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

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

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

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

2. Ozone

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

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

3. Port-Specific Modeling

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

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

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

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

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

E. Economic Valuation of Health Effects

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

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

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

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

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

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

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

Table A-7 Collected Valuations of Premature Deaths Prevented

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

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

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

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

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

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

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

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

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

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

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

F. Uncertainty Calculations

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

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

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

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