2005_upper=upper))
```

Appendix A-119

```
gm_effect_type_ab_4(rename=(gm_effect_2005=mean gm_effect_2005_lower=lower
gm_effect_2005_upper=upper))
gm_effect_type_ab_1b(rename=(gm_effect_2010=mean gm_effect_2010_lower=lower
gm_effect_2010_upper=upper))
gm_effect_type_ab_2b(rename=(gm_effect_2010=mean gm_effect_2010_lower=lower
gm_effect_2010_upper=upper))
gm_effect_type_ab_3b(rename=(gm_effect_2010=mean gm_effect_2010_lower=lower
gm_effect_2010_upper=upper))
gm_effect_type_ab_4b(rename=(gm_effect_2010=mean gm_effect_2010_lower=lower
gm_effect_2010_upper=upper))
gm_effect_type_ab_1c(rename=(gm_effect_2020=mean gm_effect_2020_lower=lower
gm_effect_2020_upper=upper))
gm_effect_type_ab_2c(rename=(gm_effect_2020=mean gm_effect_2020_lower=lower
gm_effect_2020_upper=upper))
gm_effect_type_ab_3c(rename=(gm_effect_2020=mean gm_effect_2020_lower=lower
gm_effect_2020_upper=upper))
gm_effect_type_ab_4c(rename=(gm_effect_2020=mean gm_effect_2020_lower=lower
gm_effect_2020_upper=upper))
;
run;
PROC EXPORT DATA= results_all
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_all.xls"
            DBMS=EXCEL REPLACE;
                SHEET="factors";
RUN;

data gmp.results_dpm_all; set results_all; run;

proc sort data=gmp.results_dpm_all out=temp800;
by year endpoint type;
run;
proc univariate data=temp800 noprint;
var lower mean upper;
by year endpoint;
output out=gm_dpm_yr_endpoint sum=lower mean upper n=n1 n2 n3;
run;
PROC EXPORT DATA=gm_dpm_yr_endpoint
            OUTFILE= "C:\My
Documents\CARB\GoodsMovtPlan\PrimaryDPM\gm_dpm_yr_endpoint.xls"
            DBMS=EXCEL REPLACE;
                SHEET="DPM_112805";
RUN;

data gmp.gm_dpm_yr_endpoint_112805; set gm_dpm_yr_endpoint; run;

/* end of 11/28/05, h.t */
```

Appendix A-120

The next table shows the basin-specific factors (tons per case of health endpoint) used in calculating the health impacts related to PM exposures. Details on how these factors are used can be found in the Health Impacts Methodology Section E of this Appendix.

Air Basin	Premature Death		Asthma Attack		Work Loss Days		MRAD	
	DPM	NOx	DPM	NOx	DPM	NOx	DPM	NOx
GBV	257	2004	14.0	116	1.6	13.4	0.31	2.58
LC	79	1240	6.2	96	0.8	12.1	0.15	2.33
LT	30	3397	3.7	422	0.4	46.5	0.08	8.94
MC	29	1313	3.0	130	0.3	15.2	0.07	2.93
MD	85	862	4.1	40	0.5	4.8	0.09	0.93
NC	96	3283	6.3	213	0.7	24.3	0.14	4.68
NCC	30	1681	1.3	74	0.1	8.3	0.03	1.61
NEP	79	5107	5.1	322	0.6	36.4	0.11	7.00
SC	6	191	0.3	9	0.0	1.1	0.01	0.21
SCC	21	789	1.0	38	0.1	4.5	0.02	0.86
SD	12	312	0.6	15	0.1	1.8	0.01	0.34
SF	12	959	0.6	48	0.1	5.2	0.01	1.00
SJV	28	1058	1.4	54	0.2	6.6	0.03	1.27
SS	17	567	0.9	30	0.1	3.7	0.02	0.72
SV	21	1080	1.2	60	0.1	7.0	0.03	1.34

C. Scientific Peer Review Comments and CARB Staff Responses

Comments on the draft methodology document (see Supplement) sent on November 10 to scientific peer reviewers are given verbatim (except as noted in brackets) below along with CARB staff responses (in italics).

Comments from Professor John Balmes (University of California at San Francisco)

[Professor Balmes was contacted by phone and told of CARB staff's plan to use the Pope et al. (2002) associations between PM2.5 and premature death rather than Krewski et al. (2001), and to use Jerrett et al. (2005) as a sensitivity test. He concurred.]

CARB Staff Response: We proceeded as recommended.

Comments from Professor John Froines (University of California at Los Angeles)

[Addressed to Cal/EPA Secretary Lloyd – Co-signed by Professor John R. Froines (UCLA), Edward Avol, M.S. (USC), and Professor Michael Jerrett (USC).]

The purpose of this letter is to comment on the current process of developing a draft analysis for "Death and disease estimates associated with goods movement in California". I would have preferred talking with you directly but I understand you are in China with the Governor. I have appreciated your inclusion of me and other scientists in the review process; I consider participation by academic scientists to be crucial to developing the most scientifically sound document to address this major social, economic and policy issue which will have widespread ramifications for the future of California especially in the Southern California region. To develop the best response to the proposed methodology document and subsequent draft I decided that a collective effort by scientists would be most valuable and as a result I contacted Arthur Winer (UCLA), Michael Jerrett (USC), who recently published a paper on increased mortality from PM2.5 in the LA area, and Nino Kuenzli (USC), whose expertise is burden of disease analysis. We had a conference call Tuesday [November 15] to discuss the methodology document and the overall process. I have also received input from Ed Avol (USC) who has expertise regarding port emission inventories/reduction strategies, based on his efforts with the Port of LA and the NO Net Increase Task Force. Our conclusions follow:

1. Everyone expressed high regard for CARB/OEHHA scientists/professionals who are working on the analysis. We think excellent work is being conducted under difficult circumstances and respect that effort.

CARB Staff Response: One note of clarification. While we rely primarily on peer-reviewed literature reviews, analyses, and methodologies for health effects previously conducted by OEHHA scientists, the goods movement risk assessment is being conducted by CARB staff scientists with expertise in emissions, exposure, health, and economic valuation. OEHHA staff will provide internal scientific peer review for the assessment.

2. Overall, there was general dissatisfaction with the "Proposed methodology" document. Everyone expressed similar views that the document is extremely difficult to

evaluate given its limited nature. The document does not provide sufficient information for an adequate scientific evaluation and overall is not clear. The four of us all have significant questions which are not addressed in the document. This is problematic and has implications for the value of the subsequent document being developed.

CARB Staff Response: The 'Proposed Methodology' document was a first draft written to give potential scientific peer reviewers an overview of the scope of the risk assessment, information how to access the approximately ten existing risk assessments on diesel sources and goods movement facilities already conducted by CARB staff, and planned enhancements to include pollutants, sources, and health outcomes not included in the previous analyses. We did not ask the scientific peer reviewers to pre-endorse the methodology without seeing the details and results (i.e., the methodology document is six pages versus the over one hundred pages of this document), but rather to give us as much advance notice as possible of specific concerns with our planned approach. We noted that we expected the methodology to evolve as we received comments from peer reviewers and as the analysis proceeds, which has been the case. Without specific questions, we cannot respond in more detail to this comment. However, we had contacted Professors Jerrett and Winer independently and their specific questions and comments are included and responded to below.

3. The lack of transparency of the methodology document raises serious questions about whether the analysis to be completed in about a week will be comprehensive in its content and adequately assess emissions, exposure and the anticipated health risks associated with the goods movement. Given what we have seen so far there is general concern about the potential underestimation of health risks associated with proposed goods movement policies.

CARB Staff Response: Actually, the analysis took about a dozen CARB staff three weeks (including evenings and weekends) to conduct, not one week. Again, without specific questions (as provided by Professors Jerrett and Winer), we cannot respond in more detail to this comment.

4. It is not apparent to us why there is such a tight timetable for completion of a major document that will affect the health of millions of Californians in the future. There has been major research on the health effects of air pollution conducted in California in the past decade including considerable work supported by CARB. That research has demonstrated new health outcomes at current exposure levels and reinforced our understanding of the major issues associated with exposure to air pollution in the Los Angeles Basin. There are major control and technology issues to be addressed even at current levels, and the expansion of a major transportation sector will have major implications beyond our existing concerns. A careful and thoughtful analysis of the potential human and economic consequences is required if we are to avoid adverse health consequences.

Chronic disease is difficult to measure epidemiologically and given the health endpoints including cancer, cardiovascular disease, neurological, immunological and developmental disorders, as well as allergic airway disease including asthma it will be extremely problematic to accurately assess the true impact of expanded goods movement in coming decades on the health status of exposed populations in any timely

fashion. The current approach means that we may end up assessing the death and disease, that is, the health consequences many, many years after the social/economic decisions have been made. This means that we should take the time to do the best possible job on the potential health risks and not be rushed into decisions based on incomplete information and evaluation. A longer timeframe is required.

CARB Staff Response: It is important to quantify the health effects of goods movement now (with proper acknowledgment of caveats, uncertainties, and unquantified risks) so that ongoing mitigation efforts can be based on the best available science. This risk assessment is part of an overall mitigation plan for goods movement. Waiting years or decades for new scientific findings to emerge is not an option as there are clear health and economic impacts that need to be mitigated now.

5. There was also concern that while the input of the scientific community would be included in the public record it was not apparent how the concerns we would raise would be incorporated into a final document given the timing. Inclusion of comments into the record without a commitment to modify the final document to address concerns was a matter of concern.

CARB Staff Response: As with all scientific peer reviews conducted by CARB staff, we will acknowledge and respond to all comments received into the final document.

To conclude: we believe a more deliberate process should be initiated that has a more realistic timetable and will maximize the input of the scientific community. This could include at least a one day-long meeting between members of the scientific community and scientists from CARB and OEHHA to address the wide ranging questions and methodologic issues prior to developing a draft document. I know everyone is pressed for time on this issue and, again, we respect the current efforts underway, but we also think there are too many unresolved issues at this point to develop a comprehensive document for peer review. I am available for further discussion and the other scientists would welcome a conference call to address the concerns expressed here. Finally, we have communicated with you because we think these issues require attention at the highest levels of Cal/EPA and State Government.

CARB Staff Response: We provided all the details of our risk assessment into this draft risk assessment. This includes references to the underlying literature, the computer program code, detail inputs and results, and acknowledgment of all uncertainties, assumptions, caveats, and unquantified risks of which we are aware. We have given the peer reviewers two weeks to review this document and scheduled a workshop for public input. We are also available to meet with any individuals or groups who requests and can also provide programs or conduct further calculations (i.e., sensitivity tests) as requested.

Comments from Aaron Halberg (Abt Associates Inc.)

(1) You mention that given more time and resources, a modeling-based approach would be appropriate. Longer term, you might want to talk with Bryan Hubbell at EPA about the response-surface models he has been working on - with an initial investment of fairly significant time and resources, this approach can potentially lead to simulations of

air quality models which produce remarkably accurate results essentially instantaneously.

CARB Staff Response: We contacted Bryan Hubbell of USEPA as we are interested in any short- and long-term improvements to our methodology He informed us that, at this time, the response surface modeling is divided into two areas: 1) ozone modeling at 12-km grid resolution using CAMx in the Eastern U.S.; and 2) CMAQ modeling at 36-km grid resolution for the entire U.S., with outputs of PM2.5 and component species, deposition, visibility, and ozone. As a whole, the model performs very well in replicating CMAQ responsiveness to changes in precursor emissions. However, the model's predictions are good but not quite as good in California, but might be improved with additional runs they are conducting. We are also planning on conducting some focused 12km response surface modeling in some additional urban areas in the spring of 2006. When complete, these modeling results will be useful to compare to California-specific modeling being conducted for State Implementation Plans and the potential SECA request (see Section V-C)).

(2) In the exposure section, you mention that interpolation of NO_X and SO_X will be done to the census tract - this seems like it might be overkill to me, given that you are estimating the impacts of ambient exposures (people don't tend to spend all of their time in the tract in which they live, epi [epidemiology] studies tend to use county averages, etc.) and that other sources of data will be at much higher levels of spatial aggregation (e.g. regional estimates of background levels, county-level adjustment factors).

CARB Staff Response: We interpolated PM nitrates down to the census tract level to make sure of census populations in developing population-weighted exposures. However, the population-weighted exposures were developed at the county and the basin level, consistent with the higher levels of spatial aggregation used in the epidemiologic studies.

(3) In the exposure section, you mention getting uncertainty estimates using a Kriging analysis of interpolation uncertainty, while the interpolation approach used is a simple inverse-square weighting. How exactly do you plan to generate interpolation uncertainties? Do you plan to try to propogate this uncertainty through the health impact and economic benefit calculations?

CARB Staff Response: Both Kriging and simple inverse squared distance weighting schemes come with cross-validation errors that could be used as interpolation uncertainty. In this phase of the report, we have not incorporated this source of uncertainty (due to exposure estimation into our calculations).

(4) You should be careful to avoid double counting when generating your benefits estimates - in particular, when valuing premature mortality across both PM and Ozone (are you using single or multi-pollutant studies?), MRADs across both PM and Ozone (again, single or multi-pollutant studies?), Asthma Attacks (PM) and Respiratory Hospital Admissions (Ozone) (not sure if there is overlap there or not).

CARB Staff Response: The estimates associated with PM exposures were based on studies that consider PM with various other potential confounders, including ozone. Likewise, estimates associated with ozone exposures were based on studies that

consider ozone with various other potential confounders, including PM. Since the studies do not coincide, we minimized the potential chance of double-counting.

Comments from Dr. Jean Ospital (South Coast Air Quality Management District)

[Dr. Ospital was contacted by phone and told of CARB staff's plan to use the Pope et al. (2002) associations between PM2.5 and premature death rather than Krewski et al. (2001), and to use Jerrett et al. (2005) as a sensitivity test. He concurred. Dr. Ospital stated that the local community residents in the South Coast Air Basin would also be interested in the near-source diesel PM cancer risk (i.e., increased lifetime lung cancer risk per million exposed using OEHHA's upper 95th percentile unit risk factors).]

CARB Staff Response: We proceeded as recommended on the PM2.5 and premature death concentration-response functions. These premature death estimates include lung cancer deaths as discussed in Section II D. Separate diesel PM cancer risks using the OEHHA unit risk factors can only be calculated if we know the diesel PM concentration and the size of the affected population, which generally means a dispersion modeling study as there is no routine, reliable measurement method for diesel PM. The necessary modeling analyses have been conducted for diesel sources associated the Ports of Los Angeles and Long Beach, the Roseville Rail Yard, and air basin averages. These diesel cancer risks are presented in Section II C, and CARB is conducted similar modeling analyses for the Port of Oakland expected by next year and the 16 largest rail yards in California over the next three years.

Comments from Professor Michael Jerrett (University of Southern California)

[The following comments refer to the No Net Increase report risk assessment (see www.portoflosangeles.org/DOC/NNI_Final_Report.pdf, beginning on page 4-23)]

I had a quick glance at the document. My first comment would that the health benefits should be based on the attached paper [Jerrett,et al., Epidemiology, 16: 1-10, 2005] (which I led, but has had substantial input from Pope, Krewski and Burnett). This paper gives direct estimates for the LA region, while the Krewski 2000 report is based on a national study where the majority of the exposure contrast comes from sulfates in the Ohio River Valley. I did the spatial analysis and much of the statistical modeling for Krewski, so I have a detailed understanding of these exposure contrasts that may not come through without reading all 298 pages and all the appendices of Krewski. The final version of the attached paper is now in print on the website (www.epidem.com). The risk estimates here are about 2-3 times higher than reported in Krewski (and given that Krewski and Pope are co-authors, the methods used are either identical or better, based on our latest understanding of the statistical methods and likely confounding effects). I anticipate that further modeling will produce even larger health effects because we have an even better exposure surface, which is ready go and will be used in a follow up where we compare effects in LA to NYC.

Bottom line: this benefits assessment underestimates the benefits. The benefits are probably two to three times greater than stated in the report. I am confident that Krewski, Burnett, Pope and all the other ACS researchers would agree that the LA estimates are a better basis for benefits estimation in LA,. There are many other concerns I could voice about the report, including from what I can glean a vaguely

defined geographic scope. Another concern is that PM from diesel is likely to be more toxic that some of the secondary components, and none of the ACS studies (Krewski, Pope, Jerrett and others) has done an direct analysis of primary diesel. If we extend our toxicology findings, we might expect the primary diesel to elicit a higher concentration-response.

CARB Staff Response: For premature death due to diesel PM2.5, the study by Pope et al. (2002), updating the original mortality estimates of the original ACS cohort study for all-cause, cardiopulmonary, and lung cancer mortality, was used to derive the concentration-response function. For this study a 6% increased risk for all-cause mortality was identified for each 10-μg/m^3 difference in fine particle concentration (Pope et al. 2002).

A California-specific study of the same mortality endpoints in relation to ambient PM2.5 has recently been published. This study (Jerrett et al., 2005) employs many methodological advances and uses the latest techniques in spatial analysis with the intent of reducing exposure misclassification. Staff and peer reviewers felt it was premature to use these new estimates to calculate statewide mortality estimates. Several arguments are put forth by Jerrett et al. (2005) to explain the larger effect estimates found in this analysis. These include: underlying differences in the subcohort; differing rates of decline in ambient PM2.5 concentration from one metropolitan area to another (in the ACS study); greater traffic exposure; meteorological or topographic differences; and, larger exposure measurement error due to heterogeneous change in air pollution levels during follow-up. The authors provide well-developed arguments against any of these factors having a significant impact on the estimates. Given the number of potential areas for differences to occur, however, and the variability of all of these parameters in different regions throughout the state, it seems reasonable not to use these estimates before confirmatory studies can be performed in different metropolitan regions. The Jerrett et al. (2005) study does suggest that intra-urban exposure gradients may be associated with higher mortality estimates than previously supposed and 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). These new estimates, once confirmed, may be particularly relevant to areas experiencing higher exposures due to goods movement.

Comments from Professor Constantinos Sioutas (University of Southern California)

Emissions

[Methodology document – We have already developed goods movement emissions estimates for TOG, ROG, CO, NO$_X$, SO$_X$, PM, PM10, PM2.5, and diesel] are these data published? This is crucial information and there is not sufficient material in this report for the uninitiated reader, like myself, to figure out how this was done.

CARB Staff Response: The emissions estimates are a combination of published data and new estimates.

[Methodology document – Goods movement emissions are split into emissions associated with imports, exports, and other emissions] what are these other emissions? This is also important to mention; or is this "other" sources what is listed below.

CARB Staff Response: These details are provided in Section III-A of this appendix].

<u>Ocean-going Vessels</u>

Emission of PM and gaseous co-pollutants? What is exactly included in these emission profiles? Is it the same information that we have for example for trucks in dyno facilities?

CARB Staff Response: Yes, both PM and gaseous pollutants are included. The analysis is for ozone and the major components of PM2.5, so the only speciation data needed is to have the direct PM, nitrate, sulfate, and VOC emissions broken out.

<u>Trucks</u>

Not clear to me what exactly is T4-T7.

CARB Staff Response: These are the same VMT categories as in EMFAC. T4 and T5 correspond to light heavy duty trucks, T6, corresponds to medium heavy duty trucks, and T7 corresponds to heavy-heavy duty trucks.

<u>Trains</u>

[Methodology document – This means that some emissions from several rail yards will be excluded from the health analysis because their activity is domestically focused.] This is also not very clear to me. How can cargo train activities be unrelated to goods movement?

CARB Staff Response: For the purposes of the Goods Movement report, locomotive emissions are included if they are directly related to international (import or export) goods movement. Locomotive emissions associated with domestic goods movement are not included in this report.

<u>Exposure</u>

[Protocol document – For primary and secondary diesel PM, we will use the methodology already employed in the diesel ATCMs.] How can you tell what is the fraction of diesel PM emissions that are associated with goods movement by the county-level exposure estimate? This, to me, seems such an important key statement that some methodological description would be appropriate.

CARB Staff Response: County emissions estimates for goods movement soures are combined to create air basin estimates and then applied to the air-basin-level exposure estimates to generate air-basin-level impacts.

[Protocol document – We will also develop adjustment factors for diesel PM emissions from sources (offshore ships) that are not distributed uniformly throughout the urbanized areas…] Aren't most of these sources distributed non- uniformly? Our recent studies in Long Beach show that in just 4 sites, 2 of which are CARB-AQMD monitoring sites, the spatial distribution of species such as EC, metals, OC etc is not homogeneous, with coefficients of divergence (CODs) in the range of 0.5-0.7, and this in sites apart by just few miles! And I am referring to PM mass based species I would not even raise the

issue of the enormous spatial heterogeneity of ultrafine numbers So what assumptions are made here about which sources are uniformly distributed and which ones are not, and on what information this distinction is based?)

CARB Staff Response: Exposure due to sources at Port of Los Angeles and Long Beach are estimated using the ISCST dispersion model. Direct PM emissions from ships in other regions are estimated using the procedures described in Methodology Section E.

[Protocol document – …by using results from existing offshore tracer studies…] Have these studies been published? What are they using for off shore tracers? If V [vanadium], I have my quite serious concerns on its validity.

CARB Staff Response: The offshore traces are inert gases (i.e. sulfur hexafluoride, perfluorocarbons) that are released from the ships during special studies.

[Protocol document – …and the intake fraction approach from UC Berkeley.] For the intake fraction methodology to be used here one would have to know quite accurately within these communities the spatial variability of PM and co-pollutants of interest. If the exposure levels are based on 1-2 stationary samplers in say the entire Long Beach area, I do not see how the population density can be matched to the 1-2 data points of each community. And there is of course issue such as indoor penetration and physico-chemical modification of PM and co pollutants from these sources, all of which would greatly affect the IF model's ability to provide accurate data. Does the board plan on addressing some of these issues?)

CARB Staff Response: The intake fraction approach has been dropped since the concentration-response functions are based on community-average outdoor exposure.

[Protocol document – Since almost all of the nitrates are in the fine fraction…] This is a very incorrect statement. Our 5 year Supersite data and related publications showed that about 40 - 50% of nitrate is in fact in the coarse mode and it is not sodium, but ammonium nitrate! I would be happy to forward the related papers).

CARB Staff Response: As a conservative assumption, we assumed all the nitrate was in the form of PM2.5. In term of data availability with maximum spatial resolution for both routine monitoring network and special study PM network, this study was focused on the mean annual calculation of nitrate concentrations for 1998. We believe that mixing PM2.5 and PM10 nitrate data in this study is reasonable for annual averages because most nitrates occurs in the PM2.5 fraction. This close linkage between PM10 and PM2.5 nitrate is shown by the relationship between PM10-nitrate from SSI and PM2.5 nitrate from special monitoring network, we have estimated ratio of PM10 nitrate to PM2.5 nitrate using PTEP data at six monitoring sites in southern California. In general, the annual mean fine PM-nitrate fraction at these sites was about 0.8.

[Protocol document – We will need to estimate and subtract background sulfate (from biogenic sources and long-range transport) since this can be a significant fraction of the observations.] Here again the definition of "long range" needs clarification. Do you mean transport from Long Beach to Riverside or from off shore emissions inland?

CARB Staff Response: We mean intercontinental transport.

General Comment on EXPOSURE: How does the above relate to CARB's Long Beach/LA Port report? That report was based entirely on *primary* Diesel. I think that somewhere in this document, efforts should be made to clearly delineate the steps and processes that will be taken by CARB to estimate the total fraction of $PM_{2.5}$ that is a result of goods movement from all sources, primary and secondary. I think the question of "what would air quality be without goods movement" is very important and I am not sure it can be addressed by characterizing what appear to be 2 sole markers of pollution, i.e., $PM_{2.5}$ and Ozone.

CARB Staff Response: In the Long Beach/LA Port Report, a detailed modeling approach was taken for the small 20 mile x 20 mile domain. In this report, staff determined that the entire state of California could not be modeled. Instead, we relied on emission estimates to develop the fractions of total emissions that are due to goods movement and documented the steps used to develop health impacts associated with goods movement. In this way, we've addressed "what health impacts would be avoided without goods movement" rather than "what would air quality be without goods movement".

Comments from Professor Arthur Winer (University of California at Los Angeles)

I did read the document over the weekend and as far as the Exposure part I have only one major concern: Whether it's appropriate to use a county level resolution for secondary air pollutants in basins like the SoCAB or Bay Area when it comes to multiplying total exposure estimates by the fraction of precursor emissions for each county. I'm not sure this will work well for secondary air pollutants for all the obvious reasons. I assume staff has thought about this or I'm being confused by the ambiguous way the discussion treats county vs. air basin.

CARB Staff Response: Based on these comments, we did all the calculations at an air basin level.

I also felt relying on CARB, 1998 and the Cass and Schaeur studies, while perhaps the best you can do, is to rely on estimates and studies that are becoming dated.

CARB Staff Response: These are just used to check the original diesel PM model estimates, which has a similar base year (1990) as the Cass and Schauer studies.

Finally, in two places in this section census "tracks" should of course be census" tracts."

CARB Staff Response: This has been fixed.

[The following comments refer to the No Net Increase report risk assessment (see www.portoflosangeles.org/DOC/NNI_Final_Report.pdf, beginning on page 4-23)]

I find inconsistencies in the way the authors of this draft treat the uncertainties in both the emissions estimates and health outcomes estimates (current and future).

I find inconsistencies in the way the authors of this draft treat the uncertainties in both the emissions estimates and health outcomes estimates (current and future). If one understands the large uncertainties that underly modeled estimates of current and future PM and NO_X emissions in any given airshed, let alone over the entire state, then one also understands why the use of four, five and six significant figures with respect to emissions or emissions reductions estimates does not represent defensible science.

Thus, the use of a number like 598,965 tpy [tons per year] for the statewide NO_X inventory is ridiculous. Similarly, quoting PM and NO_X reductions to the nearest 1 ton in Table 1 is not defensible.

To be fair, in parts of the narrative the authors do treat emissions estimates more properly, e.g. in the first paragraph using 28,000 tpy and 25,000 tons for the statewide diesel emissions inventory and PM emissions reductions estimate, respectively. What the authors need to do is go through this analysis systematically and reduce the number of sig figs [significant figures] in all cases to two, or at most three, sig figs, as appropriate.

Note, this problem of not acknowledging the uncertainty in the emissions and emissions reductions estimates has direct implications for the health outcomes estimates. Namely these also are often given to an accuracy/precision not supported by the input data used in their calculation. Again, the report is inconsistent in the way it treats significant figures for the health outcomes, in some places using two sig figs, e.g. 41,000 asthma attacks (even this should be rounded to 40,000) but in other places, e.g. in Table 2, giving mortalities to the nearest tenth of a death. Anyone who thinks we know what the avoided premature deaths in 2025 will be to the nearest tenth of a death is seriously deluded.

Personally, I'm against ever quoting a single number for these kinds of health outcomes projected far into the future. What should be given is only a range representing the 95% confidence intervals. To their credit, the authors do in many cases give the range and often to one or two significant figures, so again the report does better in some places regarding this issue than in others. But I would emphasize that Mike's [Jerrett of USC] indication the estimated benefits in this draft are too low by factors of two or three (!) is more evidence for why these authors need to be much more conservative in the way they present the data for both emissions and health outcomes (current and especially future).

Finally, the constant misuse and abuse of significant figures by the risk assessment community, failing to acknowledge the generally large uncertainties in the emissions models, exposure estimates and health outcome data, is a big part of the reason I have considerable cynicism and mistrust about the risk assessment process itself. The way many of the data in this report are presented does nothing to ameliorate my concerns.

CARB Staff Response: We agree that all uncertainties need to be acknowledged, that ranges should be presented whenever we show an central estimate, and that significant figures need to be reduced to one or two (or if we want to include in intermediate calculations so others can reproduce the final results, we should at least acknowledge that they have no meaning). Where possible, we will provide quantitative estimates of uncertainty. However, only qualitative or semi-quantitative discussions are possible for the emission and exposure estimates. To combine uncertainties for the concentration-response functions and the economic valuations, we are using a first-series Taylor series expansion.

D. Proposed Methodology – November 10, 2005 Peer Review Draft

1. Summary

California Air Resources Board (CARB) staff have been tasked to develop an estimate of the health and economic impacts caused by international goods movement as part of the California Goods Movement Report due for a public release in early December. This document represents our current thinking on methodologies that could be used. We expect this document will continue to evolve as we receive comments from peer reviewers and as the analysis proceeds.

Given more time and resources, modeling approaches using CALPUFF and/or CMAQ to estimate particulate matter (PM) and ozone concentrations associated with goods movement would be appropriate. However, given the short time frame to generate health and economic impact estimates, modeling is not an option. Thus, our exposure and health risk methodology for diesel PM and particle nitrates (Lloyd and Cackette, 2001), modified to a region-by-region approach, with the addition of similar methodologies for particle sulfates and ozone, is proposed to achieve our internal deadline (November 21). All health endpoints used in the PM and ozone standard reports (CARB and OEHHA, 2002; 2005) will be included, and annual impacts for 2005, 20010, and 2020 will be presented. An economic valuation of the health impacts will be performed using the same methods employed for airborne toxic control measures (ATCMs) by CARB (2003abc; 2004abc).

To correct for potential inconsistencies between exposure and emissions where emissions are not distributed uniformly in urban areas, we will develop adjustment factors for diesel PM emissions sources located in the outer continental shelf. This correction is assumed not to be necessary for secondary pollutant precursors (VOC, NO_X, and SO_X)

Since the health and economic impacts estimates will have large uncertainties, we propose to provide 5th and 95th percentile confidence bounds based on an integrated analysis of uncertainties in exposure estimates, human health concentration-response relationships, and the economic values. While including uncertainty due to emissions is desirable in this case, a quantitative assessment is not available. However, we will provide a qualitative description of sources of uncertainties in emissions, and how those uncertainties will affect health and economic impact estimates.

2. Emissions

We have already developed goods movement emissions estimates for TOG, ROG, CO, NO_X, SO_X, PM, PM10, PM2.5, and diesel PM. The inventory provides emissions by county, air basin, and source categories that are associated with goods movement. Goods movement emissions are split into emissions associated with imports, exports, and other emissions. The inventory also contains the following categories.

a) Ocean-Going Vessels (OGV)

The inventory contains emissions for nine vessel types. Most transit emissions occur in the outer continental shelf, which is defined as >3 miles from shore. Passenger vessels are the only category not considered related to import and export goods movement.

Emissions are allocated to imports/exports by the fraction of tonnage associated with imports and exports at each port. If data for a port was not available, we assumed 75% imports and 25% exports. We will generate all OGV emissions by county and air basin, including the outer continental shelf. Emissions will be split into hotelling (auxiliary engines at ports) and maneuvering/transit (propulsion engines).

b) Commercial Harbor Craft (CHC)

Emissions are calculated for a variety of smaller vessel types. Fishing vessels and ferryboats are included in the inventory and are not assumed related to import or export goods movement. Other categories are associated with imports and exports, which were split using the same approach above. A portion of emissions by vessel type is assigned to the outer continental shelf by county. We will generate all CHC emissions by county and air basin, including the outer continental shelf.

c) Cargo Handling Equipment (CHE)

All cargo handling equipment emissions are assumed related to import and export goods movement and were assigned using the port splits above. We will include CHE emissions.

d) Trucks (TRK)

The goods movement inventory contains all T4-T7 trucks and associated emissions from EMFAC. We have estimated vehicle miles traveled (VMT) associated with primary, secondary local, and secondary long-haul truck trips throughout California by air basin. Emissions were estimated for imports and exports for each air basin using port-specific splits for trucks originating at each port. "Other" truck emissions include port-related truck trips that are not primary or secondary trips, as well as all domestic VMT. We will generate T4-T7 truck emissions associated with imports and exports only. These represent primary and secondary trips to and from the ports.

e) Trains (RAIL)

The goods movement inventory contains all train emissions. We have estimated the fraction of rail activity associated with imports and exports (international trade) by air basin, and then applied import/export splits for each port as above. Non-import or export emissions are considered domestic rail activity. We will generate locomotive emissions associated with imports and exports only. This means that some emissions from several rail yards will be excluded from the health analysis because their activity is domestically focused.

f) Transportation Refrigeration Units (TRU) and Dredgers (DREDG)

The inventory contains these sources by county. TRU emissions were first split between emissions occurring on trucks (95%) and trains (5%), and then assigned to imports/exports/other using import/export splits for trucks and trains by county. Dredgers were not associated with imports or exports. We will generate TRU emissions associated with import and exports only. These emissions will be added to truck and train emissions for the purposes of the health analysis. Dredgers will be included in the inventory, and added to the cargo handling equipment inventory for the health analysis.

Goods movement and Statewide emissions will be provided for the years 2000, 2001, 2005, 2010, 2015, 2020, and 2025, although the focus is on 2005, 2010, and 2020.

3. Exposure

For primary and secondary PM, we will use the methodology developed by CARB (Lloyd and Cackette 2001) and employed in the diesel ATCMs (CARB, 2003abc; 2004abc). One modification is that this methodology will be conducted on a region-by-region basis (county or air basin) for consistency with the benefit analyses in the PM and Ozone Standard Reports (CARB and OEHHA, 2002; 2005). For diesel PM, the air basin-specific population-weighted exposure estimates (for the appropriate year) from the Diesel Exhaust Toxic Air Contaminant (TAC) Identification Report (CARB, 1998) will be converted to a goods movement population-weighted exposure estimate by simply multiplying by the fraction of diesel PM emissions for the air basin that are associated with goods movement. We will estimate uncertainties by comparing the Diesel PM Identification Report estimates against advanced PM source apportionment studies conducted by Glen Cass and Jaime Schauer for the Children's Health Study and more recent results from the U.S. DOE-funded Gasoline/Diesel Split Study.

We will also develop adjustment factors for diesel PM emissions from sources (offshore ships) that are not distributed uniformly throughout the urbanized areas by using results from existing offshore tracer studies, CARB's recent modeling analysis for the Ports of Los Angles and Long Beach, and the intake fraction approach from UC Berkeley.

For particle nitrates, we have already developed a statewide exposure estimate using routine and special study (CADMP, CHS) PM10 and PM2.5 nitrate data, converted to ammonium nitrate. Since almost all of the nitrates are in the fine fraction, PM10 nitrate and PM2.5 nitrate measurements are treated as equivalent. Population-weighted county exposure estimates, related to all sources, will be calculated after interpolation of monitoring data to census tracts using inverse-square weighting with a 50-km limit. Similar to diesel PM, a goods movement population-weighted nitrate exposure estimate results by simply multiplying the total exposure estimates by the fraction of NO_X emissions for the county that are associated with goods movement. We will assume an adjustment factor for offshore emissions is not necessary since it takes several hours to convert NO_X to nitrate, although there is the potential for depositional loss over water. The results will be compared to our recent review of NO_X-to-nitrate observational and modeling studies. Uncertainty estimates will be based on a CARB-funded study of nitrate measurement uncertainties and a Krigging analysis of interpolation uncertainties.

For particle sulfates (which have not been addressed in previous analyses), we have already compiled routine and special study (CADMP, CHS, dichot XRF) PM10 and PM2.5 sulfate data, converted to ammonium sulfate. Since almost all of the sulfates are in the fine fraction, PM10 sulfate and PM2.5 sulfate measurements are treated as equivalent. Population-weighted county exposure estimates, related to all sources, will be calculated after interpolation of monitoring data to census tracts using inverse-square weighting with a 50-km limit. We will need to estimate and subtract background sulfate (from biogenic sources and long-range transport) since this can be a significant fraction of the observations. This will be done on a regional basis. Similar to diesel PM, a goods movement population-weighted sulfate exposure estimate results by simply

multiplying the total exposure estimates by the fraction of SO_X emissions for the county that are associated with goods movement. We will assume an adjustment factor for offshore emissions is not necessary since it takes several hours to convert SO_X to sulfate, although there is the potential for depositional loss over water. The estimates will be evaluated in the context of limited results currently available from the ongoing SO_X Emission Control Area (SECA) technical analysis. Sulfate measurements are relatively accurate, so uncertainty estimates will only be based on a Krigging analysis of interpolation uncertainties.

For ozone (which has not been addressed in previous analyses), we have already performed a detailed population-weighted hour-by-hour exposure assessment by county, considering background and threshold levels, as part of the Ozone Standard Report (CARB and OEHHA, 2005). One important finding from a trend analysis for the South Coast Air Basin was that ozone levels have fallen by the same proportion (above global background of 40 ppb) throughout the Basin. This implies that the combined ROG-NO_X control strategy is equally effective everywhere. Thus, we will apportion ozone-related health effects to goods movement by the fraction of ROG emissions (lower bound) and NO_X emissions (upper bound) for the county that are associated with goods movement. We will assume an adjustment factor for offshore emissions is not necessary since it takes several hours for ROG and NO_X oxidation to result in ozone accumulation.

4. Health

We will calculate total annual changes in the number of incidences of health endpoints (death and disease) associated with goods movement for base year 2005 and future years 2010 and 2020. This will be based on the peer-reviewed concentration-response relationships and base incidence rates in the health benefit analyses presented in the PM and Ozone Standard Reports (CARB and OEHHA, 2002; 2005). These estimates include 5th and 95th percentile confidence bounds. The health estimates will be calculated and presented on a statewide basis as well as by air basin and source category. The linearity of the concentration-response relationship will be demonstrated by showing ACS and Harvard Six-City results. The relative toxicity of PM components (diesel PM, nitrates, sulfates) have been investigates by Harvard (Laden et al., 2000) and in the Netherlands (Hoek et al., 2003), and these results will be summarized. Lung cancer impacts will not be considered separately as they are already included to some degree in PM premature death estimates (Pope et al., 2002). We will investigate this presumed overlap by converting OEHHA's unit risk factor for diesel PM to an odds ratio for comparison with the lung cancer findings for the American Cancer Society (ACS) cohort (Pope et al., 2002).

We will acknowledge other health issues in a more qualitative manner, including other health endpoints (e.g., asthma incidence, permanent lung function deficit), nanoparticles, PAHs/quinones, other TACs, and in-vehicle exposures (Fruin et al., 2002).

5. Economic Value

As with the ATCMs (CARB 2003abc; 2004abc), we will assign economic values to each health endpoint and apply discount rates for future years. Uncertainties in the economic

values will be noted and a range of discount rates (3% and 7%) will be used. The economic valuation will be conducted and presented on a statewide basis as well as by air basin and source category.

6. Uncertainty Analysis

We will also estimate the combined uncertainty from the individual uncertainties in the exposure, heath, and economic components of the impact assessment. Because quantitative uncertainty estimates in emissions are not available, a qualitative discussion will be provided. We will also provide a robust discussion of caveats and limitations to the quantitative approaches applied in the analysis.

7. Peer Review

We will share this proposed methodology with peer reviewers from academic institutions, the Office of Environmental Health Hazard Assessment, the California Department of Health Services, the South Coast Air Quality Management District, and the U.S. Environmental Protection Agency to allow advance notice of any concerns on their part. Reviewers will be selected for their specific expertise on the various components of the risk assessment. They will review the draft assessment before release to the general public.

8. Future Work

We will highlight ongoing and future efforts to improve the emission, exposure, health, and economic methodologies. These include ongoing studies of ship activity, air quality modeling for ports, the SECA measurement program and modeling analyses, research on the health impacts of nanoparticle and chronic ozone exposures, and valuation of cardiovascular disease.

9. References

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California Air Resources Board (CARB) Staff Report: Proposed Diesel Particulate Matter Control Measure For On-Road Heavy-duty Residential and Commercial Solid Waste Collection Vehicles, available at http://www.arb.ca.gov/regact/dieselswcv/isor3.pdf, 2003a.

California Air Resources Board (CARB) Staff Report: Proposed Airborne Toxic Control Measure For In-Use Diesel-Fueled Transport Refrigeration Units (TRU) and TRU Generator Sets, and Facilities Where TRUs Operate, available at http://www.arb.ca.gov/regact/trude03/isor.pdf, 2003b.

California Air Resources Board (CARB) Staff Report: Airborne Toxic Control Measure for Stationary Compression-Ignition Engines, available at http://www.arb.ca.gov/regact/statde/isor.pdf, 2003c.

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E. Public Comments and CARB Staff Responses

Comments from the public on the Port of Los Angeles and Long Beach No Net Increase benefits analysis (see www.portoflosangeles.org/DOC/NNI_Final_Report.pdf, beginning on page 4-23) and on the Draft Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach (see xx) are given verbatim (except as noted in brackets) below along with CARB staff responses (in italics). These two documents serve as starting points for the international goods movement risk assessment presented in this document.

CARB staff will respond to this comment.

Comments from Diane Bailey (Coalition for Clean Air) on the Draft Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach

[Addressed to Pingkaun Di of CARB – Co-signed by Tom Plenys (Coalition for Clean Air), Diane Bailey (Natural Resources Defense Council), Teri Shore (Bluewater Network), Bonnie Holmes (American Lung Association of California, Joel Ervice (Regional Asthma Management and Prevention Initiative), Andrea Samulon (Pacific Institute for Studies in Development, Environment, and Security, Don Anair (Union of Concerned Scientists), Jesse N. Marquez (Coaltion For A Safe Environment), and Noel Park (The San Pedro and Peninsula Homeowners Coalition)]

We are writing to you on behalf of Coalition for Clean Air, Natural Resources Defense Council, Bluewater Network, American Lung Association of California, the Regional Asthma Management and Prevention (RAMP) Initiative, Pacific Institute for Studies in Development, Environment, and Security, Union of Concerned Scientists, Coalition For A Safe Environment, and the San Pedro and Peninsula Homeowners Coalition, regarding the recently released Draft 'Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach'. We are pleased that the Air Resources Board (CARB) has dedicated staff resources to analyze the health risks associated with port operations. However, we have significant concerns with respect to the scope of the study and the methodology used. The increased demands on port operations and California's goods movement system require that a study of this nature fully characterize the scope of the public health impacts from our goods movement system. At this critical juncture, Californians rely on CARB to provide a clear assessment of the health impacts from our goods movement system as it stands today and how predicted expansion of the goods movement system will affect health impacts in the future. Without clear information about the potential air quality and health impacts, statewide efforts to establish a plan that will truly protect public health will continue to be hampered.

Our primary areas of concern are laid out in more detail below; technical concerns are attached separately.

A) The scope of this study significantly underestimates the health impact from port operations and the goods movement system in the South Coast Air Basin

Again, we are pleased that CARB staff has focused on the health impacts from port pollution and has attempted to isolate the impacts from the different sources of pollution operating within the port boundary. The study makes impressive headway in trying to understand the connection between the pollution generated by different types of equipment and its localized effects on neighboring communities.

Unfortunately, as we have expressed in verbal comments to date, the study fails to include in its analysis major hubs and transport corridors of the goods movement system. The goods movement system is a vast network comprised of freeways, rail lines, distribution centers, rail yards and ports. Each of these arteries and hubs serve as magnets for equipment running on diesel fuel. The emissions associated with these components of the goods movement system are undoubtedly significant and should be integral to any analysis of the port and its operations. The true risks from diesel pollution associated with the goods movement system can only be ascertained when the contribution from these sources are fully calculated and combined with the impacts from on-port sources.

CARB's ports health assessment omitted major goods movement arteries linked to the Los Angeles and Long Beach ports, including the 710, 60, 99 and I5 freeways, the Alameda Corridor, and connecting rail lines. In addition, as was reported in the Port of Los Angeles' Air Emissions Inventory for 2001, trucks and locomotives operating outside the port boundaries contributed over three times more diesel particulate matter emissions than the trucks and locomotives operating within the port boundary.[1] Notably, that emissions inventory only considered trucks out to their first dropoff point and did not fully consider emissions from operations at some of the region's largest rail yards.

The health impacts from the more than 40,000 diesel trucks frequenting the ports daily are significant. It is misleading to present estimates of the health impacts of port emissions on portside communities, without reporting impacts from trucks and railyards serving the port as these sources are operating within these communities. At the very least, the assessment should have noted that although emissions from offsite trucks and locomotives may be less than overall emissions from ships, the fact that these truck and train emissions occur within the communities means that health impacts will be higher per ton of pollution.

CARB should use available transportation studies such as the one by Meyer Mohaddes Associates, Inc. referenced in Appendix A, and the recently conducted cargo handling equipment inventories to assist in producing a more complete analysis. Similarly, operations at off-port railyards, such as the intermodal facilities in Commerce/East LA and Union Pacific's Intermodal Container Transfer Facility (ICTF), must also be included in the analysis.

Other regions in the state are affected by the goods movement system. Currently, the diesel particulate pollution associated with the Port of Oakland's operations account for roughly 10% of the area's diesel pollution. The acres of distribution centers in the Inland Counties serve as magnets for diesel trucks transporting goods to and from the ports. In

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[1] Starcrest Consulting Group, LLC, "Port of Los Angeles Baseline Air Emissions Inventory – 2001, Executive Summary", July 2005, p. 7

addition, the Central Valley is a major thoroughfare for goods traveling on the I5 and 99 freeways and is home to numerous distribution centers, rail yards and the Port of Stockton. The proliferation of distribution centers, the projected growth of the Port of Stockton and increasing interest to revive the Port of Sacramento point to the increasingly important role this region plays in our goods movement system. Ships coming down the ship channel past Ventura and Santa Barbara are also contributing significantly to air pollution in those areas. We strongly urge you to consider the impacts from these areas as well.

Based on the limited scope of this health impact study, nearly 2 million people in the Los Angeles region experience elevated cancer risk from sources of pollution located within the boundary of the ports. Without highlighting the major omissions described above, we believe it is somewhat misleading to compare cancer risk from port operations as calculated by this study with the 'ambient environment'. The text box on page 4 states that the 'expected rate of cancer for all causes, including smoking, is about 200,000 to 250,000 chances in a million.' To include smoking in this figure creates an inappropriate comparison to the non-voluntary decision to breathe. A more appropriate comparison of acceptable risk is the one in one million benchmark considered "significant" by U.S. EPA and under several environmental laws.[2] Furthermore, given that only a portion of the true impact from port operations has been captured by this study, readers may conclude that the associated risks of port operations are insignificant.

In summary, this study only begins to capture the true impacts of port-related operations and the goods movement system; this is just the tip of the iceberg. Californians are depending on CARB to provide a clear assessment of the health impacts from the state's goods movement system as it stands today and how it is predicted to expand in the future. Without clear information about the extent of the impacts on our air quality and public health, it will be impossible to establish a statewide goods movement action plan that will truly protect public health.

CARB Staff Response: The Diesel PM Exposure Assessment Study for the Port of Los Angeles and Long Beach investigated the impacts from the direct diesel PM emissions from the in-port and the over water ship activities. The health impacts related to off-port goods movement activities, such as on-road trucks and locomotives, the impacts from other goods movement centers, and the impacts from indirect PM have been addressed in the health analysis prepared for the Goods Movement Action Plan Phase II. .

B) The analysis must be framed in the context of growth

The year 2002 was used as the baseline of this study, although some estimates project that emissions have already increased by 60% since 2001 (recently reported in the *LA Times*). We must recognize that according to this study, the health impacts from operations in 2002 were already unacceptable. Given that operations and resulting

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[2] Section 112 of the Clean Air Act establishes a one in one million annual per-capita risk (1×10^6) as the threshold for acceptable risk; 42 U.S.C. Section 7412 (f)(2)(A). The Food Quality Protection Act also relies uses a one in one million threshold of significance [21 U.S.C. Section 346a(b)(2)(A)(ii)], as does the Food and Drug Administration [50 Fed. Reg. 51,551, 51,557 (1985) (codified at 20 C.F.R. 700)].

emissions and health impacts have already significantly increased since 2002, it is important for CARB to assess the full extent of the associated increased health impacts.

As this study highlights, LA-area port emissions are expected to increase an additional 60% in the next 15 years as cargo volumes triple. We believe CARB must produce a follow up to this study to discuss how such growth in emissions will impact public health. The port of LA's No Net Increase (NNI) effort included such analyses, which CARB staff defended vigorously. We believe these types of estimates are doable and are critical at this time.

Although there is a promise of an CARB "health effects review" by December 1, 2005, we are extremely concerned about the prospect of finalizing the BTH/CalEPA Goods Movement process without a clear indication of when the health impacts of the goods movement system will be quantified on a statewide basis. If the Administration is to meet its commitment to reduce California air emissions to 2001 levels by 2010, it must fully understand the public health ramifications of tripling trade through our state and *whether* the public health and environmental impacts of that expansion can, in fact, be mitigated to meet the Governor's goals.

CARB Staff Response: The commentor is correct is stating that the Diesel PM Exposure Assessment Study for the Port of Los Angeles and Long Beach used the year 2002 as the baseline. The total health impacts due to the change in projected emissions in future years, including 2005, 2010, 2015, 2020, have been estimated in the health analysis prepared for the Goods Movement Action Plan Phase II. .

C) The analysis must be framed in the context of similar health studies previously conducted

It is not clear in the draft study how and why the study methodology differs from past methodologies used to characterize impacts from air pollution. It appears there has been a departure from CARB methodologies used in the past, including those used during the NNI process and in the Roseville rail yard study. For example, in the NNI report for the Port of Los Angeles, CARB strongly defended a different methodology for calculating premature death statistics than was used in this study. CARB described the different methodologies used in the NNI report as "similar to those used in developing health [impacts] for State Implementation Plans."[3] As CARB explained, use of a different methodology "would represent a departure…that would need additional peer review."[4] This study also differs significantly from the South Coast Air Quality Management District's MATES II study characterizing health risks from air toxics in the South Coast Air Basin (SCAB). As raised during the October 26 public hearing, CARB should explain early in the study report how these methods have evolved.

We are concerned that risk estimates from on-port sources alone are inappropriately conservative. For example, CARB's recent Roseville rail yard risk study estimated cancer risks for the rail yard of more than 500 in one million, which is similar to the risks the port study assigns to the combined emissions of the Los Angeles and Long Beach

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[3] NNI Report at 423

[4] NNI Report at 427

ports. The similarity in risk between a single rail yard and the third largest port complex in the world is highly questionable and deserves further explanation. We are also troubled by and would like further explanation of CARB's assertion that aerial deposition into the ocean accounts for the fact that both ports pose little more risk than the Roseville rail yard.

Further, this study asserts that the ports are responsible for 21% of the diesel pollution in the SCAB and 29 premature deaths per year. This does not comport with the CARB statistic of an estimated 2,900 premature deaths from diesel pollution statewide in 2000. One would expect an order of magnitude higher level of premature deaths caused by such a major contributor to basinwide and statewide diesel pollution. Nor does the 29 premature deaths estimate in this study from *both* the Port of Long Beach and Port of LA comport with CARB's estimate in the NNI report of 2,200 premature deaths over the next 20 years from pollution at the Port of LA *alone*. We find this severe decrease in CARB's premature death estimates from just 5 months ago particularly troubling in light of CARB's statements in the NNI report that (1) they "stand behind their [2,200 premature death] assessment [from the Port of LA alone] and recommended it to the Task Force for inclusion in the final report"[5] and (2) "it is likely that we have *underestimated* the health benefits [of NNI] in this analysis"[6] In future revisions or follow-ups to this study, we urge CARB to provide more explanation as to how these estimates compare to similar health assessments and to justify any departures from previously accepted and long used methodologies.

CARB Staff Response: Generally speaking, it is difficult to directly compare the results from the Diesel PM Exposure Assessment Study for the Ports of Los Angles and Long Beach (Ports Study) with the Roseville Rail Yard study (Rail Yard study). For the Rail Yard study, the emissions were distributed over a relativly small area. The nearest receptors in the nearby community from the busiest activity area in the rail yard were within 200 feet. For the Port study, about half of the emissions are distributed over a wide area over the ocean, about 5 to 50 miles from the coastline. The port property also occupies an area of about 6 miles x 4 miles in which most of the emissions have been diluted before reaching the nearby communities. In addition, the wind patterns are different around the Roseville Rail Yard as compared to those near the Ports of Los Angeles and Long Beach. For the Roseville Rail Yard, the predominate wind directions were toward to the northwest direction which is the most populated residential areas, while for the Ports, the predominate wind directions are toward to the southeast which is over the ocean. The risk levels reported in our study are those in the nearby communities. The higher risk levels displayed in the port property and the nearby ocean are excluded from our analysis.

With respect to a comparison to health values estimated for the Port of Los Angeles's No Net Increase (NNI) project, the two studies are based on the same methodology; however, they evaluate different scenarios and rely on are different approaches for estimating the concentrations of emissions. For the NNI project, the CARB was asked to

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[5] NNI Report at 423.

[6] NNI Report at 428 (emphasis added).

estimate the cumulative health benefits that would result from implementation of the emission reduction measures identified through the NNI process. This analysis took into consideration all the emissions from the port – both on-port emissions and regional emissions. As such, CARB staff relied on a mass based approach, similar to the approach used in the Goods Movement Phase II health analysis and estimated the health benefits accrued over a 20 year period from predicted reductions in diesel PM, NOx and ozone. The Diesel PM Exposure Assessment study on the other hand, was designed to look at a "snap shot" in time, in this case 2002, and estimate the health impacts only from dispersion modeled concentrations of directly emitted diesel PM. Because of these differences, the results are very different. The NNI numbers are much higher because they are looking at potential reductions that occur over multiple years and are based on the total mass of emissions which do not take into consideration the dispersion of emissions over water. The NNI analysis also includes the benefits from reductions in NOx and ozone. The Port Study, on the other hand, results in much smaller health values as it only represents the impacts from the emissions in 2002, only considers on-port and overwater directly emitted diesel PM and, because of the dispersion modeling, more accurately reflects the actual exposures that results from emissions from port activities in the area near the ports.

D) Additional deficiencies must be addressed in future revisions of this analysis

CARB should take particular care to address the following deficiencies in all future revisions or updates to health assessments related to goods movement.

All adverse public health outcomes must be considered

The study as drafted did not consider a full array of illnesses that have been linked to diesel PM exposure. Other health impacts known to be strongly associated with diesel particulate pollution should have been included in the analysis, such as: chronic bronchitis, chronic obstructive pulmonary disease, cardiovascular illness, asthma emergency visits and hospital admissions, and other respiratory- or cardiovascular disease-related hospital admissions. There is an increasing body of evidence that corresponds to these illnesses. CARB should evaluate *all* potential health impacts from pollution caused by the goods movement system.

Furthermore, as mentioned in this assessment, there is a growing body of scientific evidence, which directly relates goods movement pollution to public health impacts, especially from ultra fine particles. We urge you to include such critical findings in this analysis and any future analyses of the health impacts from port-related pollution. We also encourage you to work closely with other scientific experts focused on the implications from air pollution as it relates to the goods movement system. These scientists are investigating mobile source pollution and the impacts of particulate matter and other traffic-related pollutant exposure on the health of children and other vulnerable populations, and their expertise is critical to the deliberations. Findings from recent studies, such as the Children's Health Study, should be included. Given the inherent uncertainties in risk assessments, full consideration of recent results from health effects research seems appropriate. Specifically, this study (unlike the MATES II and other risk studies) provides only the *average* risk to which the affected public is exposed. CARB should let the public know the greatest risks to which members of the

public are being exposed. In addition, the literature on proximity to traffic and respiratory and cardiovascular health effects must also be assessed.

CARB staff will respond to this comment.

All pollutants emitted from goods movement related activities should be considered

This assessment should state early and more clearly that it is a source apportionment analysis for on-port diesel sources and does not in any way represent cumulative exposures or risks. Although exposure to diesel particulate pollution is known to be a major contributor to adverse health impacts, it is also known that many other sources are significant. Future analysis should include other pollutants, such as NOx and air toxics such as benzene, formaldehyde, acrolein and 1,3-butadiene at a minimum. Other port sources, such as coke piles, tank farms, petroleum terminals and fumigants at a minimum should also be included in future analyses.

CARB Staff Response: This analysis is not limited only to on-port diesel sources. Chapter 2 describes the sources and categories included in the goods movement category for this plan. The comment above is correct that emissions of toxics other than diesel PM as well as stationary sources such as coke piles, tank farms, evaporative emissions from petroleum terminals, use of fumigants, and other sources at the ports are currently excluded from the plan. However, these sources do not emit diesel PM, and generally contribute a very small fraction of the overall port emissions

Cumulative risk, sensitive populations, and exposed workers must be considered

Not only should future assessments address cumulative risks from all pollutants and sources impacting the area of study, these assessments should include hot spot analysis and focus on maximally exposed individuals. Risk assessment methodologies must be improved to better reflect the heightened vulnerability of children and sensitive populations, or at the very least acknowledge these deficiencies as factors leading to underestimated health risk.

It is not clear why this report does not characterize the risk on the terminals themselves. The maps indicate that risks where workers operate exceed 1,500 in a million. There also appear to be high-risk areas in the vicinity of the existing ICTF. These risks should be highlighted and ideally blown up in more detail. All impacts greater than 500 in a million should be discussed in much greater detail.

CARB Staff Response: We will respond to this comment at a later time.

Modeled parameters should reflect the realities of the goods movement system

CARB must ensure that modeling assumptions are "ground-truthed" to reflect actual conditions at goods movement facilities as opposed to statewide averages. For instance, while some may assume that truck fleets turn over every 12 years, it is common knowledge that truck fleets serving the ports and other goods movement facilities turn over much more slowly. In particular, it is well documented that some of the oldest trucks on the road service these ports and a large number of such trucks are over 20 years old. If these realities are not properly accounted for in the modeling, predictions of health impacts will be severely underestimated. Any future analyses that

cumulatively account for impacts of the larger goods movement system (such as freeways and distribution centers) should include assumptions that are appropriate for the goods movement model.

CARB Staff Response: We will respond to this comment at a later time.

Future assessments must include a public input process

Finally, it appears that the process behind publication of this assessment excluded public participation and input from interested stakeholders. Future endeavors such as this should include opportunities for public input and participation during the initial study design and execution.

CARB Staff Response: A public process was undertaken to encourage comment on the draft report. CARB staff released the draft Diesel PM Exposures Assessment Study for the Ports of Los Angeles and Long Beach on October 3, 2005 and requested comment. In addition, two public meetings were held on October 26, 2006 in San Pedro, CA to present the draft findings and obtain public input.

Conclusions

We appreciate CARB's significant effort to assess the health risks associated with port operations through this study and urge CARB to continue to focus attention on the serious health implications of the goods movement system in California. We are also encouraged by CARB's general commitment to issue a more comprehensive health assessment in early December. In light of this commitment and with respect to all subsequent health assessments, we strongly urge CARB to address the concerns outlined in this letter. Specifically, future health assessments should cover all adverse public health outcomes, a wider array of pollutants known to cause adverse health impacts, and all significant sources known to emit these pollutants within the context of the assessment. Other issues that must be discussed and fully incorporated into future analyses include cumulative risk, increased vulnerability of sensitive populations, and risks to exposed workers (in addition to residential populations). Modeled parameters should also reflect the realities of the goods movement system. Finally, future assessments must include a public input process, and we suggest that health assessment and modeling experts be convened to form an advisory group to the process.

As currently drafted, the results of this assessment emphasize the need for the strongest possible port related regulations scheduled for consideration at the December Board Hearing. Clearly cargo handling equipment and the auxiliary engines on ocean going vessels are two critical sources of pollution which must be addressed. In the context of California's goods movement system, we believe that a more comprehensive health assessment together with estimates of health impacts from predicted expansions of the goods movement system will continue to underscore the need for more health protective control strategies.

CARB Staff Response: We agree that a comprehensive assessment of the health impacts associated with California's goods movement system is necessary. Appendix A "Quantification of the Health and Economic Impacts of Air Pollution from Port-related Goods Movement and Port Activities in California" included in the Goods Movement

Action Plan Phase II document provides a comprehensive analysis of the potential health effects associated with exposure to air pollutants arising from port-related goods movement.

Technical Concerns Over Assessment Methodology

The draft assessment document does not contain sufficient detail to fully convey the assumptions and methodologies used. Upon review, however, we have several technical concerns outlined below. We suggest that CARB form a working group of experts to improve the methodology.

CARB Staff Response: We will respond to this comment at a later time.

Port of LA emission inventory progression from 2001 to 2002

CARB incorrectly assumes linear growth of cargo handling equipment (CHE) emissions between 2001 and 2005 in order to project the CHE emissions from 2001 to 2002. In fact, historical data indicates that the growth in trade has not been linear from 2001 to 2005.[7] Annual Twenty-foot Equivalent Units (TEUs) handled by the Port of LA jumped from roughly 5.2 million in 2001 to 6.1 million in 2002, while TEUs in 2005 can be estimated as roughly 7.5 million. CARB should have used a more accurate benchmark for CHE emissions, such as historical TEU growth. CHE emissions are likely underestimated as a result of the linear growth pattern assumed. The use of linear interpolation to determine 2002 emissions for harborcraft, trucks, and locomotives is also inappropriate for the reasons specified above. Growth in the truck and locomotive emission inventory should have been related to growth in container traffic as with CHE. Growth in harbor craft emissions should have been more carefully determined based on growth in Ocean Going Vessel (OGV) visits weighted by the average harbor craft use for each major OGV category. The surge in trade between 2001 and 2002 and resulting increased emissions are not adequately captured by the linear interpolation method used for this analysis. Finally, the strangely varying growth rates of 0.0, negative 6.0 and 11.0 for harbor craft, trucks and locomotives respectively warrant much further explanation. It is difficult to understand how emissions from trucks could have decreased between 2001 and 2002, given the significant increase in container throughput.

CARB Staff Response: The current inventory presented in this document is draft, and CARB staff are developing potential improvements to each of the categories above. The year 2002 was interpolated for convenience in this version of the report; 2002 is not a focus of the planning process in the Goods Movement Plan.

Modeling Methodology

The 200 meter by 200 meter receptor grid used in the modeling appears to be an order of magnitude more coarse than the 25 meter receptor spacing used in the MATES II microscale modeling. This may have led to a substantial underestimation of risks, especially given that some of the highest risk isopleths drop off within a distance less than or close to the 200-meter receptor spacing. The steep isopleths of the highest risk

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[7] http://www.portoflosangeles.org/factsfigures_Annual.htm

levels are difficult to read on the maps provided in the report; we suggest more detailed, finer scale maps for high risk areas in future assessments. We also recommend the use of a much finer receptor grid for future assessments covering similarly small geographic areas. Finally, it is specifically stated that this assessment was not designed to identify hot spots, however hotspot identification would be more useful than simply reporting averages.

The assessment notes that a sensitivity study was done to evaluate King Harbor meteorological monitoring data versus that from Wilmington. It would have been useful for similar sensitivity analyses to be done comparing North Long Beach site data and meteorological data sets from different years, as meteorology can change significantly from year to year.

CARB Staff Response: We will respond to this comment at a later time.

Comments from Andrea Hricko (University of Southern California)

Thanks for asking if I had any relevant articles to send you for review by CARB staff members as they work on the health document for the Goods Movement Action Plan. I have just put together for you a huge file of {mostly .pdf} scientific articles by category (cancer, respiratory, cardiovascular, traffic proximity, noise, PM/ultrafines, etc).

By the way, I certainly don't make any claims to having a "complete" reference file but there may be some articles on the CD that staff members would like to have handy as they rush through this process. I do not have very many articles from the PM Center at UCLA, but I have two review articles by Costas Sioutas and Ralph Delfino on ultrafines (recently published) that may be useful.

CARB Staff Response: These resources are greatly appreciated. Many of these references were used for discussion of specific health endpoints where clarification of the effects was needed. However, for much of this brief overview we relied on review articles such as Brook et al. (2004), in order to summarize new evidence. There was no need to do a complete update on PM or ozone health effects as these are covered in CARB's recent ambient air quality standard review, but the major endpoints and findings were covered in this document.

Comments related to health effect calculations from the public on the Port of Los Angeles and Long Beach No Net Increase benefits analysis (see www.portoflosangeles.org/DOC/NNI_Final_Report.pdf, beginning on page 4-63)

P. 4-47 (c.) No documentation is provided validating the assumption that the relationship between premature mortality attributable to changes in PM2.5 exposure is in fact linear, log-linear, or any other type of function over the range of anticipated change in PM2.5 levels attributable to the emission reductions identified in the HBA.

Furthermore, no documentation is provided that this assumption is applicable solely to changes in ambient DPM levels.

CARB Staff Response: An updated version of the premature mortality study was used for this health impact analysis and the log-linear approach to calculating diesel PM2.5 mortality is consistent with the exposure response relationships observed in this study by Pope et al. (2002). The log relative risks for all-cause, cardiopulmonary, and lung

cancer mortality increased across the exposure gradient for fine PM, and goodness-of-fit tests found that the associations were not significantly different from linear associations. Furthermore, this relationship did not appear to have a discernible lower "safe" threshold. This linear relationship between excess mortality risk and fine and coarse PM is demonstrated in the relative risks associated with all-cause mortality and long-term PM exposure in the Harvard Six-Cities study (Dockery et al. 1993) (see Figure A-2). Additionally, a number of recent studies have demonstrated the linear association between particulate matter pollution and cardiovascular disease. Chen et al. (2005) found a concentration-response function for fatal coronary heart disease in California women participants of the Adventist Health Study on the Health Effects of Smog (AHSMOG).

There is, at this point in time, no way to directly measure ambient diesel particulate matter (DPM) and therefore, no premature mortality studies are available for this specific pollutant. This health impact analysis must rely on the closest approximation of diesel PM based on emissions inventory estimates. However, diesel engines emit higher levels of particulate matter and while diesel PM is not directly measured in any of the mortality studies available for calculating a concentration-response function, an examination of various sources of particulate matter was examined in relationship to the mortality estimates made in an early study by Dockery et al. (1993); known as the Six-Cities study. This "speciation study" by Laden et al. (2000) used the elemental composition of size-fractionated particles to identify several distinct source-related fractions: mobile source, coal-combustion, and soil and crustal matter. These fractions were then associated with the daily mortality estimates to produce a meta-analysis of the overall relative risks for each source fraction. Mobile sources were associated with the strongest increase in daily mortality with an increase of 3.4% observed for each 10 $\mu g/m^3$ increase in the mean of the mobile source factor. Furthermore, the increased mortality was found to be associated with ischemic heart disease; there was a 2% increase in daily mortality from ischemic heart disease found in association with each 10 $\mu g/m^3$ increase in the mean of the mobile source factor. These estimates are very close to the estimated 6% increase in mortality from Pope et al. (2002).

There is every indication that DPM is in fact more toxic than other forms of particulate matter. A brief explanation of this toxicity is provided in the Air Pollutants of Concern section of this document. Given this enhanced toxicity and the fact that diesel engines are also responsible for most of the "mobile source fraction" of PM, it seems reasonable to use the approximation of PM2.5 mortality for this analysis. Furthermore, the Jerrett et al. (2005) study found higher excess relative risks for PM2.5 in the Los Angeles area, and the use of the Pope et al. (2002) study may likely be an underestimation, rather than an overestimation of premature mortality.

P. 4-53 (2.) Health effects and linear dose-response relationships from exposure to low levels of diesel exhaust have not been established.

CARB Staff Response: The premature mortality study used to estimate the coefficient for the concentration-response function in this analysis was Pope et al. 2002. Analysis of the relative risks in relation to ambient concentration estimates did not indicate there was a threshold or "safe exposure" level.