The Dangers of the Dirtiest Diesels:

The Health and Welfare Impacts of Nonroad Heavy-Duty Diesel Engines and Fuels

The State and Territorial Air Pollution Program Administrators
and the
Association of Local Air Pollution Control Officials

June 2002

About STAPPA and ALAPCO

The State and Territorial Air Pollution Program Administrators (STAPPA) and the Association of Local Air Pollution Control Officials (ALAPCO) are the two national associations of state and local air pollution control officials in 54 states and territories and over 165 major metropolitan areas throughout the country. The members of STAPPA and ALAPCO have primary responsibility for implementing our nation's air pollution control laws and regulations. The associations serve to encourage the exchange of information and experience among air pollution control officials; enhance communication and cooperation among federal, state and local regulatory agencies; and facilitate air pollution control activities that will result in clean, healthful air across the country. STAPPA and ALAPCO share joint headquarters in Washington, DC.

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Acknowledgements

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Executive Summary

Over the past several years, EPA has taken landmark action to regulate two of the most significant categories of mobile source air pollution. The agency's Tier 2 motor vehicle emissions standards and national low-sulfur gasoline program will cut emissions from passenger cars and light-duty trucks, while the 2007 onroad heavy-duty diesel (HDD) engine and fuel rule will slash emissions from big diesel trucks. In spite of these truly remarkable accomplishments that will yield tremendous public health and environmental benefits across the country, more must still be done with respect to reducing emissions from mobile sources and fuels. The highest priority in this regard is the rigorous control of emissions from the last really big mobile source category remaining: nonroad HDD engines. These engines include construction (e.g., bulldozers and excavators), industrial (e.g., portable generators, airport service equipment and forklifts) and agricultural (e.g., tractors, combines, and irrigation pumps) equipment.

Nonroad HDD engines are huge contributors to elevated levels of ozone and fine particulate matter (PM) – representing a significant share of the emissions inventories for both oxides of nitrogen (NO_x) and particulate matter – thus posing a substantial threat to public health, including, among other things, premature mortality from exposure to $PM_{2.5}$. In fact, PM emissions from nonroad HDD engines are greater than PM emissions from onroad HDD engines, and this disparity will grow rapidly in the future as vehicles and engines complying with EPA's recently adopted 2007 onroad HDD engine and fuel rule are phased in. In addition, the Clean Air Scientific Advisory Committee has concluded that diesel exhaust is a likely human carcinogen at environmental levels of exposure, further heightening the need to take swift and aggressive action to control nonroad HDD engines and their fuel. Given the limited authority states and localities have to regulate heavy-duty engines and diesel fuel, rigorous new federal standards for nonroad HDD engines and nonroad diesel fuel, equivalent to those for onroad HDD engines and fuel and in the same timeframe, are imperative.

Accordingly, the State and Territorial Air Pollution Program Administrators (STAPPA) and the Association of Local Air Pollution Control Officials (ALAPCO) have called upon the U.S. Environmental Protection Agency (EPA) to move forward as quickly as possible to adopt a systems-based approach for regulating nonroad HDD emissions in a timeframe consistent with that established for regulating onroad HDD engines and fuel. Specifically, to achieve parity between nonroad and onroad standards, STAPPA and ALAPCO urge that future federal standards for nonroad HDD engines and fuel be based on the following key principles:

1. Availability of 15-ppm low-sulfur nonroad diesel fuel beginning in June 2006, subject to the same flexibilities and schedules provided under the onroad low-sulfur diesel fuel program;

2. Promulgation of Tier 3 nonroad HDD engine standards for PM (for all horsepower engines covered by the rule), based on emission reductions of 90+ percent (similar to the PM reductions achieved by the 2007 onroad HDD rule) to be fully applicable in 2007; and

3. Promulgation of Tier 4 nonroad HDD engine standards for NO_x (for 50 to 750 hp engines), based on emission reductions of 95+ percent (similar to the reductions achieved by the 2007 onroad HDD rule), to be phased in between 2007 and 2010.

STAPPA and ALAPCO commissioned this study to quantify the potential human health and economic benefits of adoption and implementation of a federal regulation consistent with the associations' nonroad HDD engine and fuel recommendations. The analysis presented here attempts to answer two questions: 1) What are the physical health and welfare effects of changes in ambient air quality resulting from reductions in NO_x, sulfur oxides (SO_x) and direct diesel PM emissions? and 2) How much are the changes in air quality worth to U.S. citizens as a whole in monetary terms?

Based on predicted changes in air quality, STAPPA and ALAPCO were able to estimate changes in health and welfare effects associated with air quality and, further, to attach estimated monetary values to these changes. Our findings are striking. Most dramatic is that **an estimated 8,522 premature deaths <u>per year</u> could be avoided** if EPA were to adopt and implement a nonroad HDD rule consistent with STAPPA and ALAPCO's recommendations. These avoided premature deaths carry with them an estimated monetary benefit of over $67 billion. With respect to welfare impacts, our analysis estimates monetary benefits of nearly $3.4 billion due to improvements in visibility.

Table ES-1, on the following page, summarizes the range of potential health and economic benefits associated with air quality changes from implementation of the recommended nonroad HDD rule. The statistics that appear in this table are an estimate of the number of avoided incidences per year of a given health impact.

To further illustrate the health benefits of a strong nonroad HDD rule, STAPPA and ALAPCO extended their analysis to estimate health and economic benefits on a state-by-state basis. These results are presented in Table ES-2.

Table ES-1
PRIMARY ESTIMATE OF POTENTIAL ANNUAL HEALTH BENEFITS ASSOCIATED WITH AIR QUALITY CHANGES RESULTING IN 2030 FROM STAPPA/ALAPCO'S RECOMMENDED NONROAD HDD ENGINE AND FUEL STANDARDS

Endpoint	Avoided Incidences (cases/year)	Monetary Benefits (millions 1999$, adjusted for growth in real income)
PM-related Endpoints		
Premature mortality (adults, 30 and over)	8,522	$64,251
Chronic bronchitis (adults, 26 and over)	5,647	$2,495
Hospital Admissions – Pneumonia (adults, over 64)	1,129	$21
Hospital Admissions – COPD (adults, 64 and over)	924	$10
Hospital Admissions – Asthma (65 and younger)	924	$10
Hospital Admissions – Cardiovascular (adults, over 64)	2,772	$51
Emergency Room Visits for Asthma (65 and younger)	2,156	<$5
Asthma Attacks (asthmatics, all ages)	180,598	B_a
Acute bronchitis (children, 8-12)	18,070	<$5
Lower respiratory symptoms (children, 7-14)	198,052	<$5
Upper respiratory symptoms (asthmatic children, 9-11)	198,565	$10
Work loss days (adults, 18-65)	1,580,512	$164
Minor restricted activity days (adults, age 18-65)	8,203,798	$441
Other PM-related health effects	U_1	B_1
Monetized Total Health-related PM Benefits	—	$67,455+$\mathbf{B_H}$

$\mathbf{B_H}$ is equal to the sum of all unmonetized categories (e.g, B_a+B_1).

Table ES-2
State-By-State
Benefits of Regulating Nonroad Heavy-Duty Diesel Engines & Fuels
– Avoided Incidences (Cases/Year) –

State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
AL	102	68	14	11	11	33	26	2,167	217	2,376	2,382	18,963	98,431	809
AK	15	10	2	2	2	5	4	310	31	340	341	2,711	14,072	116
AZ	130	86	17	14	14	42	33	2,765	277	3,033	3,041	24,202	125,624	1,033
AR	116	77	15	13	13	38	29	2,458	246	2,695	2,702	21,507	111,636	918
CA	770	510	102	84	84	251	195	16,324	1,633	17,902	17,948	142,864	741,549	6,097
CO	151	100	20	16	16	49	38	3,204	321	3,514	3,523	28,043	145,561	1,197
CT	74	49	10	8	8	24	19	1,565	157	1,716	1,721	13,695	71,085	584
DE	23	15	3	2	2	7	6	488	49	535	537	4,271	22,168	182
DC	8	5	1	1	1	2	2	162	16	177	178	1,416	7,349	60
FL	424	281	56	46	46	138	107	8,978	898	9,846	9,871	78,571	407,828	3,353
GA	209	138	28	23	23	68	53	4,420	442	4,847	4,860	38,683	200,790	1,651
HI	26	17	3	3	3	9	7	559	56	613	615	4,891	25,388	209
ID	63	42	8	7	7	20	16	1,335	134	1,463	1,467	11,679	60,622	498
IL	431	286	57	47	47	140	109	9,132	914	10,014	10,040	79,916	414,809	3,411
IN	236	156	31	26	26	77	60	4,994	500	5,477	5,491	43,707	226,867	1,865
IA	267	177	35	29	29	87	68	5,655	566	6,201	6,217	49,489	256,877	2,112
KS	227	150	30	25	25	74	57	4,809	481	5,273	5,287	42,083	218,438	1,796
KY	109	72	14	12	12	36	28	2,315	232	2,539	2,546	20,262	105,174	865
LA	120	79	16	13	13	39	30	2,537	254	2,782	2,790	22,205	115,257	948
ME	28	19	4	3	3	9	7	601	60	659	660	5,257	27,285	224

A = Premature mortality (adults, 30 and over)	H = Asthma attacks (asthmatics, all ages)
B = Chronic bronchitis (adults, 26 and over)	I = Acute bronchitis (children, 8-12)
C = Hospital admissions, pneumonia (adults, over 64)	J = Lower respiratory symptoms (children, 7-14)
D = Hospital admissions, chronic obstructive pulmonary disease (COPD) (adults, 64 and over)	K = Upper respiratory symptoms (asthmatic children, 9-11)
E = Hospital admissions, asthma (adults, 65 and younger)	L = Work loss days (adults, 18-65)
F = Hospital admissions, cardiovascular (adults, over 64)	M = Minor restricted activity days (adults, 18-65)
G = Emergency room visits, asthma (adults, 65 and younger)	

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State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
MD	116	77	15	13	13	38	29	2,458	246	2,696	2,703	21,512	111,661	918
MA	193	128	26	21	21	63	49	4,099	410	4,495	4,506	35,870	186,186	1,531
MI	245	163	33	27	27	80	62	5,198	520	5,701	5,715	45,492	236,131	1,942
MN	268	178	36	29	29	87	68	5,678	568	6,227	6,243	49,693	257,938	2,121
MS	86	57	11	9	9	28	22	1,828	183	2,004	2,010	15,996	83,031	683
MO	215	143	29	23	23	70	55	4,565	457	5,006	5,019	39,952	207,375	1,705
MT	88	58	12	10	10	29	22	1,863	186	2,043	2,049	16,307	84,641	696
NE	177	118	24	19	19	58	45	3,759	376	4,123	4,133	32,900	170,770	1,404
NV	64	43	9	7	7	21	16	1,360	136	1,491	1,495	11,901	61,771	508
NH	27	18	4	3	3	9	7	565	57	619	621	4,943	25,658	211
NJ	184	122	24	20	20	60	47	3,906	391	4,283	4,294	34,182	177,424	1,459
NM	41	27	5	4	4	13	10	875	88	959	962	7,655	39,734	327
NY	376	249	50	41	41	122	95	7,963	797	8,732	8,755	69,686	361,710	2,974
NC	216	143	29	23	23	70	55	4,585	459	5,028	5,041	40,126	208,280	1,713
ND	184	122	24	20	20	60	47	3,902	390	4,279	4,290	34,146	177,240	1,457
OH	341	226	45	37	37	111	86	7,229	723	7,928	7,948	63,266	328,390	2,700
OK	134	89	18	15	15	44	34	2,834	284	3,108	3,116	24,802	128,736	1,059
OR	111	74	15	12	12	36	28	2,353	235	2,581	2,588	20,596	106,906	879
PA	248	164	33	27	27	81	63	5,259	526	5,768	5,783	46,029	238,916	1,964
RI	17	11	2	2	2	6	4	362	36	397	398	3,170	16,456	135

A = Premature mortality (adults, 30 and over)	H = Asthma attacks (asthmatics, all ages)
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D = Hospital admissions, chronic obstructive pulmonary disease (COPD) (adults, 64 and over)	K = Upper respiratory symptoms (asthmatic children, 9-11)
E = Hospital admissions, asthma (adults, 65 and younger)	L = Work loss days (adults, 18-65)
F = Hospital admissions, cardiovascular (adults, over 64)	M = Minor restricted activity days (adults, 18-65)
G = Emergency room visits, asthma (adults, 65 and younger)	

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Benefits of Regulating Nonroad Heavy-Duty Diesel Engines & Fuels
– Avoided Incidences (Cases/Year) –

State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
SC	98	65	13	11	11	32	25	2,068	207	2,268	2,274	18,097	93,934	772
SD	134	89	18	15	15	44	34	2,844	285	3,119	3,127	24,887	129,179	1,062
TN	148	98	20	16	16	48	37	3,135	314	3,438	3,447	27,435	142,403	1,171
TX	584	387	77	63	63	190	148	12,377	1,238	13,573	13,609	108,320	562,243	4,623
UT	95	63	13	10	10	31	24	2,008	201	2,202	2,208	17,572	91,210	750
VT	16	10	2	2	2	5	4	336	34	368	369	2,937	15,243	125
VA	168	111	22	18	18	55	42	3,551	355	3,895	3,905	31,080	161,325	1,326
WA	171	113	23	19	19	56	43	3,624	363	3,974	3,984	31,711	164,601	1,353
WV	34	23	5	4	4	11	9	728	73	798	800	6,367	33,049	272
WI	185	123	25	20	20	60	47	3,930	393	4,310	4,321	34,398	178,544	1,468
WY	27	18	4	3	3	9	7	579	58	635	637	5,068	26,305	216
TOTAL	**8,522**	**5,647**	**1,129**	**924**	**924**	**2,772**	**2,156**	**180,598**	**18,070**	**198,052**	**198,565**	**1,580,512**	**8,203,798**	**$67,455**

A = Premature mortality (adults, 30 and over)	**H = Asthma attacks (asthmatics, all ages)**
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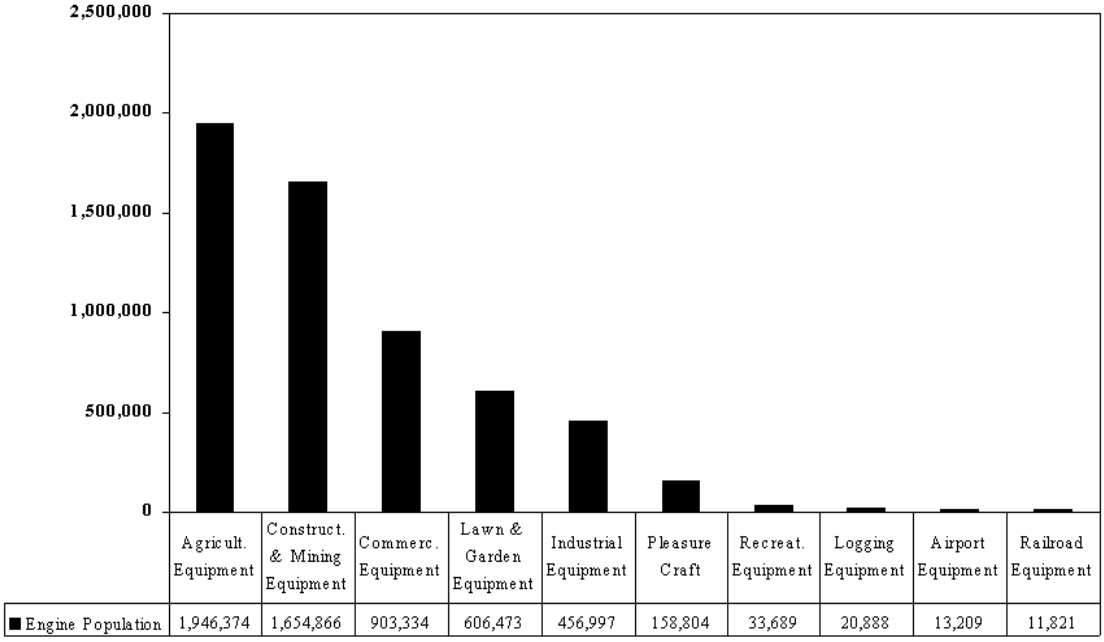
F = Hospital admissions, cardiovascular (adults, over 64)	**M = Minor restricted activity days (adults, 18-65)**
G = Emergency room visits, asthma (adults, 65 and younger)	

I. Background and Introduction

In the past several years, EPA has taken landmark action to regulate two of the most significant categories of mobile source air pollution. The agency's Tier 2 motor vehicle emission standards and national low-sulfur gasoline program will cut emissions from passenger cars and light-duty trucks, while the 2007 onroad heavy-duty diesel (HDD) engine and fuel rule will slash emissions from big diesel trucks. In spite of these truly remarkable accomplishments that will yield tremendous public health and environmental benefits across the country, more must still be done with respect to reducing emissions from mobile sources and fuels. The highest priority in this regard is the rigorous control of emissions from the last really big mobile source category remaining: nonroad HDD engines. These engines include construction (e.g., bulldozers and excavators), industrial (e.g., portable generators, airport service equipment and forklifts) and agricultural (e.g., tractors, combines and irrigation pumps) equipment. These engines are distributed around the country in proportions very similar to the distribution of onroad HDDs nationwide. Figure 1 identifies the nonroad diesel engine categories and the estimated number of engines in each category.

Figure 1
Nonroad Diesel Engine Population - 2000



A. Trends in Nonroad Diesel Emissions

Nonroad diesel engines are huge contributors to elevated levels of ozone and fine particulate matter – representing a substantial share of the emissions inventories for both oxides of nitrogen (NO_x) and particulate matter (PM) – thus posing a substantial threat to public health, including, among other things, premature mortality from exposure to $PM_{2.5}$. Table 1 provides data on nonroad diesel emissions of NO_x, PM_{10} and $PM_{2.5}$, as well as an indication of the percentage of the total mobile source inventory represented by each emissions level.

Table 1 – TRENDS IN NONROAD DIESEL EMISSIONS

	1970	1980	1990	1999
NO_x	1,109[1] 12%[2]	2,125 17.5%	2513 21%	2707 19%
PM_{10}	154 19.6%	263 33.5%	301 36%	253 34%
$PM_{2.5}$	– –	– –	277 39%	233 36%

Source: EPA, "National Air Quality and Emissions Trends Report, 1999."

[1] Units represent emissions in thousand short tons
[2] Percentages represent proportion of total transportation emissions inventory

According to EPA's most recent emissions trends report, NO_x emissions from nonroad diesels totaled 1.1 million tons in 1970 and 2.7 million tons in 1999, representing a 250-percent increase. Although NO_x emissions from onroad HDDs are slightly higher than from nonroad diesels – 1.67 million tons in 1970 and 3.62 million tons in 1999 – their rate of growth, at 215 percent, is lower than that for nonroad diesels over the same time period.

With respect to coarse particulate matter, or PM_{10}, emissions from nonroad diesels have not only grown substantially between 1970 and 1999 – from 154,000 tons to 253,00 tons – their emissions in 1999 were 36 percent higher than from onroad HDDs, which emitted 186,000 tons in that year.

An even greater disparity exists with respect to $PM_{2.5}$ emissions, with nonroad diesels emitting 42-percent higher emissions than onroad HDDs in 1999 – 233,000 versus 164,000 tons.

It is important to note that the disparity between nonroad and onroad PM emissions will grow rapidly in the future, as vehicles and engines complying with EPA's recently adopted 2007 onroad HDD engine and fuel rule are phased in.

Heightening still further the need to take swift and aggressive action to control nonroad HDD engines and their fuels is the fact that the Clean Air Scientific Advisory Committee has concluded that diesel exhaust is a likely human carcinogen at environmental levels of exposure. Given the limited authority states and localities have to regulate heavy-duty engines and their fuels, rigorous new federal standards for nonroad HDD engines and nonroad diesel fuel, equivalent to those for onroad HDD engines and fuel and in the same timeframe, are imperative.

B. The Regulation of Nonroad HDDs

On October 23, 1998, when EPA adopted Tier 3 NO_x standards for nonroad HDD engines (63 *Federal Register* 56968), the agency made three commitments: 1) to complete a technical review of the standards by the end of 2001, 2) to include as part of the review, proposal and adoption of Tier 3 PM standards for nonroad HDD engines by the end of 2001 (as stated in the rule's Preamble, "EPA has a number of reasons for deferring the establishment of Tier 3 PM standards at this time, but is actively working toward this goal. The Agency believes that Tier 3 PM standards will be more appropriately discussed in the context of the improved technical understanding that will exist by the time of the 2001 feasibility review….") and 3) to complete the development of an appropriate transient test procedure for nonroad diesel engines that ensures in-use PM emission reductions.

Specifically, EPA stated in the Preamble to its October 1998 rulemaking that "[o]ver the next several years, EPA will be actively engaged in programs to evaluate technology developments and progress toward meeting the new standards…This effort will culminate in a special review, to be concluded in 2001….The review will also include proposal and adoption of appropriate Tier 3 standards for PM." The agency goes on to say in the Preamble that "[k]ey among EPA activities directed toward completing the 2001 feasibility review are those related to adoption of a more effective PM control program for nonroad diesel engines….Establishing an appropriate test cycle is critical to the success of a more effective PM control program...Therefore, EPA is moving forward with development of a transient component to the nonroad engine test cycle to control the transient element of PM emissions generation…."

Subsequent to the publication if its 1998 rule, EPA expressed the intention to not limit its additional rulemaking efforts only to Tier 3 PM standards. Instead, the Agency indicated that its 2001 technical review would include a complete discussion of a more comprehensive next phase of regulation for nonroad HDD engines, to include not only Tier 3 PM standards, but also Tier 4 NO_x standards and low-sulfur nonroad diesel fuel. STAPPA and ALAPCO strongly supported this intention, in that it was consistent with previous recommendations made by the associations and with the associations' long-held vision of an integrated systems-based approach for regulating nonroad HDD engines.

In their November 21, 1997 comments on the *Notice of Proposed Rulemaking for the Control of Emissions of Air Pollution from Nonroad Diesel Engines* (62 FR 50152), which preceded EPA's October 23, 1998 final rule, STAPPA and ALAPCO stated that EPA should seriously consider the viability of even more stringent nonroad diesel engine standards in the context of the

technical feasibility review scheduled for 2001. "Compared to highway vehicles, emissions from nonroad equipment are relatively under controlled. Given the current inequity in emission control requirements and the availability of known control technologies and strategies, reducing emissions from this source sector could represent one of the more cost-effective available control options…. Once final, the nonroad diesel engine standards proposed by EPA will dictate the stringency of the controls for this sector well into the next millennium." Now, with the advances in fuel quality, engine technology and health research that have occurred over the past four years, EPA should be compelled to take swift action to bring parity to nonroad and onroad standards.

There is more than abundant evidence that further regulation of nonroad HDD engines emissions is imperative in order to assist states and localities in achieving and sustaining clean air and public health protection goals. Accordingly, STAPPA and ALAPCO have called upon EPA to move forward as quickly as possible to adopt a systems-based approach to regulate nonroad HDD engines and fuel in a manner consistent with that established for regulating onroad HDD engines and fuel.

C. STAPPA and ALAPCO's Recommendations for Regulating Nonroad HDD Engines and Fuels

Specifically, to achieve parity between nonroad and onroad standards, STAPPA and ALAPCO urge that future federal standards for nonroad HDDs engines and fuel be based on the following key principles:

1. Availability of 15-ppm low-sulfur nonroad diesel fuel beginning in June 2006, subject to the same flexibilities and schedules provided under the onroad low-sulfur diesel fuel program;

2. Promulgation of Tier 3 nonroad HDD engine standards for PM (for all horsepower engines covered by the rule), based on emission reductions of 90+ percent (similar to the PM reductions achieved by the 2007 onroad HDD rule) to be fully applicable in 2007; and

3. Promulgation of Tier 4 nonroad HDD engine standards for NO_x (for 50 to 750 hp engines), based on emission reductions of 95+ percent (similar to the reductions achieved by the 2007 onroad HDD rule), to be phased in between 2007 and 2010.

In addition, the associations have called for there to be no loss in NO_x emissions reductions expected beginning in 2006 and, further, for a strong program to ensure that in-use emissions from nonroad HDDs are not compromised by durability issues, the use of defeat devices or other factors.

D. Scope of This Report and Methodology

In this report, STAPPA and ALAPCO assess the potential health and economic benefits of a federal regulation consistent with these recommendations. The analysis presented here attempts to answer two questions:

1. What are the physical health and welfare effects of changes in ambient air quality resulting from reductions in NO_x, sulfur oxides (SO_x) and direct diesel PM emissions? and

2. How much are the changes in air quality worth to U.S. citizens as a whole in monetary terms?

This analysis includes three steps, each of which will be discussed separately in the sections that follow:

1. Calculation of the impact that the STAPPA/ALAPCO-recommended nonroad HDD engine standards and cleaner nonroad diesel fuel standards will have on the nationwide inventories for PM, NO_x and SO_x;

2. Assessment of the change in ambient concentrations of PM that will result from the changes in nationwide inventories of precursor pollutants; and

3. A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the changes in ambient concentrations of various pollutants.

It is important to note that there are significant categories of benefits that could not be monetized (or, in many cases, even quantified), resulting in a significant underestimate of potential benefits. For example, while the ozone benefits of tighter standards for nonroad vehicles and fuels are expected to be immense, it was beyond the scope of this study to attempt to quantify them. Also, appropriate tools for modeling changes in ambient concentrations of carbon monoxide (CO) or toxic air pollutants for input into a national benefits analysis do not exist. These pollutants, of course, have been linked to numerous adverse health effects; however, it was not possible to quantify the CO- or air toxics-related health or welfare benefits of the standards.

This study closely mirrors the U.S. Environmental Protection Agency's (EPA's) benefits assessment of the final 2007 onroad HDD engine and fuel rule. We have used the same general methodology in this benefits analysis. Key elements of the methodology include the following:

- Updating concentration-response (C-R) functions for PM-related premature mortality;

- Updating C-R functions for PM-related hospital admissions;

- Reporting asthma attacks as a separate endpoint and adjusting minor restricted activity days to remove the possibility of double-counting of asthma attacks;

- Relying only on the value of statistical life method to value reductions in the risk of premature mortality in the primary estimate; and

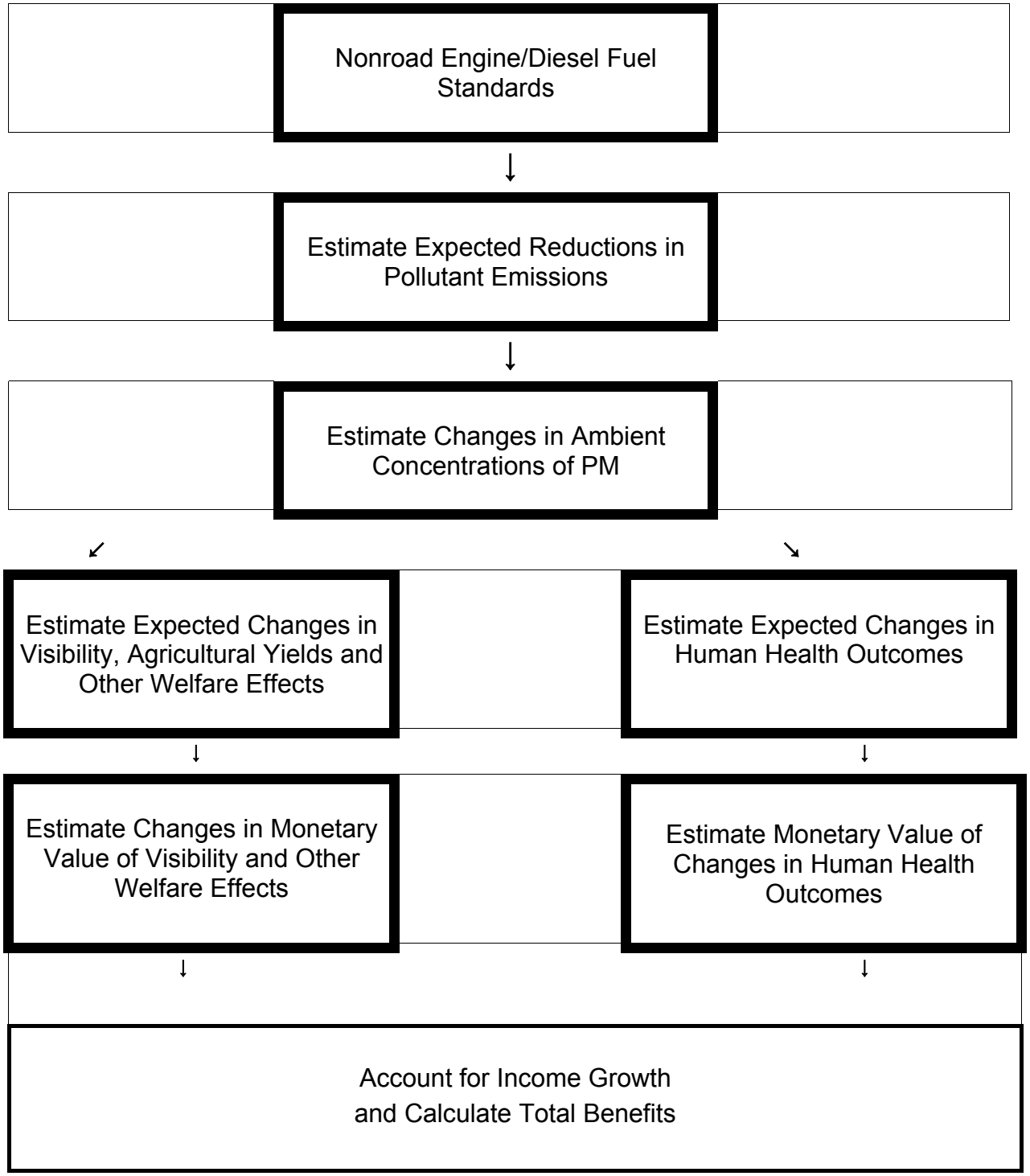
- Adjusting benefits to reflect the expected growth in willingness to pay (WTP) for health and environmental benefits as real income grows over time.

The estimated benefits presented in this report represent the potential benefits in 2030 of a strong systems-based (i.e., addressing both the engine and fuel) approach to controlling emissions from nonroad HDDs, consistent with STAPPA and ALAPCO's recommendations, as outlined above. (Please note that the predicted emissions reductions that will result from such a recommended regulation have yet to occur and, therefore, the actual changes in human health and welfare outcomes to which economic values are ascribed are predictions.)

Figure 2, on the following page, illustrates the steps necessary to link the recommended nonroad HDD engine and fuel regulatory standards with economic measures of benefits. The first two steps involve the specification and implementation of the regulation. First, the specific engine and fuel standards for reducing air pollution from heavy-duty vehicles and fuels are established. Next, the changes in pollutant emissions resulting from the vehicle and fuel changes are calculated, along with predictions of emissions for other industrial sectors in the baseline. The predicted emissions are then used as inputs to air quality models that predict ambient concentrations of pollutants over time and space. These concentrations depend on climatic conditions and complex chemical interactions. In its recently completed 2007 onroad HDD rule, EPA used the best available air quality models to estimate the changes in ambient concentrations (from baseline levels) on which the benefits analysis was based. Proportional improvements in air quality were estimated to result from equivalent PM emissions reductions.

Changes in ambient concentrations will lead to new levels of environmental quality in the U.S., reflected both in human health and in non-health welfare effects. Thus, the predicted changes in ambient air quality serve as inputs into functions that predict changes in health and welfare outcomes. The term "endpoints" is used to refer to specific effects that can be associated with changes in air quality. For changes in risks to human health from changes in PM, quantified endpoints include changes in mortality and in a number of pollution-related non-fatal health effects. To estimate these endpoints, EPA combines changes in ambient air quality levels with epidemiological evidence about population health response to pollution exposure. For welfare effects, the endpoints are defined in terms of levels of physical damage (for materials damage), economic output for (agriculture and forestry), light transmission (for visibility), and increases in terrestrial and estuarine nutrient loading (for ecological effects).

Figure 2 – STEPS IN THE NONROAD DIESEL ENGINE/FUEL BENEFITS ANALYSIS



II. Analysis and Findings

A. PM, NO_x and SO_x Emissions Impacts

It is estimated that under the federal regulations that are currently in place, nonroad HDD engines will emit about 162,000 tons of direct PM in 2030. Adoption of a 15-ppm nonroad diesel fuel sulfur standard and a PM engine standard equivalent to 0.01 grams per brake-horsepower hour (g/bhp-hr), as recommended by STAPPA and ALAPCO, would reduce direct PM emissions by at least 93 percent, or about 151,000 tons.

With respect to NO_x, it is estimated that under current regulations, about 1.3 million tons will be emitted from nonroad HDDs in 2030. Adoption of an engine standard of 0.2 g/bhp-hr, along with 15-ppm sulfur fuel, would result in a reduction of at least 90 percent, or almost 1.2 million tons.

Total SO_x emissions in 2030 are expected to be approximately 400,000 tons. Reducing the sulfur content of nonroad diesel fuel from 3000 ppm to 15 ppm would bring about an approximate 98 percent reduction in SO_x emissions, or about 392,000 tons.

The overall reductions in direct PM, NO_x and SO_x emissions from STAPPA/ALAPCO's recommended nonroad HDD engine and fuel standards are compared to the equivalent reductions per year from the recently adopted Tier 2 and 2007 onroad HDD rules in Table 2. As indicated, the direct PM reductions from the recommended nonroad standards are estimated to be substantially greater than from either Tier 2 or onroad HDD. Similarly, the SO_x reductions would be greater from the recommended nonroad standards than from either of the others. For NO_x, on the other hand, the Tier 2 rule provides the greatest reduction, followed by the 2007 onroad HDD rule, with the recommended nonroad approach giving the least (though still substantial) reduction.

Table 2 - ESTIMATED OVERALL EMISSIONS REDUCTIONS IN 2030 (tons)

	Tier 2 Rule	2007 Onroad HDD Rule	STAPPA/ALAPCO-Recommended Nonroad HDD Standards
PM	3,600	109,000	150,660
NOx	2,790,000	2,570,000	1,170,000
SOx	281,000	141,000	392,000

In the years before 2030, the benefits from the recommended nonroad HDD engine and fuel standards will be less than those estimated here, because the compliant fleet will not be fully phased in. Moreover, to the extent that a lower ratio of benefits to costs early in the program is the result of the mismatch of costs and benefits in time, a simple analysis of an individual year would be misleading. A more appropriate means of capturing the impacts of timing differences in benefits and costs would be to produce a net present value comparison of the costs and benefits over some period of years. Unfortunately, while this is relatively straightforward for the costs, it is currently not feasible to do a multi-year analysis of the benefits, as this would require a significant amount of air quality modeling to capture each year. Such modeling was beyond the scope of this study. Instead, for the purpose of the benefits calculations, it is assumed that 2030 is a representative year for the fully implemented rule. The resulting analysis represents a snapshot of benefits in a future year in which the nonroad HDD fleet consists almost entirely of vehicles and fuel meeting STAPPA/ALAPCO's recommended engine and fuel standards.

B. Air Quality Impact on PM from Reductions in Precursor Pollutants

While it would be ideal to carry out extensive air quality modeling to determine the overall air quality benefits of STAPPA and ALAPCO's recommendations for nonroad HDD diesel engine and fuel standards, such modeling is well beyond the scope of this effort. However, from knowing the emissions reductions that would result from the recommended nonroad standards and comparing these to the emissions reductions from the 2007 onroad HDD rule, it is possible to estimate the benefits that would result.

With regard to direct PM emissions, as indicated in Table 2, above, there would be substantially greater reductions from the recommended nonroad HDD standards compared to the 2007 onroad HDD rule – 38 percent more. However, in estimating overall PM impacts, it is also important to account for indirect secondary PM formation, especially from NO_x and SO_x transformations into nitrates and sulfates, respectively.

To determine the relative impacts of NO_x and SO_x on secondary PM, an analysis of the EPA estimates for the Tier 2 rule and the 2007 onroad HDD onroad rule was conducted. For each rule, the total tons of NO_x, SO_x, and direct PM was estimated, along with the overall PM benefit. Looking at both assessments, it was possible to estimate the relative impact of NO_x and SO_x reductions. Based on this review of the Tier 2 and 2007 onroad HDD rules, a conservative estimate is that approximately 3 percent of the NO_x and SO_x emissions are converted into secondary PM.

With respect to ozone, while it is clear that the emissions reductions resulting from the recommended nonroad HDD standards will reduce ozone levels in much of the country, as discussed above, photochemical modeling is beyond the scope of this effort. Therefore, no estimates of ozone benefits have been made.

C. Assessment of Human Health Benefits

The most significant monetized benefits of reducing ambient concentrations of PM are attributable to reductions in health risks associated with air pollution. EPA's 1996 Criteria Document for PM lists numerous health effects known to be linked to ambient concentrations of this pollutant. Individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects are described below.

Accounting for Potential Health Effect Thresholds

When conducting clinical (chamber) and epidemiological studies, C-R functions may be estimated with or without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a non-zero risk of response to at least one segment of the population.

The possible existence of an effect threshold is a very important scientific question and a key issue for policy analyses such as this one. In the benefits analyses for some recent Regulatory Impact Analyses (see EPA's PM NAAQS RIA, Regional Haze RIA, and NO_x SIP Call RIA), the low-end estimate of benefits assumed a threshold in PM health effects at 15 $\mu g/m^3$. However, the Science Advisory Board (SAB) subsequently advised EPA that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ or any other specific threshold for the PM-related health effects considered in this analysis (EPA-SAB-Council-ADV-99-012, 1999). Therefore, for

this benefits analysis, it was assumed that there are no thresholds for modeling health effects. It is not appropriate to adopt a threshold for use in either the primary analysis or any alternative calculations, because no adequate scientific evidence exists to support such a calculation.

Quantifying and Valuing Individual Health Endpoints

PM-only health effects include premature mortality, chronic bronchitis, acute bronchitis, upper and lower respiratory symptoms, and work loss days. Other health effects related to PM include hospital admissions, asthma attacks, and minor restricted activity days.

While a broad range of serious health effects has been associated with exposure to elevated PM levels (as noted earlier and described more fully in EPA's 1996 PM Criteria Documents), only a subset of health effects is included in this quantified benefit analysis. Health effects are excluded from this analysis for three reasons: 1) the possibility of double counting (such as hospital admissions for specific respiratory diseases), 2) uncertainties in applying effect relationships based on clinical studies to the affected population, or 3) lack of an established C-R relationship. Table 3 lists the endpoints and studies included in this analysis.

TABLE 3 – ENDPOINTS AND STUDIES INCLUDED IN THE PRIMARY ANALYSIS

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Long-term exposure	$PM_{2.5}$	Krewski, et al. (2000)[A]	Adults, 30 and older
Chronic Illness			
Chronic Bronchitis (pooled estimate)	$PM_{2.5}$ PM_{10}	Abbey, et al. (1995) Schwartz, et al. (1993)	> 26 years > 29 years
Hospital Admissions			
COPD	PM	Samet, et al. (2000)	> 64 years
Pneumonia	PM	Samet, et al. (2000)	> 64 years
Asthma	PM	Sheppard, et al. (1999)	< 65 years
Total Cardiovascular	PM	Samet, et al. (2000)	> 64 years
Asthma-Related ER Visits	PM	Schwartz, et al. (1993)	All ages
Other Illness			
Asthma Attacks	PM,	Whittemore and Korn (1980)	Asthmatics, all ages
Acute Bronchitis	PM	Dockery et al. (1996)	Children, 8-12 years
Upper Respiratory Symptoms	PM	Pope et al. (1991)	Asthmatic children, 9-11
Lower Respiratory Symptoms	PM	Schwartz et al. (1994)	Children, 7-14 years
Work Loss Days	PM	Ostro (1987)	Adults, 18-65 years
Minor Restricted Activity Days (minus asthma attacks)	PM,	Ostro and Rothschild (1989)	Adults, 18-65 years

[A] Estimate derived from Table 31, $PM_{2.5}$ (DC), All Causes Model (Relative Risk =1.12 for a 24.5-$\mu g/m^3$ increase in mean $PM_{2.5}$).

The appropriate economic value of a change in a health effect depends on whether the health effect is viewed *ex ante* (before the effect has occurred) or *ex post* (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is, therefore, *ex ante* WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, the cost of treating or mitigating the effect was used as a primary estimate. For example, for the valuation of hospital admissions the avoided medical costs were used as an estimate of the value of avoiding the health effects causing the admission. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment, but not the value of avoided pain and suffering from the health effect. Table 4 summarizes the value estimates per health effect that were used in this analysis.

In the following sections, we discuss the individual health endpoints and the C-R functions selected to provide quantified estimates of the avoided health effects associated with the recommended nonroad HDD engine and fuel standards. In addition, how these changes in health effects should be valued is discussed and the value functions selected to provide monetized estimates of the value of changes in health effects indicated.

Table 4 – UNIT VALUES USED FOR ECONOMIC VALUATION OF HEALTH ENDPOINTS

Health or Welfare Endpoint	Estimated Value Per Incidence (1999$) Central Estimate	Derivation of Estimates
Premature Mortality	$6 million per statistical life	Value is the mean of value-of-statistical-life estimates from 26 studies (5 contingent valuation and 21 labor market studies) reviewed for the Section 812 Costs and Benefits of the Clean Air Act, 1990-2010 (EPA, 1999).
Chronic Bronchitis (CB)	$331,000	Value is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., 1991) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB.

Table 4 – UNIT VALUES USED FOR ECONOMIC VALUATION OF HEALTH ENDPOINTS

Health or Welfare Endpoint	Estimated Value Per Incidence (1999$) Central Estimate	Derivation of Estimates
Hospital Admissions		
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	$12,378	The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Elixhauser (1993).
Pneumonia (ICD codes 480-487)	$14,693	The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Elixhauser (1993).
Asthma Admissions	$6,634	The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Elixhauser (1993).
All Cardiovascular (ICD codes 390-429)	$18,387	The COI estimates are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular illnesses) reported in Elixhauser (1993).
Emergency Room Visits for Asthma	$299	COI estimate based on data reported by Smith, et al. (1997).
Respiratory Ailments Not Requiring Hospitalization		
Upper Respiratory Symptoms (URS)	$24	Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the 7 different types of URS.
Lower Respiratory Symptoms (LRS)	$15	Combinations of the 4 symptoms for which WTP estimates are available that closely match those listed by Schwartz, et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS.
Acute Bronchitis	$57	Average of low and high values recommended for use in Section 812 analysis (Neumann, et al. 1994)
Restricted Activity and Work Loss Days		
Work Loss Days (WLDs)	Variable	Regionally adjusted median weekly wage for 1990 divided by 5 (adjusted to 1999$) (US Bureau of the Census, 1992).
Minor Restricted Activity Days (MRADs)	$48	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986).

Quantifying Premature Mortality

Both acute and chronic exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. Because of the extreme nature of this endpoint and the high monetary value associated with risks to life, reductions in the risk of premature mortality are the most important health endpoints quantified in this analysis. Although these endpoints account for over 90 percent of the total monetized benefits, considerable uncertainty exists, both among economists and policymakers, as to the appropriate way to value reductions in mortality risks. Because of these factors, a more detailed discussion of premature mortality is provided than for other health effects.

Health researchers have consistently linked air pollution, especially PM, with increases in premature mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. Much of this literature is summarized in EPA's 1996 PM Criteria Document (1996a).

Just within the past few months, this linkage was reinforced by a major new study. Long-term exposure to levels of air pollution common in many U.S. metropolitan areas was found to increase the risk of death from lung cancer and other heart-lung diseases.

The analysis is based on data collected by the American Cancer Society (ACS) as part of the Cancer Prevention Study II (CPS-II), an ongoing prospective mortality study of approximately 1.2 million adults. ACS volunteers enrolled individual participants in the fall of 1982. Participants resided in all 50 states, the District of Columbia, and Puerto Rico; participation was restricted to persons who were aged 30 years or older and who were members of households with at least one individual aged 45 years or older. Participants completed a confidential questionnaire, which included questions about age, sex, weight, height, smoking history, alcohol use, occupational exposures, diet, education, marital status, and other characteristics.

This study demonstrated associations between ambient fine particulate air pollution and elevated risks of both cardiopulmonary and lung cancer mortality. Each 10-$\mu g/m^3$ elevation in long-term average $PM_{2.5}$ ambient concentrations was associated with approximately a 4-percent, 6-percent, and 8-percent increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively.

The findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many U.S. metropolitan areas is an important risk factor for cardiopulmonary mortality. In addition, the large cohort and extended follow-up have provided an unprecedented opportunity to evaluate associations between air pollution and lung cancer mortality. Elevated fine particulate air pollution exposures were associated with significant increases in lung cancer mortality. Although potential effects of other unaccounted for factors cannot be excluded with certainty, the associations between fine particulate air pollution and lung cancer mortality, as well as cardiopulmonary mortality, are observed even after controlling for cigarette smoking, BMI, diet, occupational exposure, other individual risk factors, and after controlling for regional and other spatial differences.

The largest monetized health benefit is associated with reductions in the risk of premature mortality, which accounts for over $60 billion, or more than 90 percent of total monetized health benefits. The next largest benefit is for chronic bronchitis reductions, although this value is more than an order of magnitude lower than for premature mortality. Minor restricted activity days, work loss days, and worker productivity account for the majority of the remaining benefits.

The remaining categories account for less than $10 million each, however, they represent a large number of avoided incidences affecting many individuals.

Table 5 presents the results of STAPPA and ALAPCO's analysis with respect to the health and economic benefits of a strong nonroad HDD rule. Table 6 presents the state-by-state breakout of these results.

Table 5 – PRIMARY ESTIMATE OF POTENTIAL ANNUAL HEALTH BENEFITS ASSOCIATED WITH AIR QUALITY CHANGES RESULTING IN 2030 FROM STAPPA/ALAPCO'S RECOMMENDED NONROAD HDD ENGINE AND FUEL STANDARDS

Endpoint	Avoided Incidence (cases/year)	Monetary Benefits (millions 1999$, adjusted for growth in real income)
PM-related Endpoints		
Premature mortality (adults, 30 and over)	8,522	$64,251
Chronic bronchitis (adults, 26 and over)	5,647	$2,495
Hospital Admissions – Pneumonia (adults, over 64)	1,129	$21
Hospital Admissions – COPD (adults, 64 and over)	924	$10
Hospital Admissions – Asthma (65 and younger)	924	$10
Hospital Admissions – Cardiovascular (adults, over 64)	2,772	$51
Emergency Room Visits for Asthma (65 and younger)	2,156	<$5
Asthma Attacks (asthmatics, all ages)	180,598	B_a
Acute bronchitis (children, 8-12)	18,070	<$5
Lower respiratory symptoms (children, 7-14)	198,052	<$5
Upper respiratory symptoms (asthmatic children, 9-11)	198,565	$10
Work loss days (adults, 18-65)	1,580,512	$164
Minor restricted activity days (adults, age 18-65)	8,203,798	$441
Other PM-related health effects	U_1	B_1
Monetized Total Health-related PM Benefits	—	$67,455+$\mathbf{B_H}$

$\mathbf{B_H}$ is equal to the sum of all unmonetized categories (e.g., B_a+B_1).

Table 6
State-By-State
Benefits of Regulating Nonroad Heavy-Duty Diesel Engines & Fuels
– Avoided Incidences (Cases/Year) –

State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
AL	102	68	14	11	11	33	26	2,167	217	2,376	2,382	18,963	98,431	809
AK	15	10	2	2	2	5	4	310	31	340	341	2,711	14,072	116
AZ	130	86	17	14	14	42	33	2,765	277	3,033	3,041	24,202	125,624	1,033
AR	116	77	15	13	13	38	29	2,458	246	2,695	2,702	21,507	111,636	918
CA	770	510	102	84	84	251	195	16,324	1,633	17,902	17,948	142,864	741,549	6,097
CO	151	100	20	16	16	49	38	3,204	321	3,514	3,523	28,043	145,561	1,197
CT	74	49	10	8	8	24	19	1,565	157	1,716	1,721	13,695	71,085	584
DE	23	15	3	2	2	7	6	488	49	535	537	4,271	22,168	182
DC	8	5	1	1	1	2	2	162	16	177	178	1,416	7,349	60
FL	424	281	56	46	46	138	107	8,978	898	9,846	9,871	78,571	407,828	3,353
GA	209	138	28	23	23	68	53	4,420	442	4,847	4,860	38,683	200,790	1,651
HI	26	17	3	3	3	9	7	559	56	613	615	4,891	25,388	209
ID	63	42	8	7	7	20	16	1,335	134	1,463	1,467	11,679	60,622	498
IL	431	286	57	47	47	140	109	9,132	914	10,014	10,040	79,916	414,809	3,411
IN	236	156	31	26	26	77	60	4,994	500	5,477	5,491	43,707	226,867	1,865
IA	267	177	35	29	29	87	68	5,655	566	6,201	6,217	49,489	256,877	2,112
KS	227	150	30	25	25	74	57	4,809	481	5,273	5,287	42,083	218,438	1,796
KY	109	72	14	12	12	36	28	2,315	232	2,539	2,546	20,262	105,174	865
LA	120	79	16	13	13	39	30	2,537	254	2,782	2,790	22,205	115,257	948

A = Premature mortality (adults, 30 and over)	**H = Asthma attacks (asthmatics, all ages)**
B = Chronic bronchitis (adults, 26 and over)	**I = Acute bronchitis (children, 8-12)**
C = Hospital admissions, pneumonia (adults, over 64)	**J = Lower respiratory symptoms (children, 7-14)**
D = Hospital admissions, chronic obstructive pulmonary disease (COPD) (adults, 64 and over)	**K = Upper respiratory symptoms (asthmatic children, 9-11)**
E = Hospital admissions, asthma (adults, 65 and younger)	**L = Work loss days (adults, 18-65)**
F = Hospital admissions, cardiovascular (adults, over 64)	**M = Minor restricted activity days (adults, 18-65)**
G = Emergency room visits, asthma (adults, 65 and younger)	

Table 6
State-By-State
Benefits of Regulating Nonroad Heavy-Duty Diesel Engines & Fuels
– Avoided Incidences (Cases/Year) –

State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
ME	28	19	4	3	3	9	7	601	60	659	660	5,257	27,285	224
MD	116	77	15	13	13	38	29	2,458	246	2,696	2,703	21,512	111,661	918
MA	193	128	26	21	21	63	49	4,099	410	4,495	4,506	35,870	186,186	1,531
MI	245	163	33	27	27	80	62	5,198	520	5,701	5,715	45,492	236,131	1,942
MN	268	178	36	29	29	87	68	5,678	568	6,227	6,243	49,693	257,938	2,121
MS	86	57	11	9	9	28	22	1,828	183	2,004	2,010	15,996	83,031	683
MO	215	143	29	23	23	70	55	4,565	457	5,006	5,019	39,952	207,375	1,705
MT	88	58	12	10	10	29	22	1,863	186	2,043	2,049	16,307	84,641	696
NE	177	118	24	19	19	58	45	3,759	376	4,123	4,133	32,900	170,770	1,404
NV	64	43	9	7	7	21	16	1,360	136	1,491	1,495	11,901	61,771	508
NH	27	18	4	3	3	9	7	565	57	619	621	4,943	25,658	211
NJ	184	122	24	20	20	60	47	3,906	391	4,283	4,294	34,182	177,424	1,459
NM	41	27	5	4	4	13	10	875	88	959	962	7,655	39,734	327
NY	376	249	50	41	41	122	95	7,963	797	8,732	8,755	69,686	361,710	2,974
NC	216	143	29	23	23	70	55	4,585	459	5,028	5,041	40,126	208,280	1,713
ND	184	122	24	20	20	60	47	3,902	390	4,279	4,290	34,146	177,240	1,457
OH	341	226	45	37	37	111	86	7,229	723	7,928	7,948	63,266	328,390	2,700
OK	134	89	18	15	15	44	34	2,834	284	3,108	3,116	24,802	128,736	1,059
OR	111	74	15	12	12	36	28	2,353	235	2,581	2,588	20,596	106,906	879
PA	248	164	33	27	27	81	63	5,259	526	5,768	5,783	46,029	238,916	1,964

A = Premature mortality (adults, 30 and over)	**H = Asthma attacks (asthmatics, all ages)**
B = Chronic bronchitis (adults, 26 and over)	**I = Acute bronchitis (children, 8-12)**
C = Hospital admissions, pneumonia (adults, over 64)	**J = Lower respiratory symptoms (children, 7-14)**
D = Hospital admissions, chronic obstructive pulmonary disease (COPD) (adults, 64 and over)	**K = Upper respiratory symptoms (asthmatic children, 9-11)**
E = Hospital admissions, asthma (adults, 65 and younger)	**L = Work loss days (adults, 18-65)**
F = Hospital admissions, cardiovascular (adults, over 64)	**M = Minor restricted activity days (adults, 18-65)**
G = Emergency room visits, asthma (adults, 65 and younger)	

Table 6
State-By-State
Benefits of Regulating Nonroad Heavy-Duty Diesel Engines & Fuels
– Avoided Incidences (Cases/Year) –

State	A	B	C	D	E	F	G	H	I	J	K	L	M	Monetary Millions
RI	17	11	2	2	2	6	4	362	36	397	398	3,170	16,456	135
SC	98	65	13	11	11	32	25	2,068	207	2,268	2,274	18,097	93,934	772
SD	134	89	18	15	15	44	34	2,844	285	3,119	3,127	24,887	129,179	1,062
TN	148	98	20	16	16	48	37	3,135	314	3,438	3,447	27,435	142,403	1,171
TX	584	387	77	63	63	190	148	12,377	1,238	13,573	13,609	108,320	562,243	4,623
UT	95	63	13	10	10	31	24	2,008	201	2,202	2,208	17,572	91,210	750
VT	16	10	2	2	2	5	4	336	34	368	369	2,937	15,243	125
VA	168	111	22	18	18	55	42	3,551	355	3,895	3,905	31,080	161,325	1,326
WA	171	113	23	19	19	56	43	3,624	363	3,974	3,984	31,711	164,601	1,353
WV	34	23	5	4	4	11	9	728	73	798	800	6,367	33,049	272
WI	185	123	25	20	20	60	47	3,930	393	4,310	4,321	34,398	178,544	1,468
WY	27	18	4	3	3	9	7	579	58	635	637	5,068	26,305	216
TOTAL	**8,522**	**5,647**	**1,129**	**924**	**924**	**2,772**	**2,156**	**180,598**	**18,070**	**198,052**	**198,565**	**1,580,512**	**8,203,798**	**$67,455**

A = Premature mortality (adults, 30 and over)	**H = Asthma attacks (asthmatics, all ages)**
B = Chronic bronchitis (adults, 26 and over)	**I = Acute bronchitis (children, 8-12)**
C = Hospital admissions, pneumonia (adults, over 64)	**J = Lower respiratory symptoms (children, 7-14)**
D = Hospital admissions, chronic obstructive pulmonary disease (COPD) (adults, 64 and over)	**K = Upper respiratory symptoms (asthmatic children, 9-11)**
E = Hospital admissions, asthma (adults, 65 and younger)	**L = Work loss days (adults, 18-65)**
F = Hospital admissions, cardiovascular (adults, over 64)	**M = Minor restricted activity days (adults, 18-65)**
G = Emergency room visits, asthma (adults, 65 and younger)	

D. Assessment of Human Welfare Benefits

PM and ozone have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for PM and ozone list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants. Individual effects, including changes in visibility, commercial crop and forest yields, and nitrogen deposition to estuaries, and how to quantify and monetize them, are described below.

Visibility Benefits

Changes in the level of ambient particulate matter resulting from the recommended nonroad HDD engine and fuel standards would improve the level of visibility in much of the U.S. Visibility directly affects our enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Grand Canyon. This section discusses the measurement of the economic benefits of visibility.

It is difficult to quantitatively define a visibility endpoint that can be used for valuation. Increases in PM concentrations cause increases in light extinction. Light extinction is a measure of how much the components of the atmosphere absorb light. More light absorption means that the clarity of visual images and visual range is reduced. Light absorption is a variable that can be accurately measured. Sisler created a unitless measure of visibility based directly on the degree of measured light absorption, called the deciview. Deciviews are standardized for a reference distance in such a way that one deciview corresponds to a change of about 10 percent in available light. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many circumstances." Air quality models were used to predict the change in visibility, measured in deciviews, of the areas affected by the 2007 onroad HDD engine and fuel rule and these were then adjusted to account for the larger PM impacts from the recommended nonroad HDD rule.

EPA considers benefits from two categories of visibility changes – residential visibility and recreational visibility. For both categories, economic benefits are believed to consist of both use values and non-use values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and bird watching. Non-use values are based on our beliefs that the environment should exist free of human-induced haze. Non-use values may be a more important component of value for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas, and also in recreational areas not listed as federal Class I areas. For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the U.S. are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland, et. al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Both utilize the contingent valuation (CV) method. Over the past decade, there has been a great deal of controversy over and significant development of both theoretical and empirical knowledge about how to conduct CV surveys. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas. This study serves as an essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland, et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (EPA-SAB-COUNCIL-ADV-00-002, 1999). Residential visibility benefits are, therefore, not included.

The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their willingness to pay to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the U.S. The value of visibility changes in the other Class I areas was inferred by transferring values of visibility changes at Class I areas in the study regions. However, these values are not as defensible.

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the 2007 onroad HDD engine and fuel rule. A general WTP equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from the 2007 onroad HDD engine and fuel rule. The method of developing calibrated WTP functions is based on the approach developed by Smith, et al. (1999). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1-percent increase in income is associated with a 0.9-percent increase in WTP for a given change in visibility.

Using the methodology outlined above, EPA estimated that the total WTP for the visibility improvements in California, Southwestern, and Southeastern Class I areas brought about by the 2007 onroad HDD engine and fuel rule is $3.3 billion. This value includes the value to households living in the same state as the Class I area, as well as values for all households in the U.S. living outside the state containing the Class I area; the value accounts for growth in real income.

Based upon our analysis, the potential visibility benefits from adoption of STAPPA/ALAPCO's recommended nonroad HDD engine and fuel rule would be $3.4 billion.

Other Effects

STAPPA/ALAPCO's recommended engine and fuel limits for nonroad diesels will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects. The share of nitrogen requirements provided by this deposition is small, and the marginal cost of providing this nitrogen from alternative sources is quite low. In some areas, agricultural lands suffer from nitrogen over-saturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen from PM represent additional agricultural benefits.

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (EPA, 1993).

On the other hand, there is evidence that forest ecosystems in some areas of the U.S. are nitrogen saturated (EPA, 1993). Once saturation is reached, adverse effects of additional nitrogen begin to occur, such as soil acidification that can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements, such as aluminum. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems.

Benefits from Reductions in Materials Damage

EPA's 2007 onroad HDD engine and fuel rule is expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. Criteria pollutants also have corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects. Similar benefits should result from implementation of STAPPA and ALAPCO's recommendations for nonroad HDD engine and fuels standards.

Previous EPA benefit analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, EPA determined that the existing data (based on consumer expenditures from the early 1970s) are too out of date to provide a reliable enough estimate of current household soiling damages (EPA-SAB-Council-ADV-003, 1998).

EPA was unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor was EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter

category in Sweden (Grosclaude and Soguel, 1994) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

Benefits from Reduced Ecosystem Damage

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_X that would result from the recommended nonroad HDD rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms, such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Reductions in nitrogen loadings are estimated for 12 eastern estuaries (including two on the Gulf Coast). These estimated reductions are described earlier in this report. Four of these estuaries have established consensus goals for reductions in annual nitrogen loads, indicating an intention of reaching these goals through implementation of controls on nitrogen sources. These four estuaries and their reduction goals are listed in Table 7.

Table 7 – Reduction Goals and 1998 Nitrogen Loads to Selected Eastern Estuaries (tons per year)

Estuary	Total Nitrogen Loadings	Nitrogen Loadings from Atmospheric Deposition	Overall Reduction Goal
Albemarle/Pamlico Sound	25,300	11,000	7,600
Chesapeake Bay	185,000	49,500	35,600
Long Island Sound	53,700	13,200	31,460
Tampa Bay	3,900	2,100	100

Source: EPA, 1998.

Estimated reductions in deposition of atmospheric nitrogen to these four estuaries are listed in Table 8, along with the percentage of the reduction goal accounted for by these reductions. These figures suggest that the reductions in nitrogen deposition resulting from the 2007 onroad HDD engine and fuel rule will provide significant progress towards meeting nitrogen reduction goals in several of these estuaries. NO_x reductions that would result from STAPPA and ALAPCO's recommended nonroad HDD engine and fuels standards would also contribute to achievement of these goals.

Table 8 – Estimated Annual Reductions in Nitrogen Loadings in Selected Eastern Estuaries for the 2007 Onroad HDD Engine and Fuel Rule in 2030 (tons per year)

Estuary	Change in Nitrogen Loadings	% of Estuary Nitrogen Reduction Goal
Albemarle/Pamlico Sound	1,804	23.7%
Chesapeake Bay	2,706	7.6%
Long Island Sound	1,067	3.4%
Tampa Bay[A]	385	over 100%

[A] Tampa Bay had a very low nitrogen loadings reduction goal. As such, the HD Engine/Diesel Fuel rule provides more reductions than are necessary to achieve the stated goal.

Direct C-R functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP-based measure of benefits depends on the availability of these C-R functions and on estimates of the value of environmental responses. Because neither appropriate C-R functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible. An alternative is to use an avoided cost approach to estimate the welfare effects of PM on estuarine ecosystems. The use of the avoided cost approach to establish the value of a reduction in nitrogen deposition is problematic if there is not a direct link between reductions in air deposited nitrogen and the abandonment of a costly regulatory program. However, there are currently no readily available alternatives to this approach.

Based on SAB advice, EPA used the avoided cost approach only to derive an alternative calculation of the value of reductions in atmospheric nitrogen loadings to estuaries (EPA-SAB-COUNCIL-ADV-00-002, 1999). The SAB believes that the avoided cost approach for nitrogen loadings is valid only if the state and local governments have established firm pollution reduction targets, and that displaced costs measured in the study represent measures not taken because of the Clean Air Act (EPA-SAB-COUNCIL-ADV-00-002, 1999). Because the nitrate reduction targets in the studied estuaries are not firm targets, and there is not assurance that planned measures would be undertaken in the absence of the Clean Air Act, EPA is currently unable to provide a meaningful primary estimate.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

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Appendix A

Health and Environmental Effects of Nonroad Diesel Emissions

Nonroad vehicles and engines emit significant quantities of particulate matter (PM), toxic air pollutants, hydrocarbons, nitrogen oxides (NO_x) and carbon monoxide (CO). As a result of the high growth in these engines and vehicles and their emissions, many areas across the U.S. are severely polluted. This appendix includes a discussion of some of the consequences of nonroad-related pollutants.

I. Particulate Matter

Particulate matter (PM) represents a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a wide range of sizes. Human-generated sources of particles include a variety of stationary and mobile sources. Particles may be emitted directly to the atmosphere or may be formed by transformations of gaseous emissions such as sulfur dioxide ($S0_2$) or NO_x. The major chemical and physical properties of PM vary greatly with time, region, meteorology, and source category, thus complicating the assessment of health and welfare effects as related to various indicators of particulate pollution. At elevated concentrations, PM can adversely affect human health, visibility, and materials. Components of PM (e.g., sulfuric or nitric acid) contribute to acid deposition.[1]

The key health effects categories associated with PM include premature death; aggravation of respiratory and cardiovascular disease, as indicated by increased hospital admissions and emergency room visits, school absences, work loss days, and restricted activity days; changes in lung function and increased respiratory symptoms; changes to lung tissues and structure; and altered respiratory defense mechanisms. Most of these effects have been consistently associated with ambient PM concentrations, which have been used as a measure of population exposure, in a large number of community epidemiological studies. Additional information and insights on these effects are provided by studies of animal toxicology and controlled human exposures to various constituents of PM conducted at higher than ambient concentrations. Although mechanisms by which particles cause effects are not well known, there is general agreement that the cardio-respiratory system is the major target of PM effects.

Individuals with respiratory disease (e.g., chronic obstructive pulmonary disease, acute bronchitis) and cardiovascular disease (e.g., ischemic heart disease) are at greater risk of premature mortality and hospitalization due to exposure to ambient PM.

Individuals with infectious respiratory disease (e.g., pneumonia) are at greater risk of premature mortality and morbidity (e.g., hospitalization, aggravation of respiratory symptoms) due to exposure to ambient PM. Also, exposure to PM may increase individuals' susceptibility to respiratory infections.

Elderly individuals are also at greater risk of premature mortality and hospitalization for cardiopulmonary problems due to exposure to ambient PM.

[1] U.S. EPA, 1996, Air Quality Criteria for Particulate Matter, EPA/600/P-95/001aF.

Children are at greater risk of increased respiratory symptoms and decreased lung function due to exposure to ambient PM.

Asthmatic individuals are at risk of experiencing exacerbated asthma-related symptoms and increased need for medical attention due to exposure to PM.

There are fundamental physical and chemical differences between fine and coarse fraction particles. The fine fraction contains acid aerosols, sulfates, nitrates, transition metals, diesel exhaust particles, and ultra-fine particles and the coarse fraction typically contains high mineral concentrations, silica, and resuspended dust. Exposure to coarse fraction particles is primarily associated with the aggravation of respiratory conditions, such as asthma. Fine particles are most closely associated with premature death, as well as hospital admissions and such health effects as aggravated cardiopulmonary disease.

The strongest evidence of ambient PM exposure health risks is derived from epidemiologic studies. Many such studies have shown statistically significant associations of ambient PM levels with a variety of human health endpoints in sensitive populations, including mortality, hospital admissions and emergency room visits, respiratory illness and symptoms, and physiologic changes in mechanical pulmonary function. The epidemiologic science points to fine PM as being more strongly associated with some health effects, such as premature mortality, than coarse fraction PM, which is associated with other health effects.

Time-series analyses strongly suggest a positive effect on daily mortality across the entire range of ambient PM levels. Relative risk (RR) estimates for daily mortality in relation to daily ambient PM concentration are consistently positive, and statistically significant (at $P \leq 0.05$), across a variety of statistical modeling approaches and methods of adjustment for effects of relevant covariates such as season, weather, and co-pollutants.

Within the body of evidence, there is considerable agreement among different studies that the elderly are particularly susceptible to effects from both short-term and long-term exposures to PM, especially if they have underlying respiratory or cardiac disease. These effects include increases in mortality and increases in hospital admissions. Children, especially those with respiratory diseases, may also be susceptible to pulmonary function decrements associated with exposure to PM or acid aerosols. Respiratory symptoms and reduced activity days have also been associated with PM exposures in children.

Numerous time-series analyses published in the late 1980s and early 1990s demonstrate significant positive associations between daily mortality or morbidity and 24-hour concentrations of ambient particles indexed by various measures (black smoke, total suspended particulate, PM_{10}, $PM_{2.5}$, etc.) in numerous U.S. metropolitan areas and in cities around the world (e.g., Athens, São Paulo, Santiago). These studies collectively suggest that PM alone or in combination with other commonly occurring air pollutants (e.g., SO_2) is associated with daily mortality and morbidity, the effect of PM appearing to be most consistent. In both the historic and recent studies, the association of PM exposure with mortality has been strongest in the elderly and for respiratory and cardiovascular causes of death.

Evidence from studies that looked at PM indicators other than PM_{10} also suggests that fine particles may be important contributors to the observed PM-health effects associations given the increased risks (of mortality, hospitalization, respiratory symptoms, etc.) associated with several different fine particle indicators (e.g., $PM_{2.5}$, $SO_4^{=}$, H^+). In particular, more recent reanalysis of the Harvard Six-City Study by Schwartz et al. examined the effects on daily mortality of 24-hour concentrations of fine particles ($PM_{2.5}$), inhalable particles ($PM_{15/10}$), or

coarse fraction particles ($PM_{15/10}$ minus $PM_{2.5}$) as exposure indices. Overall, these analyses suggest that, in general, the association between excess mortality and thoracic particles appears to be stronger for the fine fraction than for the coarse.

In addition to short-term exposure effects, mortality and morbidity effects associated with long-term exposure to PM air pollution have been assessed in cross-sectional studies and more recently, in prospective cohort studies. A number of older cross-sectional studies provided indications of increased mortality associated with chronic exposures to ambient PM (indexed mainly by TSP or sulfate measurements); however, unresolved questions regarding adequacy of statistical adjustments for other potentially important covariates tended to limit the degree of confidence that could be placed on such studies.

Some more recent studies used improved methods to examine relationships between chronic PM exposures indexed by different particle size indicators (PM_{15}, $PM_{2.5}$, PM_{15} to $PM_{2.5}$). These studies observed associations between increased risk of mortality/morbidity and chronic (annual average) exposures to PM_{10} or fine particle indicators in contemporary North American urban air sheds.

Recently, the Health Effects Institute (HEI) released the results of two major studies that are central to the debate over the adverse impact of particulate on human health. The first report is a re-analysis of two long-term community health studies: the Harvard Six-Cities Study (1993), and the American Cancer Society Study (1995). The second, called the National Morbidity, Mortality, and Air Pollution Study, is original research on hospitalization and deaths associated with air pollution in major U.S. cities.

A. The Particle Epidemiology Reanalysis Project

The Harvard Six City Study and the American Cancer Society Study examined the long-term effects of exposure to particulate air pollution on mortality. The Harvard Six-Cities Study by Dr. Douglas Dockery of the Harvard School of Public Health, and others, was published in December 1993 in the *New England Journal of Medicine*. Researchers followed the health of more than 8,000 people in six small cities that fell along a gradient of air pollution concentrations for a period of 14 to 16 years. As particle concentrations increased, there was an almost directly proportional increase in the death rate in the residents studied. Residents of the most polluted city in the study, Steubenville, Ohio, had a 26-percent increased risk of premature mortality, compared to the residents of the cleanest city studied, Portage, Wisconsin. According to the study's authors, this translates into a shortened life expectancy of one to two years for residents of Steubenville, compared to residents of Portage.

The March 1995 American Cancer Society Study, by Brigham Young University's Dr. Arden Pope, and others, found an association between chronic exposure to fine particle air pollution and premature death in a study group of over half a million people in 151 cities. Sulfate pollution was also associated with early death. The study reported strong associations between sulfates and fine particles and death by cardio-pulmonary causes.

These original studies used statistical techniques to adjust for age, and to control for the effects of smoking, diet, and occupational exposure.

Dr. Daniel Krewski of the University of Ottawa and his associates conducted a newly released reanalysis of these two studies for HEI. First, the HEI-funded researchers undertook a reanalysis of the original studies and a quality audit of the underlying data. Second,

researchers performed an extensive sensitivity analysis using alternative statistical methods, and considering the role of 20 potential confounders, such as other pollutants, climate, and socio-economic factors, on study results. This reanalysis by independent investigators validates the original studies.

B. The National Morbidity, Mortality and Air Pollution Study

HEI also commissioned the National Morbidity, Mortality and Air Pollution Study (NMMAPS), an original nationwide study of the short-term effects of air pollution on human health in the 90 largest American cities. A team of investigators led by Dr. Jonathan Samet and Dr. Scott Zeger of the Johns Hopkins University School of Hygiene and Public Health examined short-term increases in mortality rates caused by short-term elevations in particulate air pollution. Harvard School of Public Health researchers Dr. Douglas Dockery and Dr. Joel Schwartz studied effects on hospitalization in a subset of these cities. NMMAPS developed a new standardized methodology for examining pollution effects across many cities. Investigators developed state-of-the-art statistical techniques to examine the effects of multiple pollutants and the extent of life shortening.

Some critics have argued that short-term increases in the death rate are unimportant because the individuals affected are very frail and near death, even in the absence of air pollution. NMMAPS dispels this “harvesting” notion. NMMAPS investigators report that life is not shortened by a matter of days, but that life shortening is on the order of months or more.

Critics have also argued that other pollutants may be responsible for observed health effects. NMMAPS found strong evidence linking daily increases in particulate pollution to increases in death in the 20 largest U.S. cities. The association between particulate matter and mortality persisted even when other pollutants were included in the analysis.

In addition, NMMAPS found stable and robust associations between particulate pollution and increased hospital admissions for cardiovascular disease, pneumonia, and chronic obstructive pulmonary disease.

In an abstract prepared for the HEI Annual Conference, investigators concluded “these complementary analyses of mortality and morbidity provide new and strong evidence linking particulate air pollution at current levels to adverse health effects.”

C. Visibility/Regional Haze

The presence of tiny particles in the air causes light to be scattered or absorbed, thereby reducing visibility. This phenomenon, known as visibility impairment or regional haze, is a complex problem that relates to natural conditions and also several pollutants. Visibility in our national parks and monuments, and many urban areas of the country, continues to be obscured by regional and local haze.

The principle cause of visibility impairment is fine particles, primarily sulfates, but also nitrates, organics, and elemental carbon and crustal matter. Particles between 0.1 and one micrometer in size are most effective at scattering light; these same particles are also of greatest concern for human health. Of the pollutant gases, only NO_2 absorbs significant amounts of light; it is partly responsible for the brownish cast of polluted skies. However, it is responsible for less than 10 percent of visibility reduction.

In the eastern U.S., reduced visibility is mainly attributable to secondary particles, particularly those less than a few micrometers in diameter. Based on data collected by the Interagency Monitoring of Protected Visual Environments (IMPROVE) network for visibility monitoring, sulfate particles account for about 50-70 percent of annual average light extinction in eastern locations. Sulfate plays a particularly significant role in the humid summer months, most notably in the Appalachian, northeast, and mid-south regions. Nitrates, organic carbon, and elemental carbon each account for between 10-15 percent of total light extinction in most eastern locations. Rural areas in the eastern U.S. generally have higher levels of impairment than most remote sites in the western U.S., generally due to the eastern U.S.'s higher levels of man-made pollution, higher estimated background levels of fine particles, and higher average relative humidity levels.

The relative contribution of individual pollutants to visibility impairment varies geographically. While secondary particles still dominate in the West, direct particulate emissions from sources such as wood smoke contribute a larger percentage of the total particulate load than in the East. In the rural western U.S., sulfates also play a significant role, accounting for about 25-40 percent of estimated total light extinction in most regions. In some areas, such as the Cascades region of Oregon, sulfates are estimated to account for over 50 percent of annual average light extinction. Organic carbon typically is estimated to be responsible for 15-35 percent of total light extinction in the rural western U.S. and elemental carbon (absorption) accounts for about 15–25 percent, so the total carbonaceous contribution is between 30 and 60 percent. Soil dust (coarse PM) accounts for about 10–20 percent. Nitrates typically account for less than 10 percent of visibility impairment.[2]

Visibility is greatly affected by ambient $PM_{2.5}$ concentrations, with concentrations below the National Ambient Air Quality Standard (NAAQS) being sufficient to impair visibility. Black elemental carbon particles are a dominant light-adsorbing species in the atmosphere[3], and a major component of diesel exhaust.

II. Air Toxics from Engines and Vehicles

In addition to contributing to the health and welfare problems associated with exceedances of the air quality standards for ozone and PM_{10}, emissions from diesel and gasoline vehicles include a number of air pollutants that increase the risk of cancer or have other negative health effects. These air pollutants include benzene, formaldehyde, acetaldehyde, 1,3-butadiene, and diesel particulate matter. All of these compounds are products of combustion; benzene is also found in non-exhaust emissions from gasoline-fueled vehicles.

There are hundreds of different compounds and elements that are known to be emitted from passenger cars, on-highway trucks, and various nonroad equipment. EPA has recently proposed a methodology for identifying which of these compounds and elements are toxic and has developed a preliminary mobile source air toxics (MSAT) list.

In identifying compounds for the MSAT list, EPA first compiled all available recent studies that speciated emissions from motor vehicles and their fuels. To do this, EPA reviewed a number of databases that contain information on the various species of compounds emitted from motor vehicles and their fuels. It identified recent (less than 10 years old) speciation profiles for emissions from light-duty gas vehicles (LDGV), heavy-duty diesel vehicles (HDDV), heavy-duty

2 "National Air Quality and Emissions Trends Report, 1996," EPA Document Number 454/R-97-013.
3 Source Contributions to Atmospheric Fine Carbon Particle Concentrations, Gray and Cass, Atmospheric Environment, Vol. 32, No. 22, pp. 3805-3825 (1998).

gasoline vehicles (HDGV), gasoline-powered nonroad engines, and turbine engine aircraft. Forty-four speciation studies were found that met this age criteria. All of these speciation profiles attempt to accomplish more or less the same objective: separating and identifying the compounds that comprise the hydrocarbon portion and particulate phase of mobile source emissions.

EPA then looked to its Integrated Risk Information System (IRIS), which is a database of scientific information that contains the Agency's consensus scientific positions on potential adverse health effects that may result from lifetime (chronic) or short-term (acute) exposure to various substances found in the environment.[4] IRIS currently provides health effects information on over 500 specific chemical compounds. The information contained in the IRIS database includes an EPA finding for each compound for which: 1) there is a health hazard, either cancer or noncancer, associated with exposure to the compound, 2) current data indicate that the compound is noncarcinogenic, or 3) the data is insufficient to determine if the compound is a hazard.

IRIS contains chemical-specific summaries of qualitative and quantitative health information. IRIS information may include the reference dose (RfD) for noncancer health effects resulting from oral exposure, the reference concentration (RfC) for noncancer health effects resulting from inhalation exposure, and the carcinogen assessment for both oral and inhalation exposure. Combined with information on specific exposure situations, the summary health hazard information in IRIS may be used in evaluating potential public health risks from environmental contaminants.

Before a substance is listed on the IRIS database, it goes through a thorough scientific evaluation. This consensus and review process, managed by EPA's Office of Research and Development, consists of 1) an annual *Federal Register* announcement of the IRIS agenda and a call for scientific information from the public on the selected chemical substances, 2) a search of the current literature, 3) development of health assessment and draft IRIS summaries, 4) internal EPA peer review, 5) external peer review, 6) Agency consensus review and management approval within EPA, 7) preparation of final IRIS summaries and supporting documents, and 8) entry of summaries and supporting documents into the IRIS database.

By comparing the list of compounds in IRIS to the motor vehicle emissions identified in the speciation studies, EPA identified 21 MSAT, as listed in the table below. Each of these pollutants is a known, probable, or possible human carcinogen (Group A, B or C) or was considered by the Agency to pose a risk of adverse noncancer health effects.

Proposed List of Mobile Source Air Toxics

Acetaldehyde	Diesel Exhaust	MTBE***
Acrolein	Ethylbenzene	Naphthalene
Arsenic Compounds*	Formaldehyde	Nickel Compounds*
Benzene	n–Hexane	POM****
1,3-Butadiene	Lead Compounds*	Styrene
Chromium Compounds*	Manganese Compounds*	Toluene
Dioxin/Furans**	Mercury Compounds*	Xylene

4 EPA IRIS Database, http://www.epa.gov/ngispgm3/iris/index.html

Notes on Above Table Listing Proposed Mobile Source Air Toxics:
*Although the different species of the same metal differ in their toxicity, the onroad mobile source inventory contains emissions estimates for total compounds of the metal identified in particulate speciation profiles (i.e., the sum of all forms).
**This entry refers to two large groups of chlorinated compounds. In assessing their cancer risks, their quantitative potencies are usually derived from that of the most toxic, 2,3,7,8-tetrachlorodibenzodioxin.
***MTBE is listed due to its potential inhalation air toxics effects and not due to ingestion exposure associated with drinking water contamination.
****Polycyclic Organic Matter includes organic compounds with more than one benzene ring and a boiling point greater than or equal to 100 degrees centigrade. A group of seven polynuclear aromatic hydrocarbons, which have been identified by EPA as probable human carcinogens (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, 7,12-dimethylbenz(a)anthracene, and indeno(1,2,3-cd)pyrene) are sometimes used as a surrogate for the larger group of POM compounds.

A. Diesel Health Assessment

Diesel emissions deserve a special discussion because they are dominant source of mobile source cancer risk. EPA determined a reference concentration in 1993 to minimize noncancer health effects resulting from exposure to diesel exhaust. EPA has summarized available information to characterize the cancer and noncancer health effects from exposure to diesel exhaust emissions in the draft *Health Assessment Document for Diesel Emissions*. The key components of the current draft Assessment are: 1) information about the chemical components of diesel exhaust and how they can influence toxicity, 2) the cancer and noncancer health effects of concern for humans, and 3) the possible impact or risk to an exposed human population.

Based on information provided in the draft Assessment[5] and other sources of information, EPA has concluded that diesel particulate is a probable human carcinogen. The most compelling information to suggest a carcinogenic hazard is the consistent association that has been observed between increased lung cancer and diesel exhaust exposure in certain occupationally exposed workers working in the presence of diesel engines. Approximately 30 individual epidemiological studies show increased lung cancer risks of 20 to 89 percent within the study populations, depending on the study. Analytical results of pooling the positive study results show that, on average, the lung cancer risks were increased by 33 to 47 percent. The magnitude of the pooled risk increases is not precise owing to uncertainties in the individual studies, the most important of which is a continuing concern about whether smoking effects have been accounted for adequately. While not all studies have demonstrated an increased risk (six of 34 epidemiological studies summarized by HEI[6] reported relative risks less than 1.0), the fact that an increased risk has been consistently noted in the majority of epidemiological studies strongly supports the determination that exposure to diesel exhaust is likely to pose a carcinogenic hazard to humans.

Additional evidence for treating diesel exhaust as a carcinogen at ambient levels of exposure is provided by the observation of the presence of small quantities of many mutagenic and some carcinogenic compounds in the diesel exhaust. A carcinogenic response believed to be caused by such agents is assumed not to have a threshold unless there is direct evidence to the

5 EPA is revising this draft document in response to comments by the Clean Air Scientific Advisory Committee.
6 Health Effects Institute (1995) Diesel Exhaust: A Critical Analysis of Emissions, Exposure, and Health Effects pp. 253-292. April 1995.

contrary. In addition, there is evidence that at least some of the organic compounds associated with diesel particulate matter are extracted by lung fluids (i.e., are bioavailable) and, therefore, are available in some quantity to the lungs, as well as entering the bloodstream and being transported to other sites in the body.

The concern for the carcinogenic health hazard resulting from diesel exhaust exposures is widespread and several national and international agencies have designated diesel exhaust or diesel particulate matter as a "potential" or "probable" human carcinogen.[7], [8] The International Agency for Research on Cancer (IARC) in the late 1980s concluded that diesel exhaust is a "probable" human carcinogen.[9] Based on IARC findings, California identified diesel exhaust in 1990 as a chemical known to the state to cause cancer and, after an extensive review in 1998, listed diesel exhaust as a toxic air contaminant.[10] The National Institute for Occupational Safety and Health has classified diesel exhaust a "potential occupational carcinogen." The World Health Organization recommends that "urgent efforts should be made to reduce [diesel engine] emissions, specifically of particulates, by changing exhaust train techniques, engine design and fuel composition."

Just recently, the National Institute For Environmental Health Sciences, in its 9th National Toxicology Report on Carcinogens, added diesel particulate to its list of substances that are reasonably anticipated to be human carcinogens.

Another aspect of diesel particulate that is cause for concern is its size. Approximately 80-95 percent of diesel particle mass is in the size range of 0.05-1.0 micron with a mean particle diameter of about 0.2 microns. These fine particles have a very large surface area per gram of mass, which makes them excellent carriers of adsorbed inorganic and organic compounds that can effectively reach the lowest airways of the lung. Approximately 50-90 percent of the number of particles in diesel exhaust are in the ultra-fine size range of 0.005-0.05 microns, averaging about 0.02 microns. While accounting for the majority of the number of particles, ultra-fine diesel particulate matter accounts for 1-20 percent of the mass of diesel PM.

B. Diesel Exhaust or Diesel Particulate Matter

Diesel exhaust includes gaseous and particulate components. Gaseous components of diesel exhaust include organic compounds, nitrogen-containing compounds, sulfur compounds, carbon monoxide, carbon dioxide, water vapor, and excess air (nitrogen and oxygen). Among these gaseous organic compounds are benzene (a known human carcinogen), formaldehyde, acetaldehyde, and 1,3-butadiene (possible or probable human carcinogens). Particulate components include many organic compounds that are mutagenic, as well as several trace metals (including chromium, manganese, mercury, and nickel) that may have general toxicological significance (depending on the specific species). In addition, small amounts of dioxins have been measured in diesel exhaust, some of which may partition to the particle phase.

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7 National Institute for Occupational Safety and Health (1988) Carcinogenic effects of exposure to diesel exhaust. NIOSH Current Intelligence Bulletin 50. DHHS (NIOSH) Publication No. 88-116. Centers for Disease Control, Atlanta, GA.

8 World Health Organization (1996) Diesel fuel and exhaust emissions: International program on chemical safety. World Health Organization, Geneva, Switzerland.

9 International Agency for Research on Cancer (1989) Diesel and gasoline engine exhausts and some nitroarenes, Vol. 46. Monographs on the evaluation of carcinogenic risks to humans. World Heath Organization, International Agency for Research on Cancer, Lyon, France.

10 California EPA (1998) Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Appendix III Part A: Exposure Assessment. California Environmental Protection Agency. California Air Resources Board April 22, 1998.

Because diesel exhaust is a mixture of particles and gases, the choice of a measure of exposure (i.e., dosimeter) is important. EPA believes that exposure to whole diesel exhaust is best described, as many researchers have done over the years, by diesel particulate concentrations expressed in units of mass concentration (e.g., $\mu g/m^3$). The choice of this dosimeter implies that the contribution of the gaseous components and diesel particulate constituents to toxicity are related by diesel particulate mass. This assumption is consistent with historic practice, but can only be validated when there is a better understanding of the toxicological mode of action for diesel exhaust.

While some of the cancer and noncancer hazards may be associated with exposure to the gaseous component of diesel exhaust, studies suggest that the particulate component plays a substantial role in carcinogenicity and noncancer effects. Investigations show that diesel particles (the elemental carbon core plus the adsorbed organics) induce lung cancer at high doses and that the particles, independent of the gaseous compounds, elicit an animal lung cancer response. The presence of non-diesel elemental carbon particles, as well as the organic-laden diesel particles, correlates with an adverse inflammatory effect in the respiratory system of animals. Additional evidence suggesting the importance of the role of particulate matter in diesel exhaust includes the observation that the extractable particle organics collectively produce cancer and adverse mutagenic toxicity in laboratory experiments.

Given the available information, EPA has proposed to list diesel exhaust as a mobile source air toxic pollutant.

Diesel particulate matter is mainly attributable to the incomplete combustion of fuel hydrocarbons, as well as engine oil and other fuel components, such as sulfur. Diesel exhaust particulates are part of ambient $PM_{2.5}$, since diesel engines are used to power numerous types of equipment in many places. Some geographic areas may have higher diesel particulate loading because of the number of engines that exhaust into the ambient air. While diesel particulate matter contributes to ambient levels of $PM_{2.5}$, the high content of elemental carbon with the adsorbed organic compounds and the high number of ultra-fine particles (organic carbon and sulfate) in diesel exhaust distinguish it from other noncombustion sources of $PM_{2.5}$. In addition, diesel particulate matter from mobile source diesel engines is emitted into the breathing zone of humans and, thus, has a greater potential for human exposure (per kg of emissions), compared to other combustion particulates emitted out of stacks.

Overall, information suggests that the diesel particle may be playing a key role(s) in contributing to the chronic noncancer and carcinogenicity hazards associated with exposure to diesel exhaust: both as a mechanism of delivery for many of the organics and trace metals into the respiratory system, and as a physical irritant in and of itself. Given the available information, it is a reasonable and prudent step to protect public health by proposing regulations on the particulate phase of diesel exhaust.

<u>The MATES Study</u>

The Multiple Air Toxics Exposure Study (MATES-II) is a landmark urban toxics monitoring and evaluation study conducted for the South Coast Air Basin (Basin) in California. It represents one of the most comprehensive air toxics programs ever conducted in an urban environment. The study consists of several elements: a comprehensive monitoring program, an updated emissions inventory of toxic air contaminants, and a modeling effort to fully characterize Basin risk.

In the monitoring program, over 30 air pollutants were measured – as listed below – including both gas and particulates.

Pollutants Measured in MATES-II

Chemical Name	Chemical Name
Benzene	Formaldehyde
1,3 Butadiene	Acetaldehyde
Dichlorobenzene (ortho- & para)	Acetone
Vinyl Chloride	Arsenic
Ethyl Benzene	Chromium
Toluene	Lead
Xylene (m-, p-, o-)	Nickel
Styrene	Cobalt
Carbon Tetrachloride	Copper
Chloroform	Manganese
Dichloroethane [1,1]	Phosphorous
Dichloroethylene [1,1]	Selenium
Methylene Chloride	Silica
Perchloroethylene	Silver
Trichloroethylene	Zinc
Chloromethane	PAHs
Elemental Carbon	Organic Carbon

When “carcinogenic risk” is discussed, it typically refers to the probability of a person contracting cancer over the course of a lifetime if exposed to the source of cancer-causing compounds for 70 years. In other words, a cancer risk of 100 in a million at a location means that the individuals staying at that location for 70 years have a 100 in a million chance of contracting cancer. If 10,000 people live at that location, then the cancer burden for this population will be one (the population multiplied by the cancer risk). This means that one of the 10,000 people staying at the location for 70 years are expected to contract cancer.

The key result of the MATES-II study was the conclusion that the average carcinogenic risk in the Basin is about 1,400 per million people. Mobile sources (e.g., cars, trucks, trains, ships,

aircraft) are the greatest contributor. About 70 percent of all risk is attributed to diesel particulate emissions; about 20 percent to other toxics associated with mobile sources (including benzene, butadiene, and formaldehyde); and about 10 percent of all risk is attributed to stationary sources (which include industries and other certain businesses, such as dry cleaners and chrome plating operations).

The average carcinogenic risk of 1,400 per million is based on a range from about 1,120 in a million to about 1,740 in a million among the ten sites.

III. Photochemical Oxidants (Ozone)

Ground-level ozone, the main ingredient in smog, is formed by complex chemical reactions of volatile organic compounds (VOC) and NO_x in the presence of heat and sunlight. Ozone forms readily in the lower atmosphere, usually during hot summer weather. VOCs are emitted from a variety of sources, including motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. VOCs also are emitted by natural sources such as vegetation. NO_x is emitted largely from motor vehicles, nonroad equipment, power plants, and other sources of combustion.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NO_x, VOC, heat, and sunlight.[11] As a result, differences in NO_x and VOC emissions and weather patterns contribute to daily, seasonal, yearly, and geographic differences in ozone concentrations. Many of the chemical reactions that are part of the ozone-forming cycle are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and produce more ozone than typically would occur on a single high-temperature day.[12] Further complicating matters is the fact that ozone can also be transported into an area from pollution sources found hundreds of miles upwind, resulting in elevated ozone levels even in areas with low VOC or NO_x emissions.

Based on a large number of recent studies, EPA has identified several key health effects that result when people are exposed to levels of ozone found today in many areas.[13, 14]

A. Short-Term Exposures to Ozone

A large body of evidence shows that ozone can cause harmful respiratory effects including chest pain, coughing, and shortness of breath, which affect people with compromised respiratory systems most severely. When inhaled, ozone can cause acute respiratory problems; aggravate asthma; cause significant temporary decreases in lung function of 15 to over 20 percent in some healthy adults; cause inflammation of lung tissue; may increase hospital admissions and emergency room visits; and impair the body's immune system defenses, making people more susceptible to respiratory illnesses. Children and outdoor workers are likely to be exposed to elevated ambient levels of ozone during exercise and, therefore, are at greater risk of experiencing adverse health effects.

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11 Carbon monoxide also participates in the production of ozone, albeit at a much slower rate than most VOC and NOx compounds.

12 There is a growing concern that climate modification resulting from the increased buildup of greenhouse gases such as carbon dioxide may increase the amount of ozone produced from a given amount of NO_x and VOCs.

13 U.S. EPA, 1996, Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-96-007.

14 U.S. EPA, 1996, Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA/600/P-93/004aF.

Short-term exposures (1 to 3 hours) to high ambient ozone concentrations have been linked to increased hospital admissions and emergency room visits for respiratory problems. For example, studies conducted in the northeastern U.S. and Canada show that ozone air pollution is associated with 10-20 percent of all of the summertime respiratory-related hospital admissions. Repeated exposure to ozone can make people more susceptible to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma. Exposure to ozone can cause repeated inflammation of the lung, impairment of lung defense mechanisms, and irreversible changes in lung structure, which could lead to premature aging of the lungs and/or chronic respiratory illnesses, such as emphysema, chronic bronchitis and chronic asthma.

Children are most at risk from ozone exposure because during the summer when ozone levels are highest, they typically are active outside, playing and exercising. For example, summer camp studies in the eastern U.S. and southeastern Canada have reported significant reductions in lung function in children who are active outdoors. Further, children are more at risk than adults from ozone exposure because their respiratory systems are still developing. Adults who are outdoors and moderately active during the summer months, such as construction workers and other outdoor workers, are also among those most at risk. These individuals, as well as people with respiratory illnesses such as asthma, especially asthmatic children, can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to ozone during periods of moderate exertion.

Evidence also exists of a possible relationship between daily increases in ozone levels and increases in daily mortality levels. While the magnitude of this relationship is still too uncertain to allow for direct quantification, the full body of evidence indicates a likely positive relationship between ozone exposure and premature mortality.

B. Prolonged and Repeated Exposures to Low Levels of Ozone

A large body of scientific literature regarding health and welfare effects of ozone has associated health effects with certain patterns of ozone exposures that do not include any hourly ozone concentration above the 0.12 parts per million (ppm) level of the one-hour NAAQS. The science indicates that there are health effects attributable to prolonged and repeated exposures to lower ozone concentrations. Studies of six- to eight-hour exposures showed health effects from prolonged and repeated exposures at moderate levels of exertion to ozone concentrations as low as 0.08 ppm.

Studies of acute health effects have shown transient pulmonary function responses, transient respiratory symptoms, effects on exercise performance, increased airway responsiveness, increased susceptibility to respiratory infection, increased hospital and emergency room visits, and transient pulmonary respiratory inflammation. Such acute health effects have been observed following prolonged exposures at moderate levels of exertion at concentrations of ozone well below the current national standard of 0.12 ppm. The effects are more pronounced at concentrations above 0.09 ppm, affecting more subjects or having a greater effect on a given subject in terms of functional changes or symptoms.

With regard to chronic health effects, the collective data have many ambiguities, but provide suggestive evidence of chronic effects in humans. There is a biologically plausible basis for considering the possibility that repeated inflammation associated with exposure to ozone over a lifetime, as can occur with prolonged exposure to moderate ozone levels below peak levels,

may result in sufficient damage to respiratory tissue that individuals later in life may experience a reduced quality of life, although such relationships remain highly uncertain.

In addition to the effects on human health, ozone is known to adversely affect the environment in many ways. These effects include reduced yield for commodity crops, fruits and vegetables, and commercial forests; ecosystem and vegetation effects in such areas as National Parks (Class I areas); damage to urban grass, flowers, shrubs, and trees; reduced yield in tree seedlings and non-commercial forests; increased susceptibility of plants to pests; materials damage; and visibility.

In addition to their contribution to ozone levels, VOC emissions contain toxic air pollutants that may have a significant effect on the public health.

IV. Nitrogen Oxides

As a class of compounds, the oxides of nitrogen are involved in a host of environmental concerns that have an adverse impact on human health and welfare. Nitrogen dioxide (NO_2) has been linked with increased susceptibility to respiratory infection, increased airway resistance in asthmatics, and decreased pulmonary function.[15], [16] It has been shown that even short-term exposures to NO_2 have resulted in a wide-ranging group of respiratory problems in school children – cough, runny nose, and sore throat are among the most common.[17] Further, in France, in an ingenious experiment, Dr. Orehek has shown that asthmatics are especially sensitive to even one-hour exposures.[18] A small group of asthmatics was initially exposed to carbachol, a bronchoconstrictor representative of urban pollen, and then to NO_2; adverse effects, such as increased airway resistance, were experienced by some of the individuals at levels as low as 0.1 parts per million for one hour.

NO_x also is a contributor to acid deposition, which can damage trees at high elevations and increases the acidity of lakes and streams, which can severely damage aquatic life. Finally, NO_x emissions can contribute to increased levels of particulate matter by changing into nitric acid in the atmosphere and forming particulate nitrate.

A. Eutrophication and Nitrification

NO_x also result in nitrogen deposition into sensitive nitrogen-saturated coastal estuaries and ecosystems, causing increased growth of algae and other plants.[19] Long-term monitoring in the U.S., Europe, and other developed regions of the world shows a substantial rise in nitrogen levels in surface waters, which are highly correlated with human-generated inputs of nitrogen to their watersheds. Fertilizers and atmospheric deposition dominate these nitrogen inputs.

Human activity can increase the flow of nutrients into those waters and result in excess algae and plant growth. This increased growth can cause numerous adverse ecological effects and

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15 U.S.EPA, 1993, Air Quality Criteria for Oxides of Nitrogen, EPA/600/8-91/049aF.

16 U.S. EPA, 1995, Review of National Ambient Air Quality Standards for Nitrogen Dioxide, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-95-005

17 "The University Of Akron Study on Air Pollution and Human Health Effects", Mostardi et al, Archives of Environmental Health, September/October 1981.

18 "Effect of Short-Term, Low-Level Nitrogen Dioxide Exposure on Bronchial Sensitivity of Asthmatic Patients", Orehek, et. al., The Journal of Clinical Investigations, Volume 57, February 1976.

19 Vitousek, Pert M., John Aber, Robert W. Howarth, Gene E. Likens, et al. 1997. Human Alteration of the Global Nitrogen Cycle: Causes and Consequences. *Issues in Ecology*. Published by Ecological Society of America, Number 1, Spring 1997.

economic impacts, including nuisance algal blooms, dieback of underwater plants due to reduced light penetration, and toxic plankton blooms. Algal and plankton blooms can also reduce the level of dissolved oxygen, which can also adversely affect fish and shellfish populations. This problem is of particular concern in coastal areas with poor or stratified circulation patterns. In such areas, the "overproduced" algae tends to sink to the bottom and decay, using all or most of the available oxygen and thereby reducing or eliminating populations of bottom-feeder fish and shellfish, distorting the normal population balance between different aquatic organisms, and, in extreme cases, causing dramatic fish kills.

Collectively, these effects are referred to as eutrophication, which the National Research Council identified as the most serious pollution problem facing the estuarine waters of the U.S. Nitrogen is the primary cause of eutrophication in most coastal waters and estuaries.[20] On the New England coast, for example, the number of red and brown tides and shellfish problems from nuisance and toxic plankton blooms have increased over the past two decades, a development thought to be linked to increased nitrogen loadings in coastal waters.

B. Acid Deposition

Acid deposition, or acid rain as it is commonly known, occurs when SO_2 and NO_x react in the atmosphere with water, oxygen, and oxidants to form various acidic compounds that later fall to earth in the form of precipitation or dry deposition of acidic particles.[21] It contributes to damage of trees at high elevations and in extreme cases may cause lakes and streams to become so acidic that they cannot support aquatic life. In addition, acid deposition accelerates the decay of building materials and paints, including irreplaceable buildings, statues, and sculptures that are part of our nation's cultural heritage.

Acid deposition primarily affects bodies of water that rest atop soil with a limited ability to neutralize acidic compounds. The National Surface Water Survey investigated the effects of acidic deposition in over 1,000 lakes larger than 10 acres and in thousands of miles of streams. It found that acid deposition was the primary cause of acidity in 75 percent of the acidic lakes and about 50 percent of the acidic streams. Acid deposition also has been implicated in the degradation of high-elevation spruce forests that populate the ridges of the Appalachian Mountains from Maine to Georgia. This area includes national parks such as the Shenandoah and Great Smoky Mountain National Parks.

V. Carbon Monoxide

Carbon monoxide (CO) – an odorless, invisible gas created when fuels containing carbon are burned incompletely – poses a serious threat to human health. Persons afflicted with heart disease and fetuses are especially at risk. Because the affinity of hemoglobin in the blood is 200 times greater for carbon monoxide than for oxygen, carbon monoxide hinders oxygen transport from blood into tissues. Therefore, more blood must be pumped to deliver the same amount of oxygen. Numerous studies in humans and animals have demonstrated that those individuals with weak hearts are placed under additional strain by the presence of excess CO in the blood. In particular, clinical health studies have shown a decrease in time to onset of angina pain in

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20 Much of this information was taken from the following EPA document: *Deposition of Air Pollutants to the Great Waters-Second Report to Congress*, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011.

21 Much of the information in this subsection was excerpted from the EPA document, *Human Health Benefits from Sulfate Reduction*, written under Title IV of the 1990 Clean Air Act Amendments, U.S. EPA, Office of Air and Radiation, Acid Rain Division, Washington, DC 20460, November 1995.

those individuals suffering from angina pectoris and exposed to elevated levels of ambient CO.[22]

Healthy individuals also are affected, but only at higher levels. Exposure to elevated CO levels is associated with impairment of visual perception, work capacity, manual dexterity, learning ability, and performance of complex tasks.[23]

22 "Effect of Carbon Monoxide On Exercise Performance In Chronic Obstructive pulmonary Disease", Aronow, et. al., Am. J. Med., 1977, "Health Effects of Exposure To Low Levels of Regulated Air Pollutants, A Critical Review", Ferris, Journal of The Air Pollution Control Association, May 1978

23 "Air Quality Criteria For Carbon Monoxide", U.S. Environmental Protection Agency, Second External Review Draft, October 1999.