

Appendix F
Non-Cancer Health Risk

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I. Health Impacts Assessment

A substantial number of epidemiologic studies have found a strong association between exposure to ambient PM_{2.5} and a number of adverse health effects (ARB, 2002). For this report, ARB staff quantified seven non-cancer health impacts associated with the change in exposures to the diesel PM emissions.

Staff estimates that approximately 90 premature deaths (30 – 160, 95 percent confidence interval (95% CI)) are associated with the baseline uncontrolled emissions from commercial harbor craft in the year 2004. Other health impacts are listed in the Table 1. The methodology for estimating these health impacts is outlined below. Details can be found in Appendix A of the Emission Reduction Plan for Ports and Goods Movement in California (ARB, 2006).

Table 1: Baseline Health Effects Associated With Commercial Harbor Craft Emissions for the year 2004*

Endpoint	Pollutant	# of Cases 95% CI (Low)	# of Cases (Mean)	# of Cases 95% CI (High)
Premature Death	PM	10	50	80
	NOx	10	50	80
	<i>Total</i>	<i>30</i>	<i>90</i>	<i>160</i>
Hospital admissions (Respiratory)	PM	10	10	10
	NOx	10	10	10
	<i>Total</i>	<i>10</i>	<i>20</i>	<i>30</i>
Hospital admissions (Cardiovascular)	PM	10	20	30
	NOx	10	20	30
	<i>Total</i>	<i>20</i>	<i>40</i>	<i>60</i>
Asthma & Lower Respiratory Symptoms	PM	450	1,200	1,900
	NOx	490	1,200	2,000
	<i>Total</i>	<i>940</i>	<i>2,400</i>	<i>3,900</i>
Acute Bronchitis	PM	0	100	210
	NOx	0	100	220
	<i>Total</i>	<i>0</i>	<i>200</i>	<i>430</i>
Work Loss Days	PM	6,600	7,800	9,000
	NOx	6,600	7,800	9,000
	<i>Total</i>	<i>13,000</i>	<i>16,000</i>	<i>18,000</i>
Minor Restricted Activity Days	PM	37,000	45,000	54,000
	NOx	37,000	45,000	53,000
	<i>Total</i>	<i>74,000</i>	<i>90,000</i>	<i>110,000</i>

* Health effects from primary and secondary PM are labeled PM and NOx, respectively. The sum of PM and NOx impacts may not equal the total given due to rounding.

Since diesel PM is a constituent of ambient PM2.5, using the epidemiologic study results to quantify diesel PM health effects is reasonable. This analysis shows that the statewide cumulative impacts of the emissions reduced through this regulation from year 2009 through 2025 are approximately:

- 310 premature deaths (90 – 530, 95% CI)
- 70 hospital admissions due to respiratory causes (40 – 90, 95% CI)
- 120 hospital admissions due to cardiovascular causes (80 – 190, 95% CI)
- 8,100 cases of asthma-related and other lower respiratory symptoms (3,100 – 13,000, 95% CI)
- 670 cases of acute bronchitis (0 – 1,500, 95% CI)
- 53,000 work loss days (45,000 to 61,000, 95% CI)
- 300,000 minor restricted activity days (250,000 to 360,000, 95% CI)

Table 2 lists the impacts associated with primary and secondary diesel emissions separately. Details can be found in Appendix A of the Emission Reduction Plan for Ports and Goods Movement in California (ARB, 2006).

Table 2: Total Health Benefits Associated with Reductions in Emissions from Commercial Harbor Craft (2009-2025)*

Endpoint	Pollutant	# of Cases 95% CI (Low)	# of Cases (Mean)	# of Cases 95% CI (High)
Premature Death	PM	50	200	340
	NOx	30	110	190
	<i>Total</i>	<i>90</i>	<i>310</i>	<i>530</i>
Hospital admissions (Respiratory)	PM	30	40	60
	NOx	20	20	30
	<i>Total</i>	<i>40</i>	<i>70</i>	<i>90</i>
Hospital admissions (Cardiovascular)	PM	50	80	120
	NOx	30	40	70
	<i>Total</i>	<i>80</i>	<i>120</i>	<i>190</i>
Asthma & Lower Respiratory Symptoms	PM	1,900	5,000	8,100
	NOx	1,200	3,100	4,900
	<i>Total</i>	<i>3,100</i>	<i>8,100</i>	<i>13,000</i>
Acute Bronchitis	PM	0	420	920
	NOx	0	250	540
	<i>Total</i>	<i>0</i>	<i>670</i>	<i>1,500</i>
Work Loss Days	PM	28,000	34,000	39,000
	NOx	16,000	19,000	22,000
	<i>Total</i>	<i>45,000</i>	<i>53,000</i>	<i>61,000</i>
Minor Restricted Activity Days	PM	160,000	190,000	230,000
	NOx	91,000	110,000	130,000
	<i>Total</i>	<i>250,000</i>	<i>300,000</i>	<i>360,000</i>

* Health effects from primary and secondary PM are labeled PM and NOx, respectively. The sum of PM and NOx impacts may not equal the total given due to rounding.

1. Primary Diesel PM

Lloyd and Cackette (2001) estimated that, based on the study by Krewski and colleagues (2000) of the American Cancer Society (ACS) cohort, a statewide population-weighted average diesel PM_{2.5} exposure in year 2000 of 1.8 µg/m³ can be associated with a mean estimate of 1,985 premature deaths per year in California (Lloyd and Cackette, 2001). In 2002, Pope and colleagues published new findings with the same ACS cohort based on a longer follow-up time and improved statistical modeling techniques. Consistent with U.S. EPA (2004), ARB has been using the new PM-premature death relationship from Pope et al. (2002) since the approval of the Ports and Goods Movement Emission Reduction Plan (ARB, 2006). Using the study by Pope et al. (2002), a statewide population-weighted average diesel PM_{2.5} exposure of 1.8 µg/m³ can be associated with a mean estimate of 2,200 premature deaths per year in California, about 10% higher than previous estimates. The diesel PM_{2.5} emissions corresponding to the diesel PM_{2.5} concentration of 1.8 µg/m³ is 36,000 tons for the year 2000 based on the emission inventory developed for this rule. Using this information, we estimate that for every reduction of 17 tons per year of diesel PM_{2.5} emissions, one fewer premature death would result. This factor is derived by dividing 36,000 tons of diesel PM by 2,168 deaths (unrounded number of deaths described above). Although a single statewide factor (tons per death) is discussed in this example, staff actually developed basin-specific factors for the health impacts assessment of emissions from commercial harbor craft. These basin-specific factors were developed using basin-specific diesel PM concentrations and emissions for the year 2000. After adjusting for population changes between each future year and 2000, staff estimates that the cumulative total of 2,020 tons of emissions from commercial harbor craft reduced through the implementation of this regulation in years 2009-2025 are associated with a reduction of approximately 200 deaths (50 – 340, 95% CI). Estimates of other health benefits, such as hospitalizations and asthma symptoms, were calculated using basin-specific factors developed from other health studies. Details on the methodology used to calculate these estimates, including the adjustment for off-shore PM emissions in the 3 – 24 nautical mile domain, can be found in Appendix A of the Emission Reduction Plan for Ports and Goods Movement in California (ARB, 2006).

2. Secondary Diesel PM

In addition to directly emitted PM, diesel exhaust contains NO_x, which is a precursor to nitrates, a secondary diesel-related PM formed in the atmosphere. Lloyd and Cackette (2001) estimated that secondary diesel PM_{2.5} exposures from NO_x emissions can lead to additional health impacts beyond those associated with directly emitted diesel PM_{2.5}. To quantify such impacts, staff developed population-weighted nitrate concentrations for each air basin using data not only from the statewide routine monitoring network, which was used in Lloyd and Cackette (2001), but also from special monitoring programs such as

IMPROVE and Children's Health Study (CHS) in year 1998. The IMPROVE network provided additional information in the rural areas, while the CHS added more data to southern California. Staff calculated the health impacts resulting from exposure to these concentrations of PM and then associated the impacts with the basin-specific NOx emissions to develop basin-specific factors (tons per case of health impact). Using an approach similar to that used for primary diesel PM and adjusting for population changes between each future year and 1998 (the year with the greatest geographic extent of nitrate monitoring), staff estimates that the cumulative reduction of 36,400 tons of emissions from commercial harbor craft in 2009-2025 are associated with the reduction of an estimated 110 premature deaths (30 – 190, 95% CI). Other health effects were also estimated as outlined above.

Assumptions and Limitations of Health Impacts Assessment

Several assumptions were used in quantifying the health effects of PM exposure. They include the selection and applicability of the concentration-response functions, exposure assessment, and baseline incidence rates. These are briefly described below.

- For premature death, calculations were based on the concentration-response function of Pope et al. (2002). The ARB staff assumed that the concentration-response function for premature death in California is comparable to that developed by Pope and colleagues. This is supported by other studies (Dominici et al. 2005, Franklin et al. 2007) in California showing an association between PM_{2.5} exposure and premature death similar to that reported by Pope et al. (2002). In addition, the Pope et al. (2002) study included subjects in several metropolitan areas of California. The U.S. EPA has been using the Pope et al. (2002) study for its regulatory impact analyses since 2004. For other health endpoints, the selection of the concentration-response functions was based on the most recent and relevant scientific literature. Details are in the Emission Reduction Plan for Ports and Goods Movement in California (ARB, 2006).
- The ARB staff assumed the model-predicted diesel PM exposure estimates published in the report titled "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant" (ARB, 1998) could be applied to the entire population within each basin. That is, the entire population within the basin was assumed to be exposed uniformly to modeled concentration, an assumption typical of this type of assessment.
- The ARB staff assumed the baseline incidence rate for each health endpoint was uniform across each county, and in many cases across each basin. This assumption is consistent with methods used by the U.S. EPA for its regulatory impact assessment, and the incidence rates match those used by U.S. EPA.

- Although the analysis illustrates that reduction in diesel PM 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. Health effects such as myocardial infarction (heart attack), chronic bronchitis, and onset of asthma were unquantified due to the potential overlap with the quantified effects such as lower respiratory symptoms and hospitalizations. In addition, estimates of the effects of PM on low birth weight and reduced lung function growth in children are not presented. While these endpoints are significant in an assessment of the public health impacts of diesel exhaust emissions, there are currently few published investigations on these topics, and the results of the available studies are not entirely consistent (ARB, 2006). In summary, because only a subset of the total number of health outcomes is considered here, the estimates should be considered an underestimate of the total public health impact of diesel PM exposure.

II. Economic Valuation of Health Effects

This section describes the methodology for monetizing the value of avoiding adverse health impacts.

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 (EPA, 1999). This value is the mean estimate from five contingent valuation studies and 21 wage-risk studies. Contingent valuation and wage-risk studies examine the willingness to pay (or accept) for a minor decrease (or increase) in risk of premature death. 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.¹

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 1.201² to account for real income growth from 1990 through 2020, (EPA, 2004). 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 2006 dollars. After these adjustments, the value of avoiding one premature death is \$8.2 million in 2006, \$8.6 million in 2015 and \$9.2 million in 2025, all expressed in 2006 dollars.

¹ U.S. EPA's most recent regulatory impact analyses, (EPA, 2004, 2005), apply a different VSL estimate (\$5.5 million in 1999 dollars, with a 95 percent confidence interval between \$1 million and \$10 million). This revised value is based on more recent meta-analytical literature, and has not been endorsed by the Environmental Economics Advisory Committee (EEAC) of U.S. EPA's Science Advisory Board (SAB). Until U.S. EPA's SAB endorses a revised estimate, ARB staff continues to use the last VSL estimate endorsed by the SAB, i.e., \$4.8 million in 1990 dollars.

² U.S. EPA's real income growth adjustment factor for premature death incorporates an elasticity estimate of 0.4.

The U.S. EPA also uses the willingness-to-pay (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 2025, although at a lower rate (about 0.2% per year in lieu of 0.6% per year).

For work-loss days, the U.S. EPA uses an estimate of an individual's lost wages, (EPA, 2004), which ARB adjusts for projected real income growth, at a rate of approximately 1.5% per year.

“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 (COI) plus associated costs such as loss of time for work, recreation and household production. When adjusting these COI values for inflation, ARB uses the Consumer Price Index (CPI) for medical care rather than the CPI for all items.

Table 2 lists the valuation of avoiding various health effects, compiled from ARB and U.S. EPA publications, updated to 2006 dollars. The valuations based on WTP, as well as those based on wages, are adjusted for anticipated growth in real income.

**Table 3: Undiscounted Unit Values for Health Effects
(at various income levels in 2006 dollars)¹**

Health Endpoint	2006	2015	2025	References
Mortality				
Premature death (\$ million)	8.2	8.6	9.2	(EPA, 1999), p. 70-72, (EPA, 2000), (EPA, 2004) p. 9-121)
Hospital Admissions				
Cardiovascular (\$ thousands)	43	48	54	(ARB, 2003), p. 63
Respiratory (\$ thousands)	35	39	44	(ARB, 2003), p. 63
Minor Illnesses				
Acute Bronchitis	451	459	469	(EPA, 2004), 9-158
Lower Respiratory Symptoms	20	20	21	(EPA, 2004), 9-158
Work loss day	189	217	252	2002 California wage data, U.S. Department of Labor
Minor restricted activity day (MRAD)	64	65	66	(EPA, 2004), 9-159

¹The value for premature death is adjusted for projected real income growth, net of 0.4 elasticity. Wage-based values (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.

Benefits from the proposed Commercial Harbor Craft Rule are substantial. ARB staff estimates the benefits to be nearly \$2.0 billion using a 3% discount rate or \$1.3 billion using a 7% discount rate. (ARB follows U.S. EPA practice in reporting results using both 3% and 7% discount rates.) Nearly all of the monetized benefits result from avoiding premature death. The estimated benefits from avoided morbidity are less than \$30 million with a 3% discount rate and less than \$20 million with a 7% discount rate. Approximately three-fifths of the benefits are associated with reduced PM from direct sources, and the remaining two-fifths with reduced NOx.

III. Conclusion

The health benefits of implementing the proposed regulation are substantial. Staff estimates that the cumulative emissions reductions over the lifetime of the rule can be associated with approximately 310 fewer premature deaths, 70 fewer hospital admissions due to respiratory causes, 120 fewer hospital admissions due to cardiovascular causes, 8,100 fewer cases of asthma-related and other lower respiratory symptoms, 670 fewer cases of acute bronchitis, 53,000 fewer work loss days, and 300,000 fewer minor restricted activity days. The uncertainty range behind each estimated benefit is on order of +/- 50%. The estimated statewide benefits over 2009 to 2025 from these reductions in adverse health effects is about \$1.3 billion using a 7% discount rate or \$2.0 billion using a 3% discount rate.

REFERENCES:

(ARB, 1998) Air Resources Board. *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*. Appendix III, Part A, Exposure Assessment. April 1998.

(ARB, 2000) Air Resources Board. *Risk Reduction Plan to Reduce Particulate Matter Emissions from Diesel-Fueled Engines and Vehicles*.

<http://www.arb.ca.gov/diesel/documents/rrpapp.htm>; October 2000.

(ARB, 2002) Air Resources Board and Office of Environmental Health Hazard Assessment. *Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates, Staff Report*.

<http://www.arb.ca.gov/research/aaqs/std-rs/pm-final/pm-final.htm>; May 2002.

(ARB, 2003) Air Resources Board. *Final Research Report: The Economic Value of Respiratory and Cardiovascular Hospitalizations*. May 2003

<ftp://ftp.arb.ca.gov/carbis/research/apr/past/99-329.pdf>

(ARB, 2006) California Air Resources Board. *Emission Reduction Plan for Ports and Goods Movement in California*; April 2004.

http://www.arb.ca.gov/planning/gmerp/march21plan/appendix_a.pdf

(Dominici et al, 2005) *Revised analyses of the National Morbidity, Mortality, and Air Pollution Study: mortality among residents of 90 cities*. J Toxicol Environ Health A. Vol. 68(13-14):1071-92.

(Franklin et al, 2007) *Association between PM_{2.5} and all-cause and specific-cause mortality in 27 US communities*. J Expo Sci Environ Epidemiol. Vol. 17: 279-287.

(Krewski et al, 2000) Krewski D.; Burnett R.; Goldberg M.; Hoover K.; Stemiatycki J.; Jerrett M.; Abrahamovicz M.; White W. Health Effects Institute, Cambridge, Massachusetts. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*.

<http://es.epa.gov/ncer/science/pm/hei/Rean-ExecSumm.pdf>; 2000.

(Lloyd and Cackette, 2001) Lloyd, A.C.; Cackette, T.A.; J Air Waste Management Assoc. *Diesel Engines: Environmental Impact and Control*;; 51:809-847.

<http://www.arb.ca.gov/research/seminars/lloyd/AWMA2001/JAWMADieselCriticalReview.pdf>; 2001.

(Pope et al, 2002) Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. Journal of the American Medical Association. Vol. 287 (9): 1132-41.

(EPA, 1999) United States Environmental Protection Agency. *The Benefits and Costs of the Clean Air Act 1990 to 2010*. EPA-410-R-99-001. <http://www.epa.gov/air/sect812/copy99.html>; November 1999.

(EPA, 2000) United States Environmental Protection Agency., *Guidelines for Preparing Economic Analyses*. EPA240-R-00-003. <http://www.epa.gov/opei/pubsinfo.htm>; September 2000.

(EPA, 2003) United States Environmental Protection Agency, Assessment and Standards Division, Office of Transportation and Air Quality. *Draft Regulatory Impact Analysis: Control of Emissions of Air Pollution from Nonroad Diesel Engines*. EPA420-R-03-008. CD-ROM. Research Triangle Park, North Carolina. <http://www.epa.gov/otaq/cleaner-nonroad/r03008.pdf>; April 2003.

(EPA, 2004) United States Environmental Protection Agency. May, 2004. Final Regulatory Impact Analysis: Control of Emissions from Nonroad Diesel Engines. EPA-420-R-04-007. Office of Transportation and Air Quality. <http://www.epa.gov/otaq/regs/nonroad/equip-hd/2004fr.htm#ria>