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REGULATORY STUDIES PROGRAM

Public Interest Comment on EPA's Proposed Nonroad Diesel Engine and Fuel Standards[1]

The Regulatory Studies Program (RSP) of the Mercatus Center at George Mason University is dedicated to advancing knowledge of the impact of regulation on society. As part of its mission, RSP conducts careful and independent analyses employing contemporary economic scholarship to assess rulemaking proposals from the perspective of the public interest. Thus, this comment on EPA's "Proposed Nonroad Diesel Engine and Fuel Standards" does not represent the views of any particular affected party or special interest group, but is designed to evaluate the effect of the Agency's proposals on overall consumer welfare.

I. Introduction and Background

Nonroad mobile engines include hundreds of different engines and types of equipment, with sizes ranging from a few horsepower up to at least 3,000 horsepower. EPA published a proposed rule for control of emissions from nonroad diesel engines and fuel (hereafter the "Proposed Rule") in the Federal Register on May 23, 2003, along with a 1,300 page regulatory impact analysis (hereafter the "Nonroad RIA").[2] The Proposed Rule discussed in this Public Interest Comment applies to all diesel nonroad mobile equipment except locomotives and marine engines.[3]

According to EPA, on a nationwide basis, diesel engines contribute 12 percent of all nitrogen oxide (NOx) emissions and 44 percent of all diesel particulate emissions.

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[2] EPA, "Control of Emissions of Air Pollution from Nonroad Diesel Engines and Fuel; Proposed Rule," *Federal Register*, May 23, 2003, p. 28328-28603, http://www.epa.gov/otaq/url-fr/fr23my03p.pdf, EPA, *Draft Regulatory Impact Analysis: Control of Emissions from Nonroad Diesel Engines* (Washington, DC: April 2003), http://www.epa.gov/otaq/diesel.htm.

[3] For the sake of brevity and simplicity, we will hereafter refer to the engines covered by this rule as "nonroad engines," but this should be understood to refer only to nonroad mobile equipment running on diesel fuel, and to exclude locomotives, marine vessels, and aircraft.

Nonroad diesel engines also emit volatile organic compounds (VOCs) and carbon monoxide (CO), but represent less than one percent of the national inventory of these compounds. EPA has already promulgated emission control requirements for nonroad diesel engines, known as Tier 1, Tier 2, and Tier 3 requirements, that phase in between 1996 and 2008. The Proposed Rule would create Tier 4 requirements phasing in between 2008 and 2014, requiring more than a 90 percent reduction in NOx and PM emissions below the Tier 3 requirements, and would also reduce allowable sulfur in nonroad diesel fuel from 5000 parts per million (ppm) to 500 ppm in 2007 and 15 ppm in 2010. The requirements of the Proposed Rule are similar to requirements for on-road diesel trucks, but the Proposed Rule would phase in those requirements a few years later than for on-road diesel vehicles.

EPA is proposing this rule because it believes additional controls on nonroad diesel engine emissions and fuel are necessary for many areas of the country to attain federal ozone and PM health standards, and to reduce risk of cancer and other health effects from hazardous air pollutants (HAPs) emitted by diesel engines, including diesel particulate matter (DPM) and benzene.

This Public Interest Comment summarizes the requirements of the Proposed Rule, assesses the validity of EPA's health, air quality and economic analyses, and makes recommendations on how EPA should modify the Proposed Rule to ensure net benefits to Americans' health and welfare.

II. Summary of the Proposed Rule

A. Vehicles and Equipment Affected

The Proposed Rule focuses on nonroad diesel equipment that is either self-propelled, such as a tractor, and diesel equipment that is portable but stationary when in use, such as a generator. Diesel equipment that is always stationary is not included. The rule also applies only to land-based equipment, and excludes locomotives and marine vessels. For brevity, we will refer to the engines addressed by the Proposed Rule as "nonroad" engines or equipment.

Nonroad diesel equipment includes an enormous range of power ratings and applications, and hundreds of different types of equipment and dozens of manufacturers. EPA estimates there are currently 10 million nonroad diesel engines operating in the United States. Power can range from a few horsepower up to more than 3,000 horsepower. The smallest equipment includes generators, mowers, trenchers, small tractors, and pumps. Medium and heavy equipment includes agricultural tractors, forklifts, backhoes, and bulldozers. The largest equipment includes large generators, crawler tractors, and heavy off-road trucks. Tractors and generators account for the most units sold at all horsepower levels.[4]

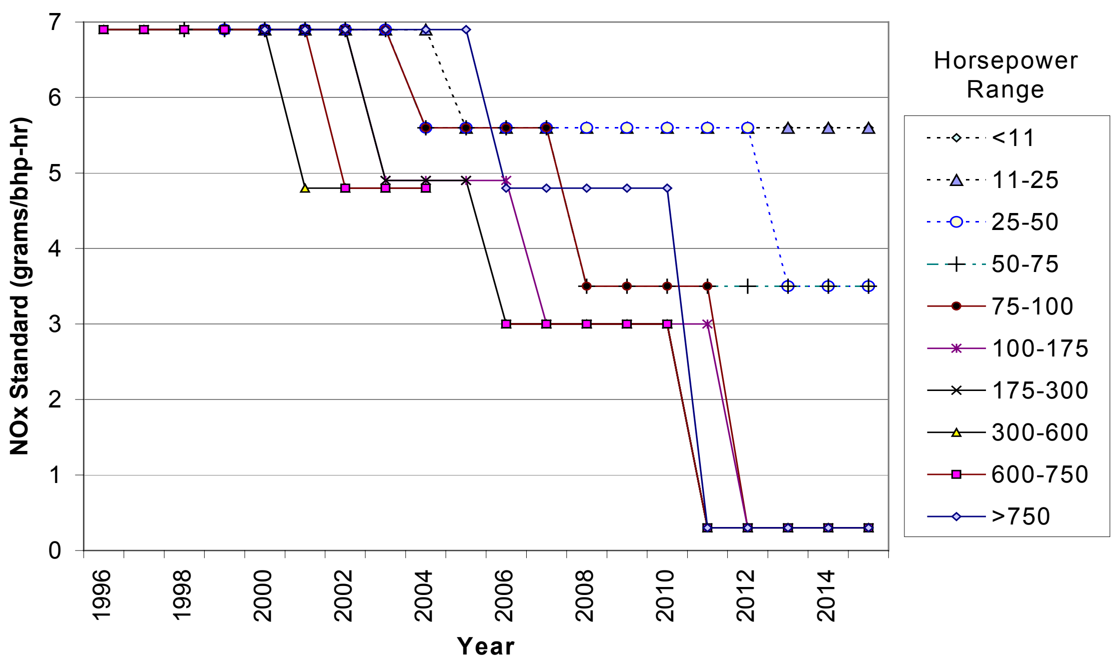
[4] EPA, *Draft Regulatory Impact Analysis: Control of Emissions from Nonroad Diesel Engines*.

B. Emission and Fuel Standards

EPA has already adopted three progressively tougher sets of emission standards for nonroad mobile equipment with engines larger than 50 horsepower. Tier 1 standards phased in from 1996-2000; Tier 2 standards from 2001-2006; and Tier 3 standards from 2006-2008. The Tier 1 standards reduce allowable NOx emissions by 30 percent from uncontrolled levels. The Tier 3 standards reduce NOx by 70 percent and PM by 40 percent from uncontrolled emission levels.[5]

The "Tier 4" standards in the Proposed Rule would phase in from 2008-2014, and would cover even the smallest nonroad diesel engines. The Tier 4 standards require more than a 90 percent reduction in allowable NOx and PM emissions when compared with Tier 3 requirements. Figures 1 and 2 display the progression of NOx and PM standards, respectively, by year and engine horsepower range. Emission limits are in grams of pollutant per "brake-horsepower-hour" (bhp-hr)—a unit of emissions per unit of energy expended. For example, an engine operating at 200 horsepower that emits one gram of NOx per bhp-hr would emit 200 grams of NOx during an hour of operation.

Figure 1. Tier 1 through Tier 4 NOx Standards by Horsepower Range

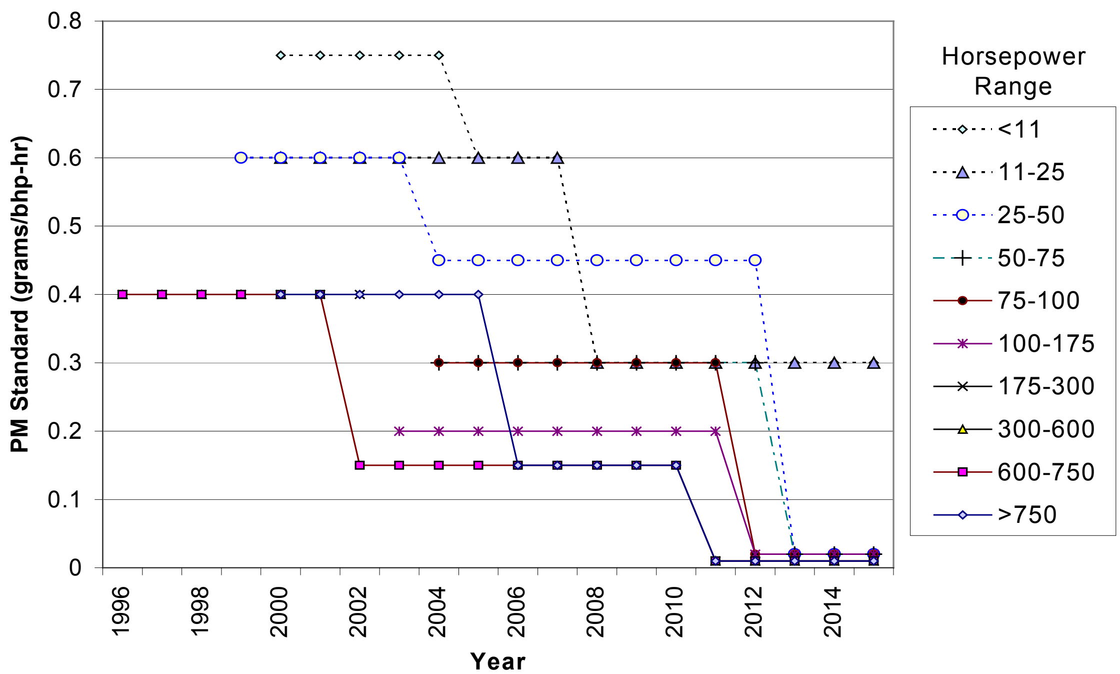


Standards begin with Tier 1 and each decline represents a new tier. Some of the NOx standards are actually limits on the sum of NOx + NMHC, where NMHC is non-methane hydrocarbons.

[5] EPA, *Program Update: Reducing Air Pollution from Nonroad Engines* (Washington, DC: April 2003), http://www.epa.gov/nonroad/f03011.pdf.

Very low fuel sulfur levels are believed to be necessary to achieve the Tier 4 NOx standards, because sulfur fouls the catalytic systems expected to be necessary to achieve such low NOx emissions. EPA plans to reduce allowable sulfur levels in nonroad diesel fuel in two steps. The current nonroad diesel sulfur limit is 5,000 ppm, with an in-use average of about 3,400 ppm. EPA would reduce this limit to a maximum of 500 ppm in 2007 (the same as the current limit for on-road diesel fuel), and 15 ppm in 2010 (the same as the 2006 limit for on-road diesel fuel). Although the Proposed Rule does not apply to locomotive or marine engines, the fuel sulfur limits would apply to locomotive and marine diesel fuel (except for the fuel used by large oceangoing vessels).

Figure 2. Tier 1 through Tier 4 PM Standards by Horsepower Range



Standards begin with Tier 2 (there are no Tier 1 PM limits for some categories) and each decline represents a new tier. Some of the NOx standards are actually limits on the sum of NOx + NMHC, where NMHC is non-methane hydrocarbons.

C. Emission Reductions

Figures 3 and 4 display EPA's emissions forecast for NOx and $PM_{2.5}$ respectively. For NOx, Figure 3 shows the baseline forecast with Tier 3 standards and the incremental reductions with the Tier 4 standards. Emissions begin to rise after 2020 under Tier 3 as projected increases in the total number of nonroad vehicles begins to offset the gains from lower emission rates for each individual engine. For $PM_{2.5}$, Figure 4 displays an additional scenario that assumes Tier 3 emission standards remain in place, but allowable fuel sulfur in reduced to 500 ppm.

Figure 3. Projected Nonroad NOx Emissions with Tier 3 and Tier 4 Standards

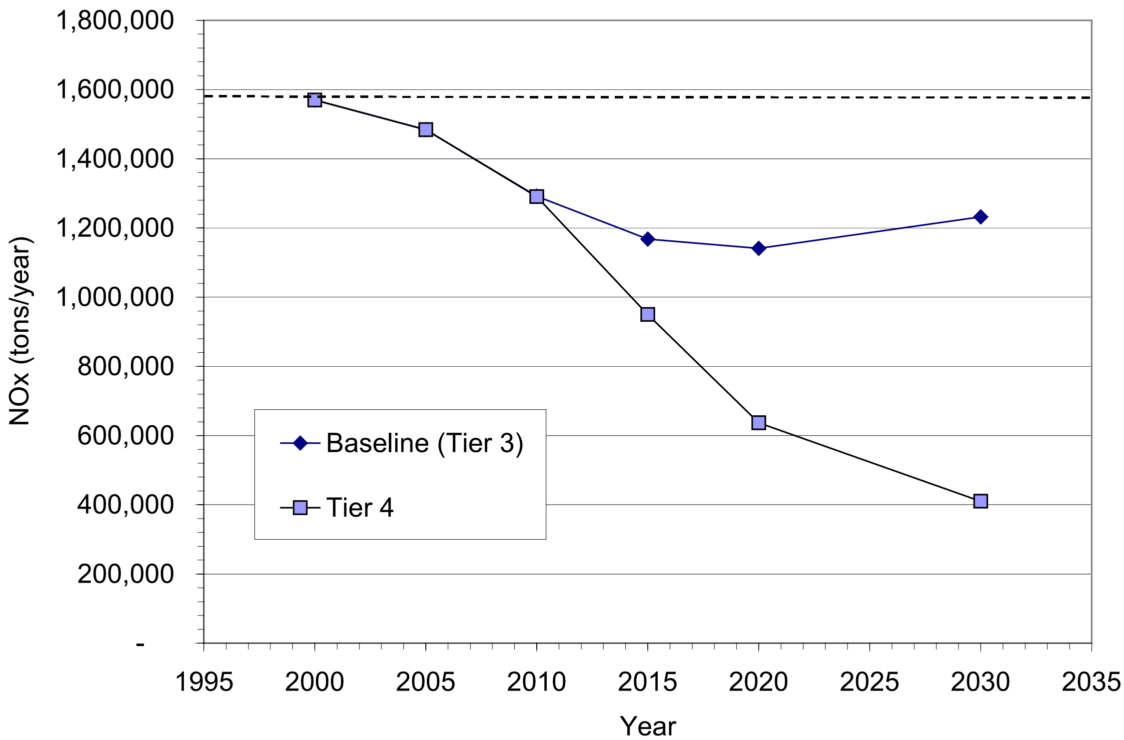
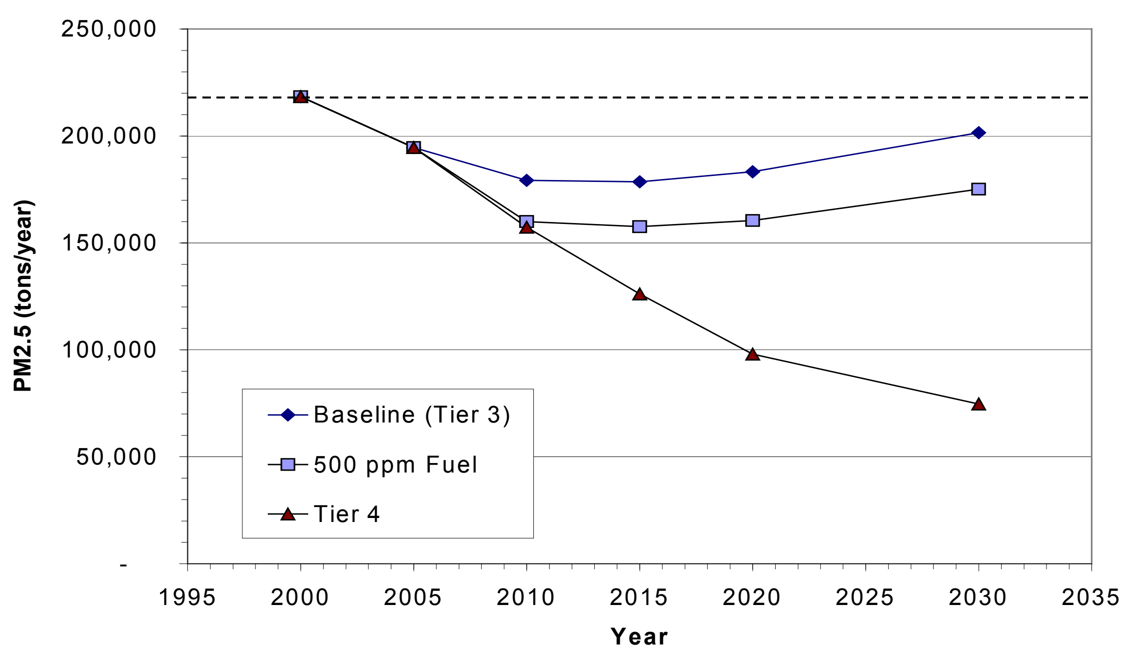


Figure 4. Projected Nonroad $PM_{2.5}$ Emissions with Tier 3, 500 ppm Sulfur Fuel, and Tier 4 Standards



EPA's national NOx inventory for 2001 estimates that nonroad engines account for about 13 percent of mobile source NOx emissions and about 7 percent of all NOx emissions. Based on EPA's projections, the Tier 4 standards would reduce NOx nonroad NOx emissions by about 500,000 tons in 2020—a 2.2 percent reduction from total estimated NOx emissions for 2001.[6] In 2030, the NOx reduction would be 3.6 percent relative to the total 2001 baseline.

$PM_{2.5}$ contributions cannot be estimated from emission inventories, because $PM_{2.5}$ is composed of both direct emissions, and "secondary" particles formed from gases through chemical reactions in the atmosphere. Based on source apportionment studies, diesel PM (that is, organic and elemental carbon) generally comprises about 5 to 15 percent of $PM_{2.5}$ in metropolitan areas, but can account for as much as 30 percent in relatively dense business and commercial districts, such as downtown Los Angeles, and even higher percentages in the densest commercial areas, such as parts of Manhattan.[7] Nonroad diesel engines also contribute small amounts of secondary PM due to NOx, sulfur dioxide (SO2) and volatile organic compound (VOC) emissions, but direct PM emissions are the major component. EPA estimates that 44 percent of diesel PM comes from non-road engines, though it is not known what percentage of metro area diesel PM comes from on-road versus nonroad sources.

D. Changes in Ambient Pollution Levels and Attainment Status

The main goals of the rule are reductions in $PM_{2.5}$ and ozone. EPA performed regional air quality modeling to predict future ozone and $PM_{2.5}$ levels based on predicted future emission reductions. Table 1 displays the number of counties predicted to be out of attainment with $PM_{2.5}$ standards, given predicted baseline emission reductions from existing requirements, and the additional emission reductions expected from the Proposed Rule. Table 2 displays EPA's modeling results for the fraction of the population that experience a given amount of $PM_{2.5}$ reduction. As the table shows, EPA predicts most people would experience $PM_{2.5}$ reductions on the order of zero to one $\mu g/m^3$. A reduction of 0.5 $\mu g/m^3$ amounts to about a 2.5 to 5 percent reduction across the typical range of $PM_{2.5}$ levels.

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[6] The nonroad NOx reduction was calculated from the RIA, Table 3.3-5b, page 3-65. The 2001 inventory is the version released in February 2003 and was downloaded from EPA's emission inventory Web page, http://www.epa.gov/ttn/chief/trends/.

[7] J. C. Chow and J. G. Watson, "Review of PM2.5 and PM10 Apportionment for Fossil Fuel Combustion and Other Sources by the Chemical Mass Balance Receptor Model," *Energy & Fuels*, vol. 16 (2002), pp. 222-260, EPA, *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements* (Washington, DC: December 2000), www.epa.gov/otaq/diesel.htm.

Table 1. EPA's Prediction of the Trend in the Number of $PM_{2.5}$ Non-Attainment Counties and Their Populations

	Counties		Population (millions)	
Year	Base	Tier 4	Base	Tier 4
2000	149	149	74	74
2020	79	67	66	60
2030	107	84	86	71

Source: Nonroad RIA at 2-15.

Table 2. Predicted Distribution of $PM_{2.5}$ Reductions Among U.S. Population Due to the Proposed Rule

Decrease in annual-average $PM_{2.5}$ Levels ($\mu g/m^3$)	2020		2030	
	Number (millions)	Percent	Number (millions)	Percent
0 – 0.25	65	20	29	8
0.25 – 0.5	185	56	147	41
0.5 – 0.75	57	17	107	30
0.75 – 1.0	15	4	39	11
1.0 – 1.25	5	2	9	2
1.25 – 1.5	4	1	16	4
1.5 – 1.75	0	0	6	2
> 1.75	0	0	4	1

Source: Nonroad RIA at 9-32.

EPA also predicts modest changes in ozone levels due to the rule, as shown in Table 3. The number of people in 8-hour non-attainment counties increases in 2020 due to the Proposed Rule, because the Bronx is projected to be pushed into exceeding the 8-hour standard due to the Proposed Rule. The ozone modeling predicts that the average non-attainment county would achieve a 1.9 ppb reduction in its 8-hour ozone design value in

2020, and 3 ppb in 2030.[8] EPA also predicts that two counties would have increased ozone in 2020 and four in 2030 due to the Proposed Rule.[9]

Table 3. EPA's Prediction of the Trend in the Number of Ozone Non-Attainment Counties and Their Populations

	Counties		Population (millions)	
Year	Base	Tier 4	Base	Tier 4
2000	291	291	111	111
2020	30	28	43	44
2030	32	28	47	46

Source: RIA 2-95 and 2-103.

E. Estimated Benefits and Costs

EPA predicts the Proposed Rule would annually prevent 9,600 premature deaths, 8,300 hospitalizations, and almost one million lost workdays. EPA estimates that in 2004, the net present value (NPV) of the cost of the Tier 4 standards from 2007-2030 would be $17 billion, while the monetized NPV of the health and welfare benefits would be $550 billion.[10] About 92 percent of the estimated benefits are due to reductions in premature mortality predicted to occur from reductions in $PM_{2.5}$. Another 5 percent are other health benefits, and the remaining three percent is visibility benefits.[11]

The program costs include expected engine maintenance and reliability benefits of lower fuel sulfur levels, which reduce the estimated net cost of the rule by about 14 percent. On an annual basis, EPA estimates that in 2020, the cost of the Proposed Rule would be $1.4 billion, and the benefits $42 billion. The respective values for 2030 are $1.5 billion and $79 billion.

For the 500 ppm fuel sulfur program alone, EPA estimates for 2007-2030 a cost of $3.3 billion and a net cost after maintenance savings of $0.4 billion. Annual net costs would be about $50 million per year in the first few years, and $18 million per year in later years.

[8] RIA at 2-110.

[9] RIA at 2-111.

[10] EPA used a 3% discount rate for this analysis.

[11] RIA at 9-34.

In terms of cost to consumers, EPA estimates that the 500 ppm sulfur requirement will raise nonroad diesel fuel costs by 2.5 cents per gallon, and that the 15 ppm requirement will add an additional 2.5 cents per gallon in costs. For engines, EPA estimates that the average cost of nonroad engines will increase about 23 percent in 2013, declining to 19 percent after 2020. The largest percentage increases would be for smaller engines—for example, with average increases of 34 percent, or $852, for 25-50 hp engines. The smallest price increases would be for larger engines—for example, about 6 percent, or $7,000, for engines greater than 600 hp.

III. Statutory Basis for Regulation

In the Preamble to the Proposed Rule EPA states, "Section 213 of the [Clean Air] Act gives us the authority to establish emissions standards for nonroad engines and vehicles. Section 213(a)(3) authorizes the Administrator to set standards for NOx, VOCs, or carbon monoxide, to reduce ambient levels of ozone and carbon monoxide which 'standards shall achieve the greatest degree of emission reduction achievable through the application of technology which the Administrator determines will be available for the engines or vehicles.' As part of this determination, the Administrator must give appropriate consideration to cost, lead time, noise, energy, and safety factors associated with the application of such technology. Section 213(a)(4) authorizes the Administrator to establish standards to control emissions of pollutants which 'may reasonably be anticipated to endanger public health and welfare.' Here, the Administrator may promulgate regulations that are deemed appropriate for new nonroad vehicles and engines which cause or contribute to such air pollution, taking into account costs, noise, safety, and energy factors. EPA believes the proposed controls for PM in today's rule would be an appropriate exercise of EPA's discretion under the authority of section 213(a)(4)."[12]

IV. Overview of Issues

EPA's main reason for promulgating the Proposed Rule is to aid attainment of the 8-hour ozone and annual $PM_{2.5}$ standards, and to reduce ambient levels of diesel exhaust.[13] Secondary concerns include visibility and ecological effects. EPA considers the Proposed Rule to be a natural follow-on to rules EPA has recently promulgated to control emissions from other mobile sources.[14]

EPA's Tier 2 rule, promulgated in 1999, requires roughly a 90 percent reduction in emissions from on-road gasoline vehicles, when compared with Tier 1 standards.[15] EPA's

[12] EPA, "Control of Emissions of Air Pollution from Nonroad Diesel Engines and Fuel; Proposed Rule."

[13] RIA at 2-3.

[14] RIA at 2-1.

[15] The Tier 2 rulemaking documents can be downloaded here: http://www.epa.gov/otaq/tr2home.htm. However, the emission reduction estimates EPA prepared for the 1999 rulemaking appear to substantially underestimate likely future emission reductions due to the rule. For a more recent EPA analysis using

heavy-duty rule imposes similarly stringent standards for on-road diesel trucks.[16] In 1998, EPA also promulgated the NOx SIP Call regulation, which will reduce ozone-season (May-September) NOx from electricity boilers in the Midwest, southeast, and northeast by 60 percent starting in 2004.[17]

While there are still areas that exceed the 1-hour ozone and PM_{10} standards, non-attainment is less widespread, and most non-attainment areas are relatively close to meeting the standards. For example, 13 percent of U.S. monitoring locations exceed the 1-hour ozone standard, and they average about four exceedance days per year.[18] Less than 3 percent of monitoring locations have more than 5 exceedances per year, and virtually all of these locations are in California. Likewise, only about 3 percent of PM_{10} monitoring locations exceed the annual or 24-hour PM_{10} standards.[19] Two thirds of these locations exceed only one of the 2 PM_{10} standards and are very close to attainment. The other locations, once again mostly in California, exceed both standards, sometimes by a substantial margin.

On the other hand, exceedance of EPA's new and more-stringent standards is much more common. Roughly 30 percent of monitoring locations exceed the annual $PM_{2.5}$ standard, and just over 40 percent exceed the 8-hour ozone standard.[20] Although EPA also promulgated a 24-hour $PM_{2.5}$ standard, this standard is relatively less stringent. Only about 2 percent of monitoring locations, almost all in California, exceed the standard.[21]

Although EPA has promulgated some broad and stringent rules geared toward aiding attainment of the 8-hour ozone and annual PM standards, the agency has unfortunately not made any serious effort to revisit whether attaining these standards will provide net benefits to human health and welfare once all costs and benefits are tallied.

MOBILE6.0, see R. A. Giannelli, J. H. Gilmore et al., *Sensitivity Analysis of MOBILE6.0* (Ann Arbor, Michigan: Environmental Protection Agency, December 2002). For a forecast using on-road and I/M data along with the requirements of the Tier 2 rule see J. Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline* (Washington, DC: American Enterprise Institute, July 2003), http://www.aei.org/docLib/20030804_4.pdf.

[16] The heavy-duty rulemaking documents can be downloaded here: http://www.epa.gov/otaq/diesel.htm#hd2007.

[17] EPA, *Addendum to the Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions* (Washington, DC: September 1998).

[18] Based on analysis of national monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. All locations with data for 2000-2002 were included.

[19] Based on analysis of national monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. All locations with data for 1999-2001 were included.

[20] Based on analysis of national monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. All locations with data for 1999-2001 were included.

[21] Based on the three-year average of the 99th percentile of daily $PM_{2.5}$ readings, less than 4 percent of monitoring locations exceeded 65 mg/m3 for 1999-2001. The standard is based on the 98th percentile, but this value was not available in EPA's AIRData database at the time of this analysis. Since the 98th percentile is significantly less rigorous a standard, I've given a rough estimate of 2 percent for the exceedance rate.

All other things being equal, less pollution is better than more. But regulations never leave all other things equal. They divert resources from other activities, increase the costs of producing useful goods and services, encourage rent-seeking, and reduce families' disposable incomes—all of which ultimately cause offsetting reductions in health and welfare. And even things we label "pollution" can sometimes confer offsetting health benefits that are lost when the pollutant is reduced.[22] Only by ensuring that a regulation will confer benefits in excess of the social costs it imposes can regulators improve people's overall welfare.

EPA itself concluded that requiring attainment of the 8-hour ozone standard would cause net harm by imposing costs on the public far in excess of the health benefits achieved. And while EPA claims enormous benefits from even small $PM_{2.5}$ reductions, these benefits appear only via a selective and uncritical reading of the epidemiologic literature. Furthermore, although not explicitly mentioned in the RIA, most of the health benefits EPA claims for the Proposed Rule accrue from reductions in pollution from baseline levels that are already below federal health standards ostensibly set to protect public health with an adequate margin of safety.

Related to the health effects issues, EPA also engages in a series of exaggerations that inflate the apparent harm from current levels of air pollution. These include exaggerating the number of people exposed to pollution in excess of federal standards, the fraction of the population "at risk" from current pollution levels, and the degree of health damage caused by a given level of exposure to air pollution.

EPA is remiss in its glib dismissal of the risk that NOx reductions will make ozone worse for tens of millions of Americans who live in VOC-limited urban areas. EPA has embarked on a strategy of large, near-term NOx reductions. However, recent ozone modeling studies and "weekend effect" research have concluded that NOx reductions are very likely detrimental to progress on ozone in many areas.

EPA's overall pollution control strategy, and the Proposed Rule in particular, suffer from a series of emission inventory problems that make the RIA's forecast of future NAAQS attainment rates implausible. Inventory issues include MOBILE6's large overestimation of current and future CO emissions, and future NOx and VOC inventories that appear to greatly underestimate baseline future emission reductions in the absence of the Proposed Rule.

At the same time that EPA has inflated the Proposed Rule's probable net health benefits, the Agency has also taken pains to minimize the probable costs to U.S. consumers, workers and investors.

The sections below discuss these issues in more detail.

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[22] As will be discussed later, EPA concluded, for example, that reducing ground-level ozone would increase the incidence of skin cancer.

V. EPA Has Overestimated The Rule's Probable Health Benefits by Exaggerating Current Air Pollution Problems

The RIA's discussion of the health effects of air pollution misleads Americans into believing they are exposed to more air pollution, more often, and with greater health damage than is actually the case. While this might serve EPA's interest in building and maintaining support for increasingly stringent regulations, the public's interest is in an *accurate* portrayal of risk. People ultimately bear regulatory costs through reductions in their disposable income, because regulations increase the costs of producing useful goods and services. If EPA leads people to overestimate their risks, they will likely demand more air pollution control than would be consistent with net gains to health and welfare.

The main reason for reducing air pollution is to improve Americans' health. EPA's case for further regulating nonroad diesel engines rests mainly on predicted reductions in mortality due to PM reductions, and to some extent on reductions in other less-severe harms. However, the vast majority of the health benefits EPA claims for the Proposed Rule are unlikely to be realized, because EPA has greatly overestimated the health damage caused by current air pollution levels.

A. EPA Overestimates Harm from Current Air Pollution Levels

An RIA is not the place for EPA to perform a comprehensive review of the health effects of PM, or to cite every study in the literature, and this commentary does not recommend that EPA do so. Nevertheless, EPA has failed in its duty to provide the public with an accurate assessment of the weight of the evidence on the health effects of current air pollution levels. As the following sections show, the case for increased mortality due to current levels of PM is much weaker than EPA claims. Because more than 90 percent of the ostensible benefits of the Proposed Rule are due to reductions in premature mortality, hardly any of the benefits EPA claims for the Proposed Rule will actually be realized.[23]

1. Long-Term Exposure to Particulate Matter and Health

EPA mischaracterizes the results of research on the relationship between long-term exposure to $PM_{2.5}$ and mortality, creating an illusion of robustness, certainty, and consistency in the data and research that does not in fact exist. For example, the RIA states, "The most extensive study and analyses has been based on data from two prospective cohort groups, often referred to as the Harvard 'Six-City study' (Dockery et al., 1993) and the 'American Cancer Society or ACS study' (Pope et al., 1995); these studies have found consistent relationships between fine particle indicators and premature mortality across multiple locations in the U.S."[24] The RIA goes on to state "The

[23] The following sections are a very condensed summary of J. Schwartz, *Particulate Air Pollution: Weighing the Risks* (Washington, DC: Competitive Enterprise Institute, April 2003), which is attached as an appendix to this commentary.

[24] RIA at 9-118. The studies referred to are C. A. Pope, 3rd, M. J. Thun et al., "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults," *American Journal of Respiratory and*

credibility of these two studies is further enhanced by the fact that they were subject to extensive reexamination and reanalysis by an independent team of scientific experts commissioned by the Health Effects Institute (Krewski et al., 2000)."[25]

This is a significant misrepresentation of the results of these studies. A number of features of the ACS and Six Cities results suggest that the reported association of $PM_{2.5}$ with mortality is spurious. For example, the ACS study assessed health effects using a statistical model that included $PM_{2.5}$ as the only pollutant. But the Health Effects Institute (HEI) reanalysis included SO_2 levels in the model as a potential confounder and found that the $PM_{2.5}$ effect disappeared. Only SO_2 appeared to be associated with mortality. This strongly suggests that the ACS results suffered from confounding by other pollutants.[26]

Other ACS study results suggest that the apparent association of $PM_{2.5}$ with mortality might instead be a spurious association caused by residual confounding. For example:

- There was no association between $PM_{2.5}$ and mortality for persons with more than a high-school education, for women, and for people between the ages of 60 and 69.[27]
- $PM_{2.5}$ was associated with increased mortality for former smokers, but not current- or never-smokers.
- $PM_{2.5}$ was associated with increased mortality for people who said they were moderately active, but not for people who said they were either sedentary or very active.

Critical Care Medicine, vol. 151, no. 3 Pt 1 (1995), pp. 669-674, D. W. Dockery, C. A. Pope, 3rd et al., "An Association between Air Pollution and Mortality in Six U.S. Cities," *New England Journal of Medicine*, vol. 329, no. 24 (1993), pp. 1753-1759.

[25] RIA at 9-119. The study referred to is D. Krewski, R. T. Burnett et al., *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality* (Cambridge, MA: Health Effects Institute, July 2000).

[26] Epidemiologists do not believe that SO_2 at current low levels could be causing harm, but rather that SO_2 may be acting as a surrogate for the pollutant mixture in a given area (see, for example, G. Hoek, B. Brunekreef et al., "Daily Mortality and Air Pollution in the Netherlands," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp. 1380-1389., S. H. Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," *Environmental Health Perspectives*, vol. 108, no. 8 (2000), pp. 777-784., and F. W. Lipfert, "Commentary on the HEI Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality," *Journal Toxicology and Environmental Health, Part B*, in press (2003). Current SO_2 levels are 50 percent below those of 1980 and 75 percent below those of the 1960s. 98% of monitoring locations never reach SO_2 levels of even half the federal health standard (current SO_2 levels are based on author's analysis of national SO_2 monitoring data downloaded from EPA's AIRData Web site, www.epa.gov/aqspubl1/select.html. SO_2 trends since 1980 come from EPA, *Latest Findings on National Air Quality: 2000 Status and Trends* (Washington, DC: September 2001 2001), www.epa.gov/oar/aqtrnd00/brochure/00brochure.pdf. Pre-1980 trends come from I. M. Goklany, *Clearing the Air: The Real Story of the War on Air Pollution* (Washington, DC: Cato, 1999). Figure 3-2. The pre-1980 data are based on only 21 monitoring locations, while more recent data are based on several hundred locations).

[27] When cardiopulmonary and lung cancer mortality were looked at separately, both men and women had an increased risk of the former, while only men had an increased risk of the latter.

- $PM_{2.5}$ was *not* associated with an increase in lung cancer mortality in the HEI reanalysis, which covered the period 1982-1989, but was associated with an increase in mortality due to other cancers.[28]

- When population change was added into the statistical model as a potential confounder, the $PM_{2.5}$ effect declined by two thirds and became statistically insignificant.[29] The hypothesis is that people who leave a city are more likely to be healthier than people who remain behind. Cities that lost population—Midwest "rust belt" cities—also had higher $PM_{2.5}$ levels on average. Thus, the apparent effect of $PM_{2.5}$ could actually have resulted from a reduction in the average health of residents caused by healthier people moving away from areas of the country that were in economic decline.

In addition, the ACS study reported that higher $PM_{2.5}$ levels were *not* associated with an increased risk of mortality due to respiratory disease; a surprising finding, given that PM would be expected to exert its effects through the respiratory system.[30]

Another concern with the ACS study is that information about participants' health-related behaviors and status, such as diet, body-mass index (BMI; a measure of relative body size) and smoking were assessed only in 1982 when they entered the study, but not afterward. If any of these factors changed after 1982, and if the changes were correlated with pollution levels, then the study results would suffer from additional uncontrolled confounding. For example, if people living in areas with higher pollution were also either more likely to get fatter, or less likely to stop smoking between 1982 and 1998 when compared with people in lower-pollution areas, researchers could mistake an effect of body weight or smoking for an effect of air pollution. The rate of BMI increases or smoking decreases and the likelihood of living in an area of greater air pollution are probably positively correlated through their common association with socio-economic factors such as income and education, suggesting this is a concern worth additional investigation.[31]

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[28] See Table 20 in Krewski, Burnett et al., *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*. ACSII did find an association between $PM_{2.5}$ and lung-cancer mortality for the period 1982-1998. However, even this association held only for men, those with no more than a high-school education, and those not in the 60-69 age range.

[29] See Table 37 in Ibid. The term "statistically significant" is a term of art in statistical analysis used to signify a result that is considered, based on objective criteria, unlikely to have occurred by chance due to random variability in the data. The word "significant" in this context does not in any way mean "important" or "noteworthy" as it would in everyday use. In addition, simply because a result is statistically significant does not mean that it represents a "real" effect, because the underlying data or statistical model could suffer from various kinds of bias (e.g., confounding), which are a much larger source of uncertainty in epidemiologic studies than the effect of random variation in the data. Statistical significance is thus generally considered a necessary, but not sufficient condition for a statistical result to be considered as genuinely representing some underlying real feature of the world.

[30] See Table 20 in Ibid.

[31] According to the Centers for Disease Control, Americans' average BMI has indeed increased substantially during the last 20 years, and poorer people and minorities are at greater risk for obesity than whites and wealthier people. People with less education were less likely to stop smoking during the last 20

Long-term studies are based on the hypothesis that chronic exposure to elevated pollution causes the development of cardiovascular disease or cancer. These diseases have latencies of 15 to 20 years between exposure and manifestation of disease, suggesting that pollution exposure should be measured during a time period years before the health effect appears. Yet the ACS pollution measurements occurred around the same time the study began in early 1980s, and the range of PM levels was about four times higher during the 1960s than during the 1980s.[32] If it was these earlier high PM levels that actually caused the health effects, then the real effect of air pollution would be one-fourth that estimated in the ACS study. This is because studies like ACS estimate the concentration-response function for PM health effects based on the range of PM levels across cities in the study. If this range is actually four times greater than the range used in the ACS study, then the health effects of a given increase in PM would be one fourth of what the ACS study estimated.[33]

The ACS results also suggest that $PM_{2.5}$ risks are decreasing with time. The ACS study reported that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 6.9 percent increase in mortality for the period 1982-1989. But this risk declined to 2.5 percent for 1990-1998 period, 64 percent lower than for 1982-1989.[34] The PM-mortality relationship for 1990-98 is also statistically insignificant.[35]

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years when compared with more educated people. Minorities are more likely to live in areas with more particulate pollution. Thus, there is a significant potential for changes in BMI, smoking or other health-related behaviors to be mistaken for an effect of air pollution through their common association with socio-economic factors. (Sources: Obesity: National Center for Health Statistics, “Health, United States, 1998, with Socio-Economic Status and Health Chartbook,” Centers for Disease Control, 1999, www.cdc.gov/nchs/data/hus/hus98.pdf; Smoking: National Center for Health Statistics, “Health, United States, 2001,” Centers for Disease Control, 2001, www.cdc.gov/nchs/data/hus/hus01.pdf; Air Pollution: National Center for Health Statistics, “Health, United States, 1998,” and Victor Brajer and Jane V. Hall, “Recent Evidence on the Distribution of Air Pollution Health Effects,” *Contemporary Policy Issues*, vol. 10 (April 1992), pp. 63-71).

Because the risks of smoking and obesity are so much larger than the risk the ACS study estimated for $PM_{2.5}$**,** even a small difference in smoking and obesity trends between areas with differing pollution levels could swamp the ostensible effect of differences in air pollution. For example, ACSII found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ increases mortality risk by 4 percent. But for a six foot, 200-pound, non-smoking man, gaining just 15 pounds increases his risk of an early death by 17 percent (E. E. Calle, T. MJ et al., “Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults,” *New England Journal of Medicine*, vol. 341 (1999), pp. 1097-1105..

[32] Lipfert, “Commentary on the HEI Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality.

[33] Ibid.

[34] C. A. Pope, 3rd, R. T. Burnett et al., “Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution,” *Journal of the American Medical Association*, vol. 287, no. 9 (2002), pp. 1132-1141. Pope et al. did not point out this key feature of their analysis in their published results. They reported only results for 1982-1989 (in the first ACS study published in 1995) and 1982-1998 (in the study cited here published in 2002). However, the results for 1990-1998 can be inferred from the data presented ACSI and ACSII.

[35] The fact that the 1990-1998 PM-mortality relationship is statistically insignificant can be inferred from the magnitude of the PM-mortality relationship for 1990-1998 and the 95 percent confidence intervals reported for the other time periods.

Contrary to EPA's claims in the RIA, the HEI reanalysis did not resolve the concerns raised above, and actually revealed several of them.

There is also evidence that the Harvard Six Cities (HSC) results suffer from residual confounding. For example, HSC did not account for physical activity level of the study participants, yet exercise is strongly correlated with health. It turns out that levels of physical activity in the six cities are inversely correlated with pollution levels in these cities.[36] HSC might therefore have attributed to air pollution a health effect that was actually caused by lower physical activity levels. Like the ACS study, there was no association between $PM_{2.5}$ and mortality in people with more than a high-school education. HSC also found that greater $PM_{2.5}$ was associated with a statistically insignificant *decrease* in mortality due to respiratory causes specifically.

The HSC study was based on $PM_{2.5}$ levels measured concurrent with the beginning of the follow-up period, even though mortality was due to diseases with long latency times. Therefore, like the ACS study, the HSC study might therefore have inflated the apparent effect of $PM_{2.5}$ on mortality, compared to an assessment based on much greater $PM_{2.5}$ levels in the two decades leading up to the HSC follow-up period.

Because HSC included only s6 locations, it was not possible to investigate whether including other pollutants in the statistical analysis affected the apparent mortality contribution of $PM_{2.5}$.

Taken together, these problems of uncontrolled confounding, latency time for disease development, and biologically implausible variations in the apparent $PM_{2.5}$-mortality association suggest that the reported relationship between $PM_{2.5}$ and mortality reported in the ACS and Harvard Six Cities studies is likely to be spurious and not representative of a cause-effect relationship.

Even taking the ACS results at face value, $PM_{2.5}$ does not increase mortality for women or for those with more than a high school education. Together these groups make up 75 percent of the population. Thus, by a plain reading of the studies EPA relied on for estimating the benefits from the Proposed Rule, 75 percent of the mortality benefits claimed by EPA would never actually be realized.

The RIA for the Proposed Rule also misrepresents the results of another study that did not find an association between $PM_{2.5}$ and mortality. The RIA states, "More recently, a cohort of adult male veterans diagnosed with hypertension has been examined (Lipfert et al., 2000). The characteristics of this group differ from the cohorts in the ACS, Six-Cities, and 7th Day Adventist studies with respect to income, race, and smoking status. Unlike previous long-term analyses, this study found some associations between

[36] F. W. Lipfert, "Estimating Air Pollution-Mortality Risks from Cross-Sectional Studies: Prospective vs. Ecologic Study Designs," Health and Regulatory Issues, Proceedings of the International Specialty Conference, Air and Waste Management Association, 1995.

mortality and ozone but found inconsistent results for PM indicators."[37] We refer to this study as the "Veterans Study" in the discussion below.[38]

Despite EPA's claims, the Veterans Study provides in several ways a better test of the effects of PM exposure on long-term health. The study population included men with preexisting high blood pressure, which should have made them more susceptible to the effects of PM than the comparatively healthy populations of the ACS and Six Cities studies. Unlike the ACS and Six Cities studies, the Veterans study assessed associations between PM and mortality for several time periods, including both concurrent and delayed health effects of pollution exposure. Thus, the Veterans Study performed a more realistic assessment of the association of PM and mortality for diseases with long latency periods.

Rather than the "inconsistent results" claimed by EPA, the Veterans Study reported a statistically significant *decrease* in mortality associated with $PM_{2.5}$. When various ecological confounding variables were added to the statistical analysis, $PM_{2.5}$ was associated with an even greater reduction in mortality. While it is not plausible that higher $PM_{2.5}$ could improve health, this study suggests that chronic exposure to $PM_{2.5}$ at levels that existed during the 1970s and 1980s is not associated with increases in mortality. EPA correctly notes that the Veterans Study "found some associations between mortality and ozone" but neglected to mention that increased mortality was only observed at ozone levels greater than about 0.13 ppm. In other words, based on the Veterans Study, the 1-hour standard is health protective in terms of premature mortality.

The Veterans Study did not control for diet and exercise. In addition, the study also assessed people only at entry, so some personal characteristics may have changed. As a result, there may be some residual confounding that could explain the anti-correlation between $PM_{2.5}$ and health. Nevertheless, this study's statistical analysis of individual health factors is more comprehensive than that of the ACS or Six Cities studies, because it includes other non-pollution health-related factors, such as age, smoking-status, blood pressure, and body-mass index. Further, these factors had the expected association with mortality (e.g., high blood pressure was associated with increased risk of death), making it more difficult to discard the pollution results. The study assessed the effect of $PM_{2.5}$ alone, and was not able to determine whether adding other pollutants to the analysis would change the apparent $PM_{2.5}$ effect.

Because the Veterans Study assessed only male veterans with high blood pressure, the results might not hold for the U.S. population in general (though recall that the ACS study found no relationship between PM and mortality for women). However, one would expect that the study group would be *more* susceptible to PM-induced health effects than the general population.

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[37] RIA at 9-119.

[38] The study is F. W. Lipfert, H. M. Perry et al., "The Washington University-EPRI Veterans' Cohort Mortality Study," *Inhalation Toxicology*, vol. 12 (suppl. 4) (2000), pp. 41-73.

The RIA also makes another serious omission in relation to the Veterans Study. The citation to the Veterans Study appears in the Cost-Benefit section of the RIA (Chapter 9), but is never discussed in the section on health effects of air pollution (Chapter 2). On the other hand, the ACS and Six Cities results are cited in both sections.

EPA omits from the RIA another important study of the relationship between PM and mortality.[39] The "County Study" was a fully ecological study,[40] included all U.S. counties with air pollution monitoring data, and assessed the relationship between pollution levels and mortality at the county level between 1960 and 1997. Like the Veterans Study, the County Study also assessed the relationship between pollution and mortality for several time periods, and assessed both concurrent and delayed health effects of pollution exposure.

The County Study found an association between greater $PM_{2.5}$ and increased mortality. However, there appeared to be a threshold somewhere between 20 and 25 $\mu g/m^3$, below which $PM_{2.5}$ had no effect. In addition, the relationship between pollution and mortality was strongest when pollution exposure occurred within a few years of death. There was little or no evidence for cumulative effects from longer-term pollution exposure. Only about three percent of $PM_{2.5}$ monitoring locations exceeded 20 $\mu g/m^3$ for 1999-2001, and most of these locations are in California.[41]

In a comparison with counties that were part of the Harvard Six Cities Study, the County Study reported that $PM_{2.5}$ was associated with an increase in mortality only for Steubenville, which had the highest pollution level in that study, and that the threshold $PM_{2.5}$ level for mortality increases was at least 23 $\mu g/m^3$.

When looking at different age groups, the health effects of pollution were larger for younger age groups. This argues against chronic effects, because effects should be greater for people with more cumulative exposure. Like the ACS study, the County Study did not find a mortality risk associated with PM_{10}.

This study included a wider range of non-pollutant confounders in the analysis when compared with other studies of long-term mortality, and found the expected directions for their effects, also adding weight to the validity of the estimated pollution effects.

The problems of uncontrolled confounding, short latencies, and biologically implausible associations in the ACS and Harvard Six Cities studies, combined with the negative results of the Veterans and County studies, suggest that PM at current levels is unlikely to

[39] F. W. Lipfert and S. C. Morris, "Temporal and Spatial Relations between Age Specific Mortality and Ambient Air Quality in the United States: Regression Results for Counties, 1960-97," *Occupational and Environmental Medicine*, vol. 59, no. 3 (2002), pp. 156-174.

[40] That is, it assessed all variables aggregated at the county level, rather than following a group of individuals. The other studies are "semi-ecological" cohort studies in that they include some individual data, and some aggregated or "ecological" data, such as pollution exposures.

[41] Based national $PM_{2.5}$ monitoring data downloaded from EPA's AirData web site, www.epa.gov/aqspubl1/select.html.

be increasing mortality due to long-term exposure. The results of these studies suggest that EPA should have set its annual $PM_{2.5}$ standard at no less than 20 μg/m^3.

2. Short-Term Exposure to Particulate Matter and Health

Because only about two or three percent of monitoring locations exceed EPA's 24-hour $PM_{2.5}$ standard, daily $PM_{2.5}$ levels are somewhat moot for policy purposes.[42] Regardless of its policy implications, EPA's discussion of short-term PM health effects creates an illusion of certainty regarding the short-term health effects of PM, because it selectively excludes from the RIA virtually all research that draws conclusions at odds with EPA's preferred findings. The RIA fails to cite or discuss a wide range of research that calls into question several fundamental tenets of the "conventional wisdom" on short-term PM health effects. For example:

- Whether confounding by other pollutants or from other health-related factors, such as temperature and humidity, have been adequately controlled.[43] A recent assessment of the difficulties in sorting out these issues in air pollution epidemiology studies concluded that, "Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results."[44]

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[42] Based on the 99th percentile of daily $PM_{2.5}$ levels, just under 4% of monitoring locations exceed 65 μg/m^3. However, the 24-hour $PM_{2.5}$ standard is based on the 98th percentile, which was not available from EPA AIRData database as of this writing.

[43] See, for example, Moolgavkar, "Air Pollution and Daily Mortality in Three U.S. Counties," R. T. Burnett, S. Cakmak and J. R. Brook, "The Effect of the Urban Ambient Air Pollution Mix on Daily Mortality Rates in 11 Canadian Cities," *Canadian Journal of Public Health. Revue Canadienne de Sante Publique*, vol. 89, no. 3 (1998), pp. 152-156, Hoek, Brunekreef et al., "Daily Mortality and Air Pollution in the Netherlands," D. M. Stieb, S. Judek and R. T. Burnett, "Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season," *Journal of the Air and Waste Management Association*, vol. 52, no. 4 (2002), pp. 470-484, S. H. Moolgavkar, *Review of Chapter 8 of the Criteria Document for Particulate Matter (Comments Submitted to EPA)*, 2002, F. W. Lipfert, S. C. Morris and R. E. Wyzga, "Daily Mortality in the Philadelphia Metropolitan Area and Size-Classified Particulate Matter," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp. 1501-1513, P. Switzer, *A Review of Statistical Methods Used in Time-Series Epidemiologic Studies of Ambient Particulate Matter and Acute Health Effects Cited by the April 2002 EPA Draft PM Criteria Document* (Palo Alto, California: Stanford University, July 8, 2002), E. Hennessy, "Air Pollution and Short Term Mortality," *BMJ*, vol. 324, no. 7339 (2002), pp. 691-692, R. L. Smith, J. M. Davis et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," *Environmetrics*, vol. 11 (2000), pp. 719-743, F. W. Lipfert and R. E. Wyzga, "Air Pollution and Mortality: The Implications of Uncertainties in Regression Modeling and Exposure Measurement," *Journal of the Air and Waste Management Association*, vol. 47, no. 4 (1997), pp. 517-523, F. W. Lipfert and R. E. Wyzga, "Statistical Considerations in Determining the Health Significance of Constituents of Airborne Particulate Matter," *Journal of the Air and Waste Management Association*, vol. 49, no. 9 (1999), pp. 182-191.

[44] T. Lumley and L. Sheppard, "Time Series Analyses of Air Pollution and Health: Straining at Gnats and Swallowing Camels?," *Epidemiology*, vol. 14, no. 1 (2003), pp. 13-14.

- Whether PM in general or a specific component is responsible for health effects. Recent evidence suggests trace metals might be more biologically plausible than carbon or sulfate particles as a cause of acute health effects at current, relatively low PM levels.[45]

- The degree to which researchers' judgment and taste affect the outcome of a modeling study, as shown by different researchers reporting different and even contradictory results based on the same data.[46]

- Whether there is a threshold below which PM has no health effects. Critics of the no-threshold hypothesis point out that any errors in the measurement of pollution exposures will cause an *underestimate* of a threshold, should one exist, and will cause a non-linear concentration-response function to appear linear.[47] In addition, a number of studies have reported identifying a threshold below which PM does not appear to affect health.[48] EPA ignores all but one of these studies when discussing the threshold issue in the RIA.

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[45] In the preamble of the Nonroad NPRM (FR p. 28339) EPA cites a study by Laden et al. that reported a relationship between vehicle-related PM and mortality, but the data for this study were collected when leaded gasoline was still in use and lead could have been the responsible component. R. S. Chapman et al., "Ambient Particulate Matter and Respiratory and Cardiovascular Illness in Adults: Particle-Borne Transition Metals and the Heart-Lung Axis," *Environmental Toxicology and Pharmacology*, vol. 4 (1997), pp. 331-8.J. A. Dye, J. R. Lehmann et al., "Acute Pulmonary Toxicity of Particulate Matter Filter Extracts in Rats: Coherence with Epidemiologic Studies in Utah Valley Residents," *Environmental Health Perspectives*, vol. 109, no. Suppl 3 (2001), pp. 395-403, A. J. Ghio and R. B. Devlin, "Inflammatory Lung Injury after Bronchial Instillation of Air Pollution Particles," *American Journal of Respiratory and Critical Care Medicine*, vol. 164, no. 4 (2001), pp. 704-708, F. Laden, L. M. Neas et al., "Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities," *Environmental Health Perspectives*, vol. 108, no. 10 (2000), pp. 941-947.

[46] See, for example, M. Clyde, "Model Uncertainty and Health Effect Studies for Particulate Matter," *Environmetrics*, vol. 11 (2000), pp. 745-763, Smith, Davis et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," S. H. Moolgavkar and E. G. Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality," *Epidemiology*, vol. 7, no. 4 (1996), pp. 420-428, Lipfert and Wyzga, "Air Pollution and Mortality: The Implications of Uncertainties in Regression Modeling and Exposure Measurement," Switzer, *A Review of Statistical Methods Used in Time-Series Epidemiologic Studies of Ambient Particulate Matter and Acute Health Effects Cited by the April 2002 EPA Draft PM Criteria Document,* S. H. Moolgavkar, E. G. Luebeck et al., "Particulate Air Pollution, Sulfur Dioxide, and Daily Mortality: A Reanalysis of the Steubenville Data," *Inhalation Toxicology*, vol. 7 (1995), pp. 35-44, R. D. Morris, "Airborne Particulates and Hospital Admissions for Cardiovascular Disease: A Quantitative Review of the Evidence," *Environmental Health Perspectives*, vol. 109, suppl. 4 (2001), pp. 495-500, P. Switzer, "Estimating Separately Personal Exposure to Ambient and Nonambient Particulate Matter for Epidemiology and Risk Assessment: Why and How," *Journal of the Air and Waste Management Association*, vol. 51, no. 3 (2001), pp. 322-323; discussion 329-338, R. Klemm "Reanalysis of Harvard Six-City Mortality Study Replication," EPA Workshop on GAM-Related Statistical Issues in PM Epidemiology, Durham, North Carolina, November 4-6, 2002, J. M. Samet, F. Dominici et al., "New Problems for an Old Design: Time Series Analyses of Air Pollution and Health," *Epidemiology*, vol. 14, no. 1 (2003), pp. 11-12.

[47] See, for example, Lipfert and Wyzga, "Statistical Considerations in Determining the Health Significance of Constituents of Airborne Particulate Matter."

[48] R. L. Smith, D. Spitzner et al., "Threshold Dependence of Mortality Effects for Fine and Coarse Particles in Phoenix, Arizona," *Journal of the Air and Waste Management Association*, vol. 50, no. 8 (2000), pp.

- To the extent acute increases in PM cause death, does PM reduce life expectancy by only days in already-frail people or by months or years in healthy people? If the latter is the case, PM could have a large effect on public health. If the former, which epidemiologists call "harvesting," the health effects of PM would be far smaller. EPA does not address harvesting in its RIA. Studies that have attempted to estimate directly when death occurs in relation to increases in pollution have concluded that acute changes in pollution levels shorten life expectancy by a matter of days at most.[49] Harvesting would also provide a more biologically plausible explanation of the association of acute changes in PM with mortality. Various pollutants are always present at some level in ambient air, and pollution levels vary from day to day. It is not clear why apparently healthy people would be suddenly killed on a given day by relatively low PM levels that they have experienced many times in the past.[50] Harvesting would also be consistent with the finding that many different types of pollution—e.g., fine and coarse PM, and various gases—appear to have effects on mortality of similar magnitude, as do changes in temperature, atmospheric pressure and other weather variables.[51] If PM and other pollutants were shortening healthy people's lives by months or years, it would be an odd coincidence if several different pollutants, each with a different intrinsic toxicity and each present at different levels in different cities, all happened to exert roughly the same effects, regardless of the pollutant or its ambient concentration. Finally, harvesting would explain the possible lack of a threshold for the effect of PM on mortality, since changes in pollution, even at low levels, might be enough to cause death in very frail people.[52]

Many of the issues raised above similarly apply to less severe health effects attributed to current $PM_{2.5}$ levels, such as respiratory aggravations. EPA has selectively cited and highlighted virtually only those studies that support a relatively alarmist view of the health effects of daily changes in $PM_{2.5}$ levels. As a result, the RIA creates an impression of certainty and severity regarding acute low-concentration $PM_{2.5}$ effects that is unwarranted based on the totality of the evidence.

1367-1379, R. L. Smith, J. M. Davis and P. Speckman, "Assessing the Human Health Risk of Atmospheric Particles," *Novartis Foundation Symposium*, vol. 220 (1999), pp. 59-72; discussion 72-59, Smith, Davis et al., "Regression Models for Air Pollution and Daily Mortality: Analysis of Data from Birmingham, Alabama," Moolgavkar and Luebeck, "A Critical Review of the Evidence on Particulate Air Pollution and Mortality."

[49] Smith, Davis and Speckman, "Assessing the Human Health Risk of Atmospheric Particles," C. J. Murray and C. R. Nelson, "State-Space Modeling of the Relationship between Air Quality and Mortality," *Journal of the Air and Waste Management Association*, vol. 50, no. 7 (2000), pp. 1075-1080.

[50] F. W. Lipfert, *Unresolved Questions in Air Pollution Epidemiology, Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft* (Annapolis, MD: Annapolis Center for Science-Based Public Policy, July 2002).

[51] Ibid., Stieb, Judek and Burnett, "Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season."

[52] Lipfert, *Unresolved Questions in Air Pollution Epidemiology, Review Comments on the U.S. Environmental Protection Agency's Air Quality Criteria for Particulate Matter, Third External Review Draft,* R. Frank and C. Tankersley, "Air Pollution and Daily Mortality: A Hypothesis Concerning the Role of Impaired Homeostasis," *Environmental Health Perspectives*, vol. 110, no. 1 (2002), pp. 61-65.

If EPA had set the annual $PM_{2.5}$ standard at 20 $\mu g/m^3$, the lowest level consistent with the evidence, only about three percent of monitoring locations, mainly in California, would fail to attain either the 24-hour or annual $PM_{2.5}$ standards—a much smaller non-attainment rate than the 30 percent that exceed the annual standard at the current level of 15 $\mu g/m^3$. Instead of a broad national problem, $PM_{2.5}$ would then be correctly thought of as a regional problem, limited to just a few areas of the country. EPA's rationale for national regulation of $PM_{2.5}$ would then disappear.

3. Ozone Health Effects

The RIA asserts that ozone could be causing increased incidence of asthma and cites two studies that reported an association between ozone and development of asthma, one in very-active children, and one in non-smoking men.[53] But even taking the results of these studies at face value, they are irrelevant to current ozone levels in southern California, where they were performed, and they are irrelevant to ozone levels ever experienced anywhere else, since no other area has ever had ozone as high as southern California used to have.

For example, in the Children's Health Study (CHS), researchers reported that children in 4 high-ozone communities who played 3 or more team sports were more than three times as likely to develop asthma as less active children.[54] But in medium- and low-ozone communities there was no difference in asthma incidence based on children's activity level.

The association was based on relatively small numbers; 20 children developed asthma in the high-ozone areas, though six cases would have been expected. But assuming the association is causal, it is irrelevant to current ozone levels. Figure 5 shows the number of days per year exceeding the 1-hour ozone standard in the four high-ozone communities of the CHS during 1994-97, when the study was performed, and during 1999-2001, the most recent data available when the study was released.

The average number of 1-hour ozone exceedances per year in the high-ozone communities declined 75 percent after the study was performed. This suggests the study does not apply to current ozone levels, which are more typical of the medium-ozone areas in the study, for which no association of ozone and asthma was found. The study also does not apply anywhere else in the country, since no area has ever had ozone levels as high as southern California used to have.

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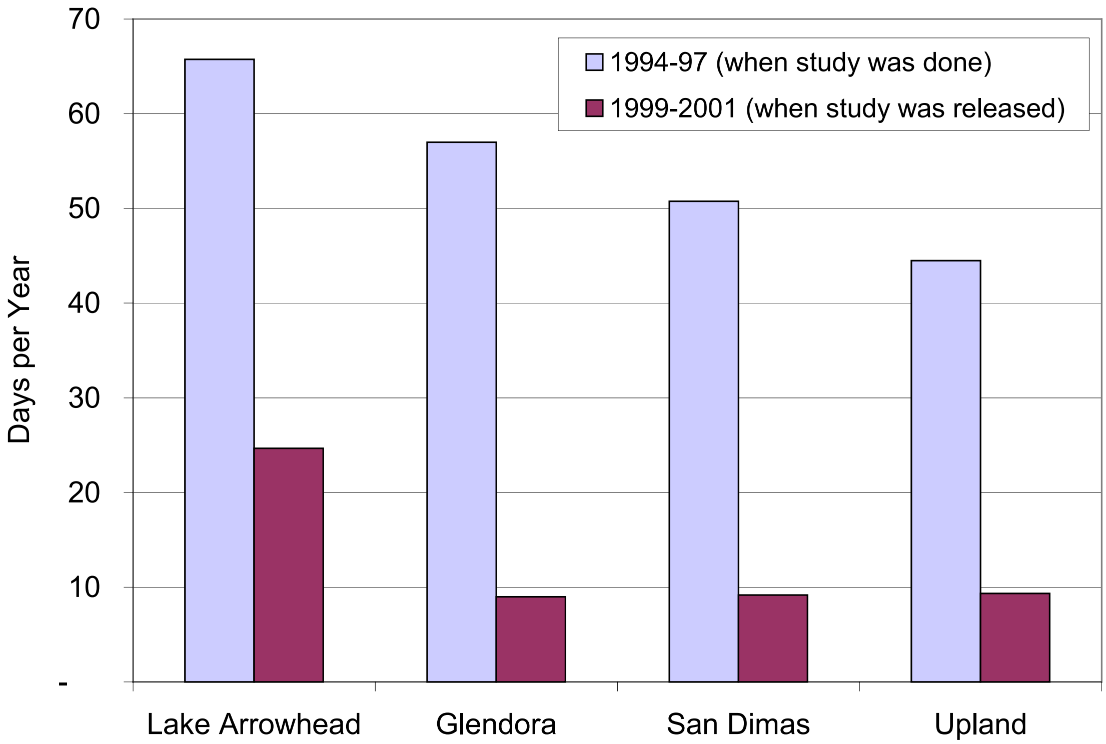
[53] RIA at 2-90. The two studies are W. F. McDonnell, D. E. Abbey et al., "Long-Term Ambient Ozone Concentration and the Incidence of Asthma in Nonsmoking Adults: The AHSMOG Study," *Environmental Research*, vol. 80, no. 2 Pt 1 (1999), pp. 110-121, R. McConnell, K. T. Berhane et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study," *Lancet*, vol. 359 (2002), pp. 386-391.

[54] McConnell, Berhane et al., "Asthma in Exercising Children Exposed to Ozone: A Cohort Study."

Figure 5. Average Number of Days per Year Exceeding the 1-hour Ozone Standard at the Six "High-Ozone" Areas in the Children's Health Study



Source: Ozone data were extracted from the California Air Resources Board's air quality data CD-ROM (# PTSD-02-017-CD). The CHS set up ozone monitors especially for the study. Because CARB has not responded to requests for these data, I've used data from CARB's general-release air pollution data CD. Because the regular monitoring locations don't always coincide with the CHS special-purpose monitors, I used nearby monitors as surrogates—Crestline for Lake Arrowhead, and an average of Glendora and Upland for San Dimas.

Another key result from the CHS omitted by EPA and others who cite the CHS results is that when looking at the total population of children in the high- and low-ozone communities (rather than the eight percent of children classified as very active), *asthma incidence was 30 percent lower in the high-ozone communities* when compared with the low-ozone communities, and the difference was statistically significant.[55] For other pollutants, there was no difference in asthma incidence between high- and low-pollution communities. Like the CHS results, the AHSMOG study results cited in the RIA also don't apply to current ozone levels.

While asthma incidence has doubled during the two decades, outdoor air pollution of all kinds has declined, suggesting that elevated air pollution cannot be a causal factor in

[55] This is based on 1-hour ozone levels. Based on 8-hour ozone levels, asthma incidence was 20% lower in the high-ozone communities and the difference was not statistically significant.

asthma's increasing prevalence. Repeated citation of the Children's Health Study by regulators, environmentalists and the media in the context of air pollution and asthma perpetuates the mistaken belief that air pollution at current levels could be causing asthma.

While EPA does not claim asthma-incidence reduction benefits from the Proposed Rule, EPA does create the impression that implementing the Proposed Rule will reduce asthma incidence—an impression grossly at odds with the data. Indeed, the Proposed Rule has an even more remote relationship to asthma incidence than suggested above, because it will be a decade or so before the Proposed Rule begins to make a significant dent in emissions from nonroad equipment. But by then other EPA rules, such as Tier2, the 2003 and 2007 on-road heavy-duty diesel standards, the Tier 3 nonroad standards, and the NOx SIP Call will have greatly reduced emissions below current levels.

Ozone can also aggravate pre-existing respiratory disease. But EPA has already concluded that this effect is relatively small. For example, in assessing the benefits of the 8-hour ozone standard, EPA concluded that going from full national attainment of the1-hour ozone standard to full national attainment of the 8-hour ozone standard would reduce hospitalizations for asthma by only 0.6 percent.[56] The small health benefits of additional ozone reductions are the main reason EPA found the costs of the 8-hour standard would far exceed the benefits (see discussion below).

B. EPA Exaggerates the Number of People Exposed to Elevated Air Pollution

The Proposed Rule and the RIA vastly inflate the number of people exposed to air pollution in excess of EPA's standards. EPA's RIA for the Proposed Rule states that 111 million people live in counties that violate the 8-hour ozone standard and 70 million people live in counties that violate the annual $PM_{2.5}$ standard.[57] These numbers are strictly speaking true but also misleading. Readers would naturally assume that these numbers represent the actual number of people exposed to air pollution in excess of federal air pollution standards.

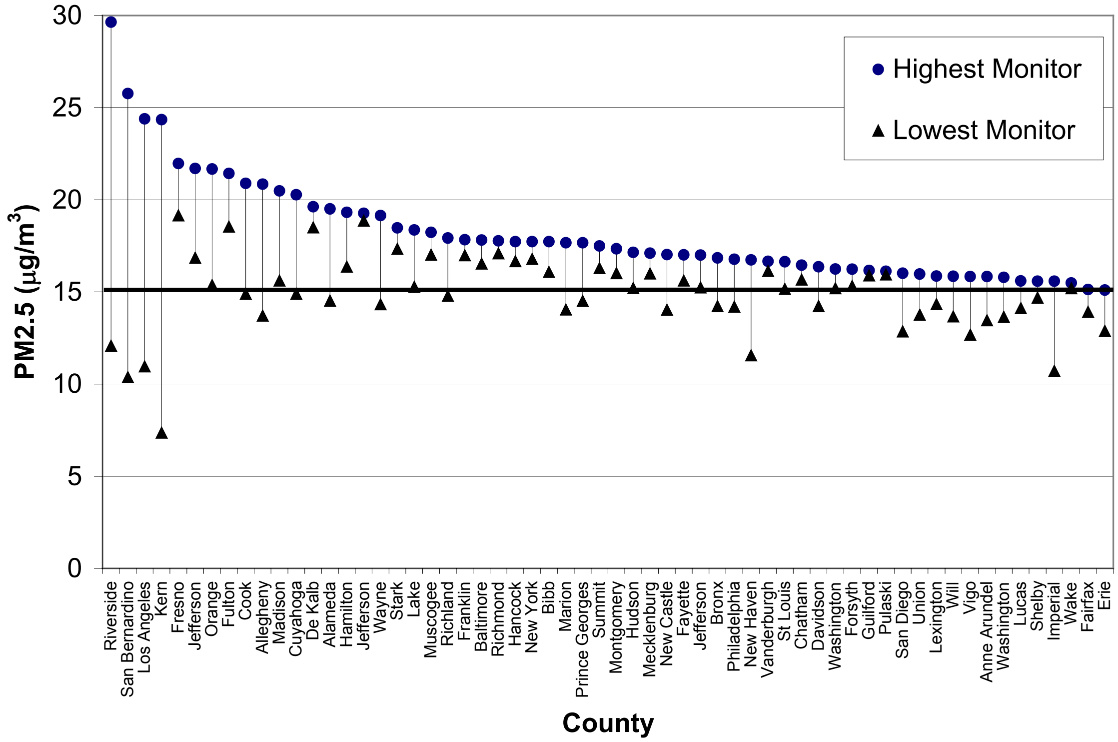
In fact, far fewer people are exposed to air pollution in excess of the standards, because only a portion of many non-attainment counties has air that exceeds the standards. Figure 6 displays annual-average $PM_{2.5}$ levels in counties that had at least two $PM_{2.5}$ monitoring locations for 1999-2001, and exceeded the annual $PM_{2.5}$ standard in at least one location. The graph shows the highest and lowest readings in each county, and the bold horizontal line marks the annual $PM_{2.5}$ standard of 15 $\mu g/m^3$. Of the 60 counties in the graph, 29 had at least one location with $PM_{2.5}$ below the standard. A more detailed geographic analysis would be necessary to determine the size of EPA's overestimate, but EPA has no doubt inaccurately counted millions and perhaps tens of millions of people as being

[56] EPA, "National Ambient Air Quality Standards for Ozone: Proposed Decision," *Federal Register*, December 13, 1996, pp. 65715-65750.

[57] RIA at 2-1.

exposed to excessive levels of $PM_{2.5}$ (by EPA's standards) when in fact they are not. The magnitude of the overestimate will increase with time, as $PM_{2.5}$ levels decline and more locations in non-attainment counties attain the standard.

Figure 6. Range of Annual-Average $PM_{2.5}$ Readings for Counties with Two or More Monitoring Locations, 1999-2001



Source: Based on analysis of national monitoring data downloaded from EPA's AIRData web site, www.epa.gov/aqspubl1/select.html. All locations with data for 1999-2001 were included.

EPA's overestimate of pollution exposure is far worse for ozone, because ozone levels vary more from place to place in counties. For example:

- Half the ozone monitoring locations in Los Angeles County comply with the 8-hour ozone standard. These areas are the most densely populated in the county and therefore probably include more than half of the county's roughly 9.5 million inhabitants.

- Only one rural location in San Diego County exceeds the 8-hour ozone standard. The rest of San Diego County's population of 2.8 million, 99.6 percent of the total county population, breathes air that complies with EPA's 8-hour ozone standard.

- Only one of Cook County's ozone monitors exceeds the 8-hour ozone standard. Almost all of the 5.4 million people in Cook County, where Chicago is located, breathe air that complies with the 8-hour ozone standard.

From these three counties alone we can remove at least 12 million people from EPA's claim regarding ozone exposure. But many other counties are in a similar situation, as shown for a selection of populous counties in Table 4. It is likely that EPA has overestimated by at least a factor of two the actual number of Americans exposed to ozone in excess of the 8-hour ozone standard.

Table 4. Percent of Ozone Monitoring Locations Complying with the 8-hour and 1-hour Ozone Standards in Selected Counties

		Percent of monitoring locations that comply with:	
County	**Major City**	**Federal 8-hour Ozone Standard**	**Federal 1-hour Ozone Standard**
Cook	**Chicago**	94%	100%
San Diego	**San Diego**	90%	100%
Maricopa	**Phoenix**	77%	100%
Ventura	**Ventura**	75%	86%
Queens	**New York**	75%	75%
Philadelphia	**Philadelphia**	50%	75%
Los Angeles	**Los Angeles**	50%	50%
Sacramento	**Sacramento**	43%	71%
Dallas	**Dallas**	33%	33%
Jefferson, KY	**Louisville**	33%	100%
San Bernardino	**San Bernardino**	8%	33%
Wake	**Raleigh**	0%	100%
Fresno	**Fresno**	0%	17%

Compliance with ozone standards is as of December 31, 2001. Based on analysis of national monitoring data downloaded from EPA's AIRData web site, www.epa.gov/aqspubl1/select.html. All locations with data for 1999-2001 were included.

Despite the 1-hour ozone standard being less stringent than the 8-hour standard, EPA states 116 million people live in areas exceeding the 1-hour ozone standard—slightly more than for the 8-hour standard.[58] This number is based on non-attainment areas, which generally cover several counties. As a result, this counting method includes not only counties that have some locations that attain the 1-hour standards (see Table 4), but also

[58] RIA at 2-92.

some whole counties that attain the standard, but that are in non-attainment regions. Several California counties fall into this category, including Orange, San Joaquin, San Francisco, and San Mateo.

EPA likewise exaggerates exposure to PM_{10} in excess of federal health standards. EPA states that 29 million people live in PM_{10} nonattainment areas. This is true, but once again misleading. Most monitoring locations in PM_{10} non-attainment areas don't exceed the PM_{10} standards. Based on data from individual monitoring locations, less than four percent of the nation's PM_{10} monitors exceed either the daily or annual PM_{10} standards, suggesting that at most a few million people are ever exposed to PM_{10} in excess of the standards.[59] Indeed, it is misleading for EPA to even include a discussion of PM_{10} in the context of the Proposed Rule, since existing requirements will eliminate PM_{10} exceedences before the Proposed Rule begins to have an effect on PM precursors.[60]

For carbon monoxide (CO), EPA states, "Over 22 million people currently live in the 13 non-attainment areas for the CO NAAQS."[61] While there may be 13 areas in technical non-attainment, there are only 3 monitoring locations in the entire country that still actually exceed the carbon monoxide standard.[62] Moreover, on-road and I/M vehicle emissions data show that automobile CO emissions have been declining about 10 percent per year due to fleet turnover to cleaner vehicles, suggesting that these few remaining locations will be in attainment soon.[63] In any case, EPA notes that nonroad diesel vehicles contribute only one percent of the CO inventory. Once again, it isn't clear why EPA would discuss CO health effects in the context of the Proposed Rule. The country has virtually eliminated excessive CO levels, the handful of remaining locations will be in attainment within no more than a few years, and the Proposed Rule could not have any effect on ambient CO levels.

In its summary of health effects related to the Proposed Rule, EPA engages in even more extreme exaggeration when it asserts, "Hundreds of millions of Americans currently live in counties with unhealthy air."[64] Since there are only about 280 million Americans, EPA is essentially claiming that *all* Americans breathe unhealthy air. This is a vast and

[59] Based national PM10 monitoring data downloaded from EPA's AirData web site, www.epa.gov/aqspubl1/select.html.

[60] The only places where this might not be true are areas heavily affected by windblown dust, but the Proposed Rule would provide no benefits to such areas in any case.

[61] RIA at 2-115.

[62] Based national CO monitoring data downloaded from EPA's AirData web site, www.epa.gov/aqspubl1/select.html. One of the three locations—Lynwood in Los Angeles—actually hasn't had an exceedance in the last two years. If EPA is counting all of the people in South Coast among those in a CO non-attainment area, then 15 million or so people are being counted based on a the monitor in Lynwood.

[63] Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline*.

[64] EPA, *Regulatory Announcement: Public Health and Environmental Benefits of EPA's Proposed Program for Low-Emission Nonroad Diesel Engines and Fuel* (Washington, DC: April 2003), http://www.epa.gov/nonroad/f03010.pdf.

irresponsible exaggeration by any measure, including EPA's already-misleading county and non-attainment-area summary methods.

C. Everyone Is At Risk

EPA states "Ozone can inflame and damage the lining of the lungs, which may lead to permanent changes in lung tissue, irreversible reductions in lung function if the inflammation occurs repeatedly over a long time period and a lower quality of life. People who are particularly susceptible to the effects of ozone include healthy children and adults who are active outdoors, people with respiratory disease, such as asthma, and people with unusual sensitivity to ozone."[65] And for particulates, "Children, the elderly, and people with cardiopulmonary disease, such as asthma, are most at risk from these health effects."[66]

Unqualified by notions of the dose making the poison, and the fraction of total respiratory or cardiovascular health risk attributable to pollution, statements like these lead readers to believe that virtually everyone is in serious danger if they live in a non-attainment county. Yet it is likely an exaggeration to say that even a few percent of people are affected by typical pollution levels in most non-attainment areas, and permanent lung damage would be far rarer still.

Ozone can indeed cause permanent damage to lung tissue if the exposures are high enough and repeated frequently enough. But where do such conditions still occur? Perhaps in a few areas in San Bernardino and the southern San Joaquin Valley. But even in these areas, with by far the worst ozone in the country,[67] ozone is still substantially lower than it was in southern California during the early to mid-1990s when the Children's Health Study failed to detect any relationship between ozone levels and children's rate of growth in lung function.[68]

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[65] Ibid.

[66] RIA at II-5.

[67] Based on 8-hour ozone data for 1999-2001 the worst 23 ozone monitoring locations are in California—specifically the eastern portion of South Coast and the southern portion of the San Joaquin Valley. These 23 areas exceeded the 8-hour standard an average of between 26 and 80 times per year. Atlanta, the next worst 8-hour area averaged 26 exceedances for 1999-2001, but this was a fluke due to uniquely high ozone levels in 1999. Based on 2000-2002 data, Atlanta averaged only about 15 exceedances. The only other locations outside California that average more than 20 8-hour ozone exceedances per year are a couple of locations in Houston and one in Tennessee. Ozone exceedances are based on 8-hour ozone data downloaded from EPA's AIRData web site, www.epa.gov/aqspubl1/select.html. For Atlanta data, see Figure 1 in J. Schwartz, *Proposed Rule to Implement the 8-Hour Ozone National Ambient Air Quality Standard, Public Interest Comment* (Arlington: Mercatus Center, July 2003), http://www.mercatus.org/pdf/materials/366.pdf.

[68] W. J. Gauderman, R. McConnell et al., "Association between Air Pollution and Lung Function Growth in Southern California Children," *Am J Respir Crit Care Med*, vol. 162, no. 4 Pt 1 (2000), pp. 1383-1390. A study of a second cohort, based on ozone levels from 1996-1999 reported roughly a 10 percent decrease in the growth rate of Peak Expiratory Flow Rate (PEFR) between the highest and lowest ozone communities, but no difference in other measures of lung function growth (W. J. Gauderman, R. McConnell et al.,

Instead of implying everyone is at risk of serious harm while maintaining plausible deniability by being vague, EPA should strive to give the public realistic estimates of risk. Both for this rule and in general, EPA should avoid alarmism and put risk in context with tables showing the estimated baseline risk of various health conditions and the fraction estimated to be attributable to air pollution. A recent study that did this for PM concluded that completely eliminating *all* power plant emissions (which account for roughly 20 to 40 percent of eastern $PM_{2.5}$ through their sulfur dioxide emissions) would reduce asthma-related hospital admissions by 1.1 percent, chronic bronchitis by three percent, and lower-respiratory symptoms by 6.6 percent.[69] These small changes from large pollution reductions are a far cry from the public health crisis suggested by EPA's quotes cited above. And to generate even these small percentages, the consultants in that study had to take the same selective and uncritical approach to the epidemiologic literature as EPA has for the Proposed Rule. As noted earlier, EPA also estimated a tiny change in the baseline rate of asthma hospitalizations from attaining the 8-hour ozone standard, likewise indicating that air pollution at current levels is a minor player in respiratory and cardiovascular health.[70]

VI. Air Quality Science and Policy

A. The 8-hour Ozone Standard Will Cause Net Harm to Americans

All estimates published to date, including EPA's own cost-benefit analysis for the 8-hour ozone standard, have concluded that requiring nationwide attainment of the 8-hour standard would cause net harm to the American public. EPA's Regulatory Impact Analysis for the 8-hour standard concluded: "Quantifiable net benefits for full attainment of the ozone standard are estimated to range from negative \$1.1 to negative \$8.5 billion"—in other words, after accounting for the value of all expected health and welfare benefits and the estimated costs of attaining the standard, EPA concluded that attaining the 8-hour ozone standard nationwide would entail billions of dollars per year in net costs to the American public.[71]

The real situation is far worse than this for two reasons. First, 90 percent of the estimated health benefits are due to projected reductions in mortality. However, the link between current ozone levels and increased mortality is tenuous, suggesting that EPA's claimed

"Association Between Air Pollution and Lung Function Growth in Southern California Children," *Am J Respir Crit Care Med*, vol. 166 (2002), pp. 76-84).

[69] Abt Associates, *The Particulate-Related Health Benefits of Reducing Power Plant Emissions* (Bethesda, MD: October 2000), http://cta.policy.net/fact/mortality/mortalityabt.pdf.

[70] And this estimate appeared only in the proposed decisions, but not in later rulemaking documents related to the 8-hour standard.

[71] EPA estimated the incremental benefits of full national attainment of the 8-hour ozone standard (beyond the 1-hour standard) would range from \$1.5 to \$8.5 billion per year. Yet EPA's "central estimate" for incremental attainment costs was \$9.6 billion—greater than even the high end of EPA's benefit estimate. EPA, "Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule".

mortality benefits might not materialize.[72] Other aspects of EPA's benefit assessment also indicate that, in real terms, the marginal health benefits of the 8-hour NAAQS would be modest at best. For example, EPA concluded that going from full national attainment of the 1-hour ozone NAAQS to full national attainment of the 8-hour ozone NAAQS would reduce hospital admissions for asthma by just 0.6 percent.[73] As EPA acknowledged, only a small fraction of all respiratory distress is due to air pollution in the first place, so reducing air pollution will have a small effect on overall respiratory morbidity.

Second, EPA made some unwarranted assumptions that caused it to substantially underestimate the costs of attaining the 8-hour standard. For example, without any empirical basis, EPA assumed that no emission control measures would cost more than $10,000 per ton of pollution reductions.[74] After a more realistic assessment of the costs of full attainment of the ozone standard, a number of economists have concluded the total cost would likely range from $54 billion to hundreds of billions of dollars per year.[75] Even taking EPA's cost-benefit estimates at face value, the 8-hour standard is a harmful policy. Based on more realistic assessments of likely attainment costs, requiring attainment of the 8-hour ozone standard is certain to cause substantial net harm to public health.

In addition to the costs of regulations, pollution reductions themselves can ironically sometimes cause offsetting harm.[76] For example, although it has harmful respiratory effects, ground-level ozone, like stratospheric ozone, has the beneficial effect of reducing people's exposure to the sun's ultra-violet (UV) light. Reducing ground-level ozone therefore also increases harm due to solar-UV exposure. EPA performed an internal analysis estimating that attaining the 8-hour ozone standard would cause an additional 696 non-melanoma skin cancer cases each year. EPA never officially made this analysis

[72] J. I. Levy, T. J. Carrothers et al., "Assessing the Public Health Benefits of Reduced Ozone Concentrations," *Environmental Health Perspectives*, vol. 12 (2001), pp. 9-20.

[73] EPA, "National Ambient Air Quality Standards for Ozone: Proposed Decision."

[74] EPA, *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule* (Washington, DC: July 17 1997), www.epa.gov/ttn/oarpg/naaqsfin/ria.html.

[75] S. E. Dudley, *Comments on the U.S. Environmental Protection Agency's Proposed National Ambient Air Quality Standard for Ozone* (Arlington, VA: Regulatory Analysis Program, George Mason University, March 12, 1997), www.mercatus.org/research/RSP19972.htm, R. Lutter, *Is EPA's Ozone Standard Feasible?* (Washington, DC: AEI-Brookings Joint Center for Regulatory Studies, December 1999), www.aei.brookings.org/publications/reganalyses/reg_analysis_99_06.pdf, A. J. Krupnick, "The Proposed National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM) and Ozone (Panel 1)," U.S. Senate Subcommittee on Clean Air, Wetlands, Private Property and Nuclear Safety, Committee on Environment and Public Works (Washington, DC: Resources for the Future, April 24, 1997), www.rff.org/testimony/remarks/naaqs1.htm, EPA, *Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule*.

[76] See Dudley and Gramm, *Risk Analysis*, Vol. 17, No 4, 1997 and Dudley, *Pace Environmental Law Review,* Vol. 16, No 1, Winter 1998.

public and did not consider it in setting the 8-hour ozone standard.[77] The U.S. Department of Energy also estimated that attaining the 8-hour ozone NAAQS could increase by several thousand the annual number of cases of cataracts as well as cause some additional deaths each year due to melanoma skin cancer.[78]

In May 1999, the United States Court of Appeals for the District of Columbia Circuit remanded the 8-hour ozone NAAQS to EPA to consider the potential beneficial health effects of ozone pollution in shielding the public from the "harmful effects of the sun's ultraviolet rays."[79] EPA published its decision in January 2003, concluding that the effect of changes in ground-level ozone on ultraviolet exposure "is too uncertain at this time to warrant any relaxation in the level of public health protection previously determined to be requisite to protect against demonstrated direct adverse respiratory effects of exposure to O_3 in the ambient air. Further, it is the Agency's view that associated changes in UV–B radiation exposures of concern, using plausible but highly uncertain assumptions about likely changes in patterns of ground-level ozone concentrations, would likely be very small from a public health perspective."[80]

EPA does not mention its internal analysis of non-melanoma skin cancer and ozone in its decision, and is here applying a double standard on the level of certainty necessary to accept scientific evidence for regulatory purposes. For example, as noted earlier, most of the benefits EPA claims for the 8-hour standard are due to presumed reductions in mortality. The uncertainty in these mortality benefits is at least as great if not greater than the uncertainties surrounding estimates of the relationship between UV exposure, ground-level ozone, and health. Nevertheless, this did not deter EPA from claiming mortality-reduction benefits due to lower ozone levels. Likewise, as will be discussed below, EPA is willing to tolerate an air pollution planning and regulatory process founded on emission inventories known to contain serious inaccuracies and biases.

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[77] The EPA analysis is now posted at aei.brookings.org/admin/pdffiles/php9v.pdf. It suggests that average summer ozone levels would need to be reduced by from one to a few ppb in most 8-hour non-attainment areas in order to attain the standard. Also see, R. Lutter and H. Gruenspect, "Assessing Benefits of Ground Level Ozone: What Role for Science in Setting National Ambient Air Quality Standards?" *Tulane Environmental Law Journal* (Winter 2001), pp. 85-96.

[78] For example, the U.S. Department of Energy estimated that a 10 ppb reduction in average ground-level ozone would result in 25 to 50 additional deaths each year from melanoma skin cancers and an additional 13,000 to 28,000 additional cases of cataracts—or health costs totaling $0.29 to $1.1 billion per year. Based on the internal EPA analysis, this represents about 3 or 4 times the ozone reduction that would be necessary to comply with the 8-hour standard in the vast majority of 8-hour non-attainment areas. On the other hand, ozone precursor reductions will go well beyond those necessary for attainment in many areas, because many of the reductions are national in scope and very stringent, for example, EPA's Tier 2 and heavy-duty rules for on-road vehicles, the NOx SIP call, and the proposed rule for off-road mobile source NOx emissions (see discussion of these rules below). Thus, the mean ozone reduction assumed by DOE could be realistic. On the DOE estimates, see Lutter and Gruenspecht, "Assessing the Benefits of Ground Level Ozone."

[79] F. 3d 1027 (D.C. Circuit 1999).

[80] EPA, "National Ambient Air Quality Standards for Ozone: Final Response to Remand; Final Rule," *Federal* Register, January 6, 2003, pp. 614-45.

The evidence is overwhelming that requiring attainment of the 8-hour ozone NAAQS in its current form would cause net harm to the American public. EPA should ideally have proposed a standard more likely to lead to net benefits. Since EPA is unwilling to abandon the current standard, the Agency faces some difficult choices, as discussed in the next section.

B. EPA's NOx Strategy Is Likely to Increase Ozone Levels for Tens of Millions

A major goal of the Proposed Rule and of the other stringent rules EPA has recently promulgated for other mobile sources and electricity boilers is to speed attainment of the 8-hour ozone standard. Because NOx is a minor PM precursor in the eastern United States, and because parts of California are the only major $PM_{2.5}$ non-attainment areas in the west, it is fair to say that EPA's rationale for major national NOx reduction rules rests almost solely on the goal reducing 8-hour ozone levels.

But these NOx reductions are a risky strategy. Air quality scientists have been aware for some time that ozone levels are higher or the same on weekends in most areas of the United States, even though NOx emissions decline 10 to 40 percent on weekends due to substantial reductions in diesel truck and off-road diesel equipment activity. The phenomenon is known as the "weekend effect" and scientists consider it a telltale sign that NOx reductions might not be effective in reducing ozone, given the current mix of VOC and NOx in most metropolitan areas.[81]

For example, out of almost 1,200 ozone-monitoring locations around the U.S., weekend 8-hour ozone is higher at 35 percent of sites, roughly the same at 60 percent of sites, and lower at only 5 percent of sites.[82] Yet NOx monitoring data indicate that NOx declines 10 to 40 percent on weekends. VOC emissions also decline on weekends, but much less so than NOx. As a result, the VOC/NOx ratio increases.

This weekend change in emissions can be thought of as a natural experiment to test out a pollution control strategy. Every weekend, NOx, an ozone precursor, declines substantially, yet ozone levels generally stay the same or increase. Based on a combination of modeling and empirical observations, the cause appears to be that when the VOC/NOx ratio falls below about 10, the chemistry of ozone formation enters a

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[81] A detailed discussion of the nature and explanation for the weekend effect can be found in a series of seven peer-reviewed research papers in the July 2003 issue of the *Journal of the Air and Waste Management Association*. A summary of the research can be found in D. R. Lawson, "The Weekend Effect—the Weekly Ambient Emissions Control Experiment," Environmental Manager, July, 2003). The same issue of Environmental Manager has a companion article by staff from the California Air Resources Board, which disputes the conclusions of the independent researchers who performed the various studies. See B. E. Croes, L. J. Dolislager et al., "The O3 'Weekend Effect' and NOx Control Strategies: Scientific and Public Health Findings and Their Regulatory Implications," Environmental Manager, July, 2003.

[82] J. M. Heuss, D. F. Kahlbaum and G. T. Wolff, "Weekday/Weekend Ozone Differences: What Can We Learn from Them," *Journal of the Air & Waste Management Association*, vol. 53, no. 7 (2003), pp. 772-788.

regime referred to as "VOC limited." In this situation, NOx reductions don't reduce ozone; at lower VOC/NOx ratios, NOx reductions can even increase ozone.[83]

Because VOC emissions have been declining more rapidly than NOx during the last two decades, the VOC/NOx ratios in most urban areas have been declining, and there is evidence that the detrimental effect of NOx reductions has been increasing in magnitude and in geographic scope.[84] This research makes a strong case that all of California's large metropolitan areas—South Coast, San Diego, and the San Francisco Bay Area—are now VOC limited, as are the urban cores of Central Valley cities.[85] Other cities, such as Philadelphia and Chicago are in a similar situation.[86] The fact that ozone levels are the same or higher on weekends at almost all monitoring locations, despite lower NOx emissions on weekends, suggests that most areas of the U.S. are at best insensitive to NOx reductions on the order of 10 to 40 percent.

EPA and the California Air Resources Board (CARB) dispute this interpretation of the weekend effect. CARB researchers published an opinion article in the trade magazine *Environmental Manager* asserting that there isn't any evidence to support the interpretation that NOx reductions are detrimental to ozone formation.[87] However, CARB's submission to the special issue of *Journal of the Air and Waste Management Association* (*JAWMA*) on the weekend effect was rejected by the journal's referees.[88] In the RIA, EPA contends, "There are other hypotheses for the cause of the 'weekend effect,'" besides a detrimental effect of NOx emissions. Ironically, the only support EPA offers for this assertion is the CARB paper that was later rejected by *JAWMA*.[89]

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[83] Lawson, "The Weekend Effect--the Weekly Ambient Emissions Control Experiment," N. Carslaw and D. Carslaw, "The Gas-Phase Chemistry of Urban Atmospheres," *Surveys in Geophysics*, vol. 22 (2001), pp. 31-53, J. H. Seinfeld, "Urban Air Pollution: State of the Science," *Science*, vol. 243 (1989), pp. 745-752.

[84] See, for example, L. C. Marr and R. A. Harley, "Spectral Analysis of Weekday-Weekend Differences in Ambient Ozone, Nitrogen Oxide, and Non-Methane Hydrocarbon Time Series in California," *Atmospheric Environment*, vol. 36 (2002), pp. 2327-2335, L. C. Marr and R. A. Harley, "Modeling the Effect of Weekday-Weekend Differences in Motor Vehicle Emissions on Photochemical Air Pollution in Central California," *Environmental Science & Technology*, vol. 36 (2002), pp. 4099-4106, Lawson, "The Weekend Effect--the Weekly Ambient Emissions Control Experiment."

[85] Marr and Harley, "Modeling the Effect of Weekday-Weekend Differences in Motor Vehicle Emissions on Photochemical Air Pollution in Central California," Marr and Harley, "Spectral Analysis of Weekday-Weekend Differences in Ambient Ozone, Nitrogen Oxide, and Non-Methane Hydrocarbon Time Series in California," B. K. Pun and C. Seigneur, "Day-of-Week Behavior of Atmospheric Ozone in Three U.S. Cities," *Journal of the Air & Waste Management Association*, vol. 53, no. 7 (2003), pp. 789-801.

[86] Pun and Seigneur, "Day-of-Week Behavior of Atmospheric Ozone in Three U.S. Cities."

[87] B. E. Croes, L. J. Dolislager et al., "The O3 'Weekend Effect' and NOx Control Strategies: Scientific and Public Health Findings and Their Regulatory Implications," *Environmental Manager* (July 2003), pp. 27-35.

[88] L. C. Larsen, "The Ozone Weekend Effect in California: Evidence Supporting NOx Emissions Reductions," *Journal of the Air & Waste Management Association* (Submitted 2003).

[89] RIA at 2-107, and endnote 278 at 2-141.

The mainly VOC-focused pollution control strategy of the last two to three decades has been relatively successful in mitigating the worst ozone problems and bringing most areas into compliance with the 1-hour ozone standard. However, the results of weekend effect research suggest that reducing 8-hour ozone levels from their current relatively moderate levels in most 8-hour non-attainment areas down to the very stringent requirements of the 8-hour standard may be difficult over much of the U.S.

For example, recent modeling results for California and the eastern U.S. suggest that NOx would have to be reduced 70 to 90 percent in order to attain the 8-hour standard at all monitoring locations in most non-attainment areas.[90] These studies concluded that more modest NOx reductions would increase ozone in some areas, including New York City, Philadelphia, and Chicago. The modeling also concluded that VOC reductions were effective in reducing ozone in VOC-limited urban core areas, such as New York City, Philadelphia and Chicago, but less so in other areas, where very large VOC reductions would be needed to produce substantial ozone reductions. Indeed, despite EPA's dislike of the NOx-disbenefit interpretation of the weekend effect, the Agency's own modeling for its Tier 2 automobile regulation predicted that the NOx reductions from Tier 2 would make ozone worse in many areas, including much of Texas and California.[91]

With its stringent NOx reduction rules for mobiles sources and the NOx SIP Call, EPA has embarked on a course of large national reductions in NOx emissions. The nation runs a serious risk that this policy will backfire and make ozone worse in some areas, particularly in urbanized areas where most people live, because they are the most likely to be VOC limited. Indeed, after a decade of spectacular ozone reductions in California's South Coast Air Basin, ozone levels have been flat or perhaps even rising since 1999, despite the fact that precursor NOx and VOC emissions have been declining. While weather or other random factors might at least partially explain the flattened trend, weekend effect research shows that the South Coast has been becoming more and more VOC limited and NOx reductions are becoming more and more detrimental to ozone reduction.

The RIA is remiss in glibly dismissing the NOx-disbenefit interpretation of the weekend effect. It is understandable that EPA would see the weekend effect and recent ozone modeling as a threat. If NOx reductions really are detrimental to further progress on ozone, it would be a major embarrassment for EPA. But ignoring the issue won't make it go away. The current state of the science provides substantial evidence to support NOx reductions as the culprit in increased weekend ozone levels, and little evidence to support

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[90] S. Reynolds and C. L. Blanchard, *Understanding the Effectiveness of Precursor Reductions in Lowering 8-Hour Ozone Concentrations in the Eastern United States* (San Rafael, CA: Envair, June 9, 2003), S. Reynolds, C. L. Blanchard and S. D. Ziman, "Understanding the Effectiveness of Precursor Reductions in Lowering 8-Hr Ozone Concentrations," *Journal of the Air & Waste Management Association*, vol. 53, no. 2 (2003), pp. 195-205. These studies concluded that peak 1-hour ozone levels are more easily reduced than 8-hour levels, consistent with actual experience during the last decade.

[91] Abt Associates, *Tier II Proposed Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results* (Research Triangle Park, NC: EPA, April 1999).

other interpretations. EPA needs to take this issue seriously and assess whether continued NOx reductions are a fruitful course for further progress on ozone. As noted in a Mercatus Public Interest Comment on EPA's 8-hour ozone implementation rule, a strategy of additional near-term VOC reductions, with NOx reductions delayed for several years, may be more likely to reduce ozone in most areas and avoid increasing it anywhere.[92]

C. EPA's Emission Inventory Projections Don't Appear to Be Plausible

Inaccurate emission inventories are an ongoing and serious problem for the credibility of regulatory air quality modeling and SIP control strategies.[93] The RIA appears to rely on implausible NOx and VOC inventories that might render the model output meaningless for substantive air quality projections.

According to emission inventory projections in the RIA, without the Proposed Rule but including other already-promulgated rules, total national NOx emissions will decline 37 percent between 1996 and 2030. With the Proposed Rule, NOx would decline 40 percent. But based on a conservative reading of the actual requirements of proposed rules for just the major NOx sources, national NOx emissions will decline by at least 50 percent during the next 20 years or so—even when calculated from the current year as a baseline, rather than the higher 1996 baseline used for the RIA.

Table 5 displays EPA's 2001 NOx inventory breakdown by source category. The third column lists the percent reduction in emissions required by existing regulations. The right column gives the sources for the percentage reductions. Most of these requirements will achieve their full benefits sometime after 2020, when fleet turnover has replaced the existing fleet with vehicles/engines meeting the new standards. The only exception is the NOx SIP Call, which comes into effect in 2004.

[92] Schwartz, *Proposed Rule to Implement the 8-Hour Ozone National Ambient Air Quality Standard, Public Interest Comment*.

[93] Ibid.

Table 5. Change in NOx Inventory Between Now and 2020s Based on Existing Federal Requirements

Major Controlled Sources	Percent of 2001 EPA Inventory[94]	Percent Reduction	Source
On-Road Trucks	17.5%	90%	95
On-Road Gasoline	19.4%	80%	96
Non-Road Diesel	7.1%	25%	97
Locomotives	4.5%	66%	98
Marine Vessels	4.5%	80%	99
Stationary Combustion	33.7%	25%*	100
All Other	13.3%	--	101
Overall	**100%**	**48%**	
Nonroad RIA		**37%**	102

*The NOx SIP Call requires a 60% reduction but only during May-September, which annualizes to a 25% reduction. Thus, the overall percentage NOx reduction would be 60% during the ozone season, but 48% on annual basis.

The estimate of a 50 percent reduction is probably conservative because there are likely NOx reduction rules on the books for other NOx sources not considered here.

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[94] The 2001 inventory is the version EPA released in February 2003 and was downloaded from EPA's emission inventory web page, http://www.epa.gov/ttn/chief/trends/.

[95] EPA, *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements*. See page II-125.

[96] Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline*.

[97] RIA at 3-66. These are the reductions from Tier 3 requirements in 2030. Reductions would be somewhat greater in 2020.

[98] EPA, *Regulatory Announcement: Environmental Benefits of Emission Standards for Locomotives* (Washingon, DC: December 1999), http://www.epa.gov/otaq/regs/nonroad/locomotv/frm/42097049.pdf.

[99] EPA, *Regulatory Announcement: Emission Standards for New Nonroad Engines* (Washingon, DC: September 2002), http://www.epa.gov/otaq/regs/nonroad/2002/f02037.pdf.

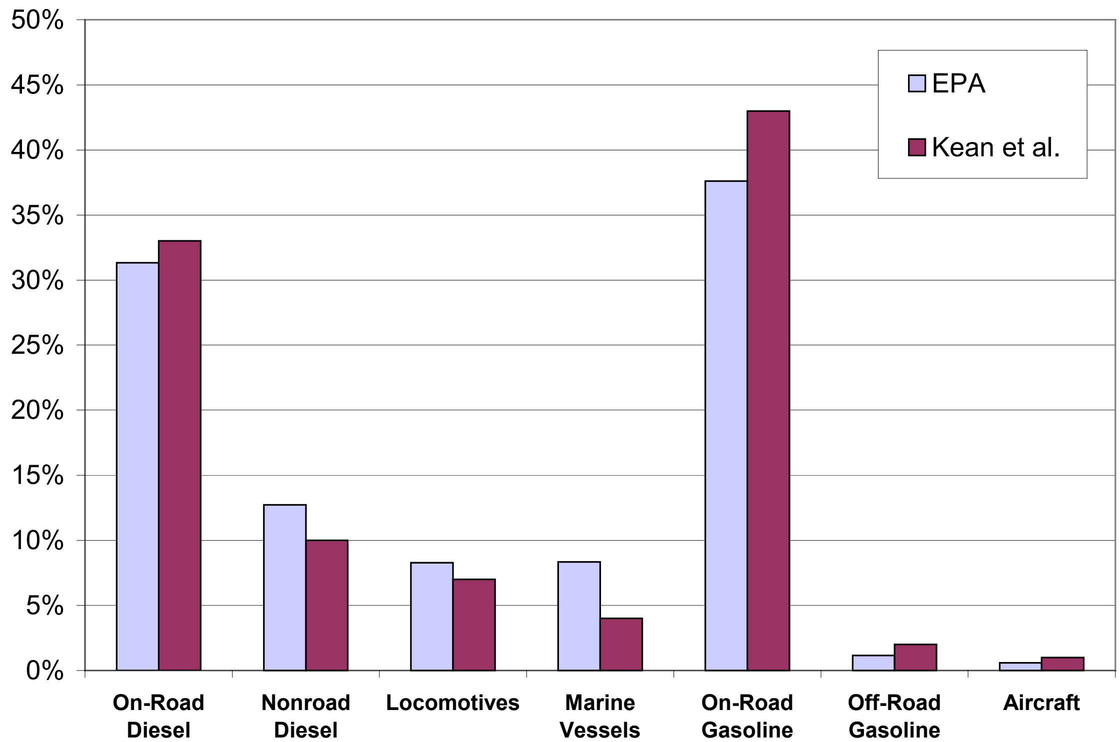
[100] EPA, *Addendum to the Regulatory Impact Analysis for the NOx SIP Call, FIP, and Section 126 Petitions*.

[101] Assumed to be zero for this exercise. In reality, various existing state and federal regulations will reduce NOx emissions from other sources as well.

[102] Baseline reduction in total NOx inventory calculated from data in RIA Table 3.2-3, page 3-31.

Furthermore, recent attempts at generating "real-world" emission inventories based on on-road emission factor measurements and estimates of fuel consumption suggest that on-road vehicles may contribute a somewhat greater fraction of the mobile-source NOx inventory than the EPA inventory suggests (see Figure 7). Since on-road sources will be much more heavily controlled than nonroad sources under existing requirements, the baseline reduction in NOx emissions will likely be even greater than suggested above.

Figure 7. Comparison of Source Contributions to the 1996 Mobile Source NOx Inventory Based on EPA Emission Models and Kean et al. "Real-World" Fuel-Based Estimate



Sources: A. J. Kean, R. F. Sawyer and R. A. Harley, "A Fuel-Based Assessment of Off-Road Diesel Engine Emissions." Kean et al. estimate the national mobile source NOx inventory for 1996. This EPA inventory for 1996 was released in February 2003, and is based on output from EPA's latest mobile emission factor models—NONROAD2002 and MOBILE6. It is substantially changed from the EPA inventory available when Kean et al.'s paper was released, and is now much closer to the Kean et al. inventory.

The VOC inventory trend is similarly implausible. For example, the RIA baseline inventory has total VOC emissions declining just 25 percent between 1996 and 2020, decreasing to 17 percent in 2030. This is probably due to EPA's continuing underestimate of the contribution of gasoline vehicles to the VOC inventory. EPA's inventory has only about 40 percent of VOC coming from gasoline vehicles. But based on source apportionment studies, gasoline-vehicles account for 50 to 75 percent of anthropogenic VOC emissions, with urban and suburban areas more likely to be at the high end of that range.[103] Tier 2 will reduce total emissions from gasoline vehicles by more than 80 percent during the next 20 years or so, suggesting that actual VOC reductions will be substantially larger than the RIA forecasts.[104]

While the CO inventory isn't affected by the Proposed Rule, CO contributes to ozone formation. CO is much less reactive than VOC, but since CO emissions are several times greater than VOC emissions, CO can account for as much as 10 to 20 percent of ozone formation. A recent MOBILE6 validation study found that MOBILE6 overestimates automobile CO emissions by as much as a factor of two.[105] Other consistency checks also indicate significant problems with MOBILE6. For example, MOBILE6 projects fleet-average CO emissions of 11 grams/mile in 2005 and 5 grams/mile in 2025. But fleet-average data from I/M programs, tunnels, and remote sensing in several cities indicate that CO emissions ranged from about 4 to 6 grams/mile during 1999-2002.[106] While these on-road data don't include cold-start emissions, the discrepancy appears much too large to be accounted for by missing cold-start emissions.[107]

Thus, EPA's future-year emission inventories appear to include too much of all three pollutants. It's not clear how a more realistic inventory would change the results of air quality modeling. Regardless, the modeling results in the RIA should be taken with a particularly large grain of salt. Furthermore, the desirability of a given emission control program depends on baseline assumptions about future air quality. Therefore, to put the Proposed Rule and other EPA policies on a firm foundation, EPA should develop more realistic estimates of future air quality.

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[103] J. G. Watson, J. C. Chow and E. M. Fujita, "Review of Volatile Organic Compound Source Apportionment by Chemical Mass Balance," *Atmospheric Environment*, vol. 32 (2001), pp. 1567-1584, E. M. Fujita, D. E. Campbell et al., *Weekend/Weekday Ozone Observations in the South Coast Air Basin: Volume II - Analysis of Air Quality Data* (Reno, Nevada: Desert Research Institute, April 2002), ftp://ftp.arb.ca.gov/carbis/aqd/weekendeffect/final_wewd_5_1/nrelp3v2f.pdf, E. M. Fujita, J. G. Watson and J. C. Chow, "Receptor Model and Emissions Inventory Source Apportionments of Nonmethane Organic Gases in California's San Joaquin Valley and San Francisco Bay Area," *Atmospheric Environment*, vol. 29 (1995), pp. 3019-3035.

[104] Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline,* Giannelli, Gilmore et al., *Sensitivity Analysis of MOBILE6.0*.

[105] C. Tran, R. Chi and A. Pollack, *Validation of the U.S. EPA MOBILE6 Highway Vehicle Emission Factor Model* (Atlanta: Coordinating Research Council, 2002), http://www.crcao.com/reports/recentstudies00-02/ENVIRON%20Final%20report_CRC%20E-64_81602.pdf.

[106] Schwartz, *No Way Back: Why Air Pollution Will Continue to Decline*.

[107] And emission models appear to overestimate cold-start emissions as well. Brett C. Singer et al., "A Fuel-Based Approach to Estimating Motor-Vehicle Cold-Start Emissions," *Journal of the Air and Waste Management Association*, vol. 49 (1999), pp. 125-135.

VII. Economic Issues

A. EPA Organizes Its Regulatory Cost Accounting Around How Quickly Affected Firms Can 'Recover' Costs From Customers, *Not* on Society's Opportunity Cost

EPA's regulatory cost accounting explicitly rejects basing cost estimates on the concept of "opportunity cost."[108] Yet, a regulatory agency seeking to improve society's net wealth (by imposing regulations that provide more benefits than costs) *should* base its perspective on opportunity cost. In other words, EPA should address this question: "What would be the most highly valued alternative use, *from society's point of view*, of the economic resources needed to implement this proposed regulation?" Since EPA does not ask—let alone attempt to answer—that basic question, the Agency's cost accounting goes awry from the very beginning.

In place of society's opportunity cost, EPA's cost accounting revolves around how quickly regulated firms will "recover"—*via* higher prices from consumers—costs for R&D, redesign, certification and other "fixed' costs" on certified units. (In EPA's cost accounting lexicon, "fixed cost" and "variable cost" have different meanings than those found in a standard "Principles of Economics" textbook. In standard "Economics 101" "fixed cost" applies to the productive inputs that, from the firm's perspective, are (relatively) fixed in the short run and "variable costs" applies to those inputs that are (relatively) variable during that same (somewhat ambiguous) time period. In contrast, EPA regulatory cost accountants apply "fixed cost" to those resources that regulated firms must purchase prior to (the deadline for) implementation of the standard. These costs do not vary with subsequent production. For instance, $10,000 spent to certify a specific engine as compliant with the regulation remains at $10,000 regardless of whether a firm ultimately produces and sells 5,000 or 50,000 or 500,000 engines. In contrast, "variable costs" in the EPA lexicon refers to those resources that *can* vary after the deadline for implementation, including certain capital costs.[109]) Had EPA based its cost

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[108] "In general, in environmental economics, it would be more conventional to simply count the total cost of the program (i.e., opportunity costs) in the year they occur. However, this approach would not directly estimate a per unit cost since fixed costs occur prior to implementation of the standards and, therefore, there are not yet any units certified as complying with the new standards to which the fixed costs can be attributed" (RIA at 6-3). Even if the reader accepts the usefulness of attributing "fixed costs" to "certified units," the EPA still could have produced estimates based on opportunity cost. Doing one does not preclude doing the other. In addition, the EPA has been advised on previous rulemakings that per unit cost estimates have limited value for decision-makers. For instance, Dr. John Graham of the U.S. Office of Management and Budget, in a September 24, 2001 "prompt letter" to the Agency with respect to (a then) proposed regulation affecting nonroad large-spark ignition engines, wrote: "EPA's analysis is focused on evaluating the direct (engineering) costs and the projected emission reductions for the selected proposal. In this respect, the analysis provides a useful 'accounting' of the directly measurable effects of the proposed standard but does not provide a benefit/cost analysis integral to the decision-making process." EPA's analysis of the Proposed Regulation under discussion has the same focus, "not integral to the decision-making process," as does the earlier regulation evaluated by Dr. Graham.

[109] The following passage from the *RIA* (at 6-3) may help the reader understand EPA's notion of "fixed cost" *vis-à-vis* "variable cost": "For engine variable cost (i.e., emission control and associated hardware),

accounting on society's opportunity cost, "recovering" costs would be a meaningless phrase. Society's members—consumers, workers, investors—ultimately, bear the regulation's costs and *never* "recover" them.

Under EPA's regulatory cost accounting, what the Agency terms "social costs" do not begin to appear until the regulated firms can start "recovering" fixed costs (as EPA defines "fixed costs"). Under that methodology, society can spend several million dollars on R&D, engine redesign, certification and the like without incurring a single penny of social cost—as EPA explicitly claims (see Table 6). This is an absurd result since society foregoes the opportunity cost of all the resources encompassed by EPA's notion of "fixed costs" when those expenditures occur, *not* years later when regulated firms begin to "recover" these costs from customers *via* higher prices. If not for the proposed regulation, the "fixed cost" resources would have gone into the manufacture of other goods and services, making the prices of those alternatives lower than they would otherwise be. The loss of those other goods and services is better measured by the dollar value of the annual expenditures spent on the "fixed costs," *not* zero!

Table 6. EPA Estimates of "Fixed Cost" Expenditures and Social Cost: 2003–2006 (thousands of 2001 dollars)

Year	"Fixed Cost" Expenditures[a]	Social Cost[b]
2003	$7,201	$0.0
2004	$7,201	$0.0
2005	$7,201	$0.0
2006	$29,328	$0.0

[a]Source: RIA, 6-12, 6-16, 6-19.

[b]Source: RIA, 10-14.

EPA's regulatory cost accounting also asserts that the direct engineering compliance costs and total social costs are almost identical, with compliance costs actually *exceeding* total social costs by a relatively tiny amount (see Table 7).

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we first estimate the cost per piece of technology. Emission control hardware costs tend to be directly related to engine characteristics—e.g., exhaust emission control devices are sized according to engine displacement so that costs vary by displacement; fuel injections systems vary in cost according to how many fuel injectors are required so that costs vary by number of cylinders. Therefore, we are able to determine a variable cost equation as a function of engine displacement or as a function of the number of cylinders." Note that, in this passage, the EPA treats "engines" as fixed costs but another form of "hardware"—fuel injection systems—as "variable" because the firm can increase or decrease the number of such systems to produce a compliant engine in the year that the engine will be produced and (hopefully) sold.

Table 7. EPA Estimates of Annual National Engineering Compliance Costs and Social Costs for the Proposed Rule: Selected Years. (Millions of 2000 dollars)

Year	Engineering Compliance Costs	Total Social Costs
2004	$0	$0
2010	$262.02	$262.01
2015	$1,260.74	$1,260.62
2020	$1,366.79	$1,366.66
2025	$1,395.98	$1,395.83
2030	$1,509.77	$1,509.61

Source: RIA, 10-14.

These results are highly unlikely on two counts. First, prestigious outside reviewers of previous EPA regulatory cost studies have repeatedly counseled the Agency that "indirect" market adjustments can—and usually do—*add* social costs that equal or *exceed* the direct compliance costs associated with environmental regulations.[110] Hence, direct engineering compliance costs and total social costs should not be nearly identical. Second, by claiming that compliance costs actually exceed total social costs (albeit by less than one hundredth of one percent for years 2010 and later), EPA implies that the indirect effects provide net benefits instead of net costs. In other words, EPA asserts that Dr. Richard Schmalensee and other distinguished SAB members are wrong on: (1) the relative size of the indirect economic effects; and also on (2) the direction of the indirect effects.

B. How Basing Regulatory Cost Accounting on Society's Opportunity Cost Can Alter EPA's Estimates

EPA, to its credit, recognizes that the proposed regulation involves both costs and benefits that will unfold over many years. Therefore, to compare costs and benefits, EPA (or other evaluator of the proposed regulation) must rely on net present values (NPVs), which in turn require use of a discount rate. However, EPA's "recovery" of "fixed costs"

[110] See, for instance: SAB letter, signed by Dr. Richard Schmalensee, Chair, to Administrator Carol Browner (re: "Council Review of the Clean Air Act Section 812 Retrospective Study entitled 'The Benefits and Costs of the Clean Air Act, 1970 to 1990'), October 23, 1966, EPA-SAB-COUNCIL-LTR-97-001; SAB letter, signed by Dr. Maureen L. Cropper, Chair, to Administrator Carol Browner (re: "Final Advisory by the Advisory Council on Clean Air Compliance Analysis on the 1999 Prospective Study of Costs and Benefits Implementation of the Clean Air Act Amendments (CAAA)"), November 19, 1999, EPA-SAB-COUNCIL,ADV-00-003; and letter from John D. Graham, Ph.D. Administrator, Office of Management and Budget to Jeffrey R. Holmstead, Assistant Administrator, EPA, September 24, 2001.

methodology asserts—incorrectly (as discussed earlier)—that society will bear no costs before the regulation is implemented and begins providing benefits. Instead, EPA's proposed regulation on nonroad diesel engines would impose "fixed" costs *before* providing benefits. Since a dollar of cost spent in 2003 is worth more than a dollar of benefit received in 2007, EPA's fiction of "no costs incurred before first benefits appear" tends to bias the comparison of NPVs in favor of benefits, at least in the near term. Table 8 shows how expensing (instead of "recovering" EPA's "fixed costs") can alter the NPV estimates.

Table 8. NPV Estimates of "Fixed Costs" (R&D[a], Tooling[b], Certification[c])—Expressed in Millions of 2001 Dollars. [Discount Rate: 3%]

NPV Estimate/Years	Mercatus	EPA
2003–2013	$283.9	$167.3
2013–2018	$0.0	$162.8
All Years[d]	$283.9	$330.0

[a]RIA, Table 6.2-4 [b]RIA, Table 6.2-6 [c]RIA, Table 6.2-8
[d]NPV estimate expressed from the point of view of 2003

EPA's fixed cost estimates indicate that expenditures would occur from 2003 through 2013 and none thereafter. Hence, by expensing these expenditures, the Mercatus estimate applies to the period 2003 through 2013. Since EPA shows regulated firms "recovering" their "fixed" costs over as many as 5 years, the time period for the Agency's estimates extends 5 years beyond 2013, or to 2018. (Keep in mind, through, that "fixed" cost *expenditures* from 2014 through 2018 are zero).

The estimates shown in Table 3 indicate that EPA's "recovery" cost accounting underestimates fixed costs during the years of actual expenditures, overestimates those costs for (in this case) five years after expenditures cease, and slightly overestimates total expenditures for all years. The latter result occurs primarily because EPA "include[s] a seven percent rate of return to the manufacturer"[111]—a rate of return that exceeds the 3 percent discount rate used by EPA to estimate the NPVs. As EPA notes, its approach "results in a higher estimate of the total costs."[112]

EPA does not explain why it knowingly uses an accounting method known to overestimate total costs. One reason for this choice may be that it enables the Agency to claim benefits exceed costs in each and every year (except for the years 2003 through

111 RIA, 6-3.

112 Ibid.

2006 when both benefits and costs are both zero).[113] Had the Agency expensed its notion of "fixed" costs, total costs from 2003 through 2006 would exceed benefits (which would remain at zero for that period since the deadline for compliance begins in 2007). By pushing a large portion of "fixed" costs to later years, EPA is able to match up those costs with benefits that have begun to rise above zero.

Another possible reason may be that EPA expects regulated manufacturers to be potentially a more potent source of political opposition to the proposed standard than the customers of those manufacturers.[114] By "promising" the manufacturers a "return" on their up-front capital expenditures that exceeds the social discount rate, these manufacturers lose nothing and may actually gain. And, as a later section discusses, EPA's cost estimates presume that the regulated manufacturers will be able to pass along virtually all of the direct compliance costs to their customers. Hence, EPA's regulatory cost accounting can be interpreted by a directly affected firm as, "Don't worry. Your bottom line will hardly notice the difference. So, it's not really in your interest—or that of your industry's trade association—to spend money on either lobbyists or a grassroots campaign whipping up political opposition to the proposed regulation."

C. EPA's Direct Cost Estimates Implicitly Assume a Relatively Large Manufacturer

The RIA's "Cost-Benefit Analysis" chapter refers to a model used in EPA's analysis that assumes the market structures of the affected diesel engine industries as "perfectly competitive."[115] However, unlike the homogeneous firms that populate a perfectly competitive market (as described in a standard "Principles of Economics" college textbook), the engine manufacturers that would be directly affected by the proposed regulation vary enormously in size and markets served.

For instance, the "Industry Characterization" chapter in the RIA sorts engine manufacturers into five categories, according to rated horsepower (hp): (1) Between 0 and 25 hp; (2) Between 25 and 75 hp; (3) Between 56 and 175 hp; (4) Between 175 and 750 hp; and (5) Over 750 hp.[116] The second category—engines rated between 25 and 75 hp—"is the largest category with 281,157 units sold in year 2000."[117] This category contains two subcategories: Direct Injection (DI) engines and Indirect Injection (IDI) engines. Yanmar had the largest share of the DI engines (19% of 165,427 units sold in

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[113] RIA, 9-47, Figure 9-2.

[114] Economists, such as George Stigler and Milton Friedman, have pointed out many times in the past that regulated firms can be expected to devote far more time and attention to proposed regulations that affect them directly than customers of these firms who also buy from a large array of firms and, therefore, can afford to spend less time and money learning about (and, possible, actively opposing) any one proposed regulation.

[115] RIA, 10, p. 6. The model is the "Nonroad Diesel Economic Impact Model (NDEIM)."

[116] RIA, 1- 2–1-3.

[117] RIA, 1-2.

2000); Kubota had the largest share of IDI engines (51% of 115,730).[118] Combining the two subcategories reveals that Kubota has the largest market share for the entire 25 to 75 hp category at 28.6%. The percentage shares for the next 8 largest firms after Kubota (arrayed from largest to smallest) mentioned explicitly by the RIA decline rapidly. Elsewhere in the RIA EPA states it expects 28 manufacturers to spend money on R&D to meet the proposed regulation.[119] Since the 9 largest firms mentioned explicitly by the RIA have a combined market share of 88%, the remaining 19 firms (28 minus the 9 explicitly-named firms) have an average market share of approximately 0.63 percent. (No doubt, some of these 19 firms have market shares above 0.63 percent and others have shares less than 0.63 percent.)

The sorts of engineering costs estimated by EPA can have much heavier relative impacts on relatively small firms (such as those with less than 1% market share) than upon relatively large firms (such as Kubota with its 28.6% market share). A report for the Small Business Administration estimates that firms employing fewer than 20 employees bear regulatory costs that are nearly 60 percent above those facing a firm employing over 500 employees.[120] Among the reasons are:

- **A relatively large firm can spread EPA's "fixed costs" over thousands more units than a relatively small firm.** For instance, EPA estimates that the industry's total R&D expenditures for the 25 to 75 hp engine category over 2003 through 2012 would total approximately $73.3 million (in 2001 dollars) before discounting.[121] If each of the 28 firms must spend approximately the same amount on R&D to arrive at end results that will earn EPA certification, then each firm—regardless of relative size—must spend approximately $2.6 million on R&D. For Kubota and its 28.6% market share, this $2.6 million can be spread over approximately 76,000 units annually (starting in 2007 when the regulation becomes effective)—or about $34 an engine. A firm with a 1 percent market share can spread its R&D over approximately 2,600 units—or about $1,000 an engine.

- **EPA assumes *all* firms sell in world markets, not just the United States.** The RIA states: "We are projecting that manufacturers would need to do this R&D to sell engines in Europe, Japan, Australia, and Canada because we expect that similar emission standards would be required on a similar timeframe for each of these regions or countries. Therefore, we have attempted to attribute the costs of R&D to the total engine sales for these regions. Since we do not have sales data for every manufacturer showing what percent of their engines are sold in the US relative to those other regions, we have used Gross Domestic Product (GDP) as a surrogate of sales. As a result, we have attributed only a portion of the R&D

[118] RIA, 1-2.

[119] RIA, 6-9.

[120] Mark Crain and Thomas D. Hopkins, *The Impact of Regulatory Costs on Small Firms,* A report for The Office of Advocacy, U. S. Small Business Administration RFP No. SBAHQ-00-R-0027W. June 2001.

[121] RIA, 6-12, Table 6.2-4.

expenditures to engine sales within the United States. Of the countries expected to have nonroad emission standards of similar stringency to our proposed standards, U.S. GDP constitutes 42 percent of the total. Therefore, we have attributed 42 percent of the R&D costs to U.S. sales."[122]

However, a firm (such as Kubota) with a 28.6% market share of the United States market is far more likely to sell engines in other countries than a firm with less than a 1% share of the U.S. market. Apparently, EPA estimates that *total* R&D expenditures—worldwide—will amount to approximately $174.5 million. (That is – per EPA's attribution method—42% of $174.5 million produces the $73.3 million mentioned in the first bullet.) Hence, each of the 28 firms doing R&D actually must spend approximately $6.2 million worldwide to arrive at results that will pass regulatory muster. For the relatively small U.S. firm selling only in the United States, the per engine R&D cost becomes about $2,380 – compared to Kubota's (approximate) $34 an engine.

- **EPA Implicitly Assumes That Firm Size Does Not Affect Information Costs *re* Meeting Regulatory Requirements.** The *RIA* states that "those manufacturers that sell engines only into the nonroad market would be able to learn from the R&D efforts already underway for both the highway rule and for the Tier 2 light-duty rule. This learning could be done via seminars, conferences, and contact with highway manufacturers, emission control device manufacturers, and the independent engine research laboratories conducting relevant R&D."[123] What EPA leaves unsaid, however, is that manufacturers that sell engines only into the nonroad market tend to be smaller than manufacturers that sell into both markets. Smaller firms may be less able to afford sending employees off to "seminars" and "conferences" than larger firms. Hence, while smaller firms would not violate any physical laws by attempting to learn in the ways suggested by the *RIA*, they may very well find that their costs of doing so exceed the costs faced by larger firms.

D. EPA's Claim That the Proposed Regulation Will Have Virtually No Impact on Sales of Nonroad Engines Strains Credulity

The RIA asserts that despite an average increase in engine prices exceeding $1,000 (about a 23 percent increase), 2013 sales of engines will fall by only 69 out of more than 500,000 because of the proposed regulation.[124] In effect, the RIA can be interpreted as

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[122] RIA, 6- 9 – 6-10.

[123] RIA, 6-6.

[124] RIA, 10-10. The EPA states here: *The market impact model predicts that even with these increases in engine prices, total demand is not expected to change very much. The expected average change in quantity is only about 69 engines per year in 2013, out of total sales of more than 500,000 engines. The estimated change in market quantity is small because as compliance costs are passed along the supply chain they become a smaller share of total production costs. In other words, firms that use these engines and equipment will continue to purchase them even at the higher cost because the increase in costs will not*

assuring engine manufacturers that they will be virtually unaffected by the proposed regulation, that they will be able to pass virtually the entire cost increase on to their customers in the form of higher prices. As mentioned earlier, a cynic would be tempted to view such an assurance as an attempt by EPA to defuse political opposition from the stakeholder group most apt to possess both the knowledge and motivation to lobby vigorously against the proposed regulation, should that group's membership fear bearing a substantial portion of the economic burden. Looked at this way, EPA appears to be telling engine manufacturers (with apologies to the late Senator Huey Long of Louisiana): "This proposal won't hurt you, won't hurt me, but will only hurt the consumers behind the trees."

Even if engine manufacturers (and subsequent members of the "supply chain") can pass on virtually the entire economic burden in the firm of higher prices—a highly dubious proposition as will be discussed shortly—then consumers would be left bearing virtually *all* of the burden. More of their disposable incomes will now go the goods and services made with the help of nonroad diesel engines. Consumers will now have less disposable incomes to spend on other goods and services. Manufacturers of these other goods and services would be able to sell less than before. It may be that this latter effect is spread among so many firms, and in such indirect ways, that none can discern the impacts from the constant "noise" generated by normal, constant change in the economy. Nonetheless, economic pain that cannot be traced back to its ultimate source is still pain suffered by living human beings.

But, how likely is it that engine manufacturers—as a group—will be able to increase prices by an average of about 23 percent and yet (with 28, or so, companies) suffer a sales loss of only 69 engines on a base exceeding 500,000? Such an outcome implies that the demand price elasticity for nonroad diesel engines is not statistically different from zero—totally inelastic. Real world examples of zero price elasticity are virtually nonexistent, even for addictive products such as cigarettes or cocaine. Even though the RIA asserts the demand for nonroad diesel engines displays a long run price elasticity that—if true—would make that market virtually unique, EPA offers only *a priori* reasoning, and no empirical evidence, in support of its remarkable claim. The late astronomer Carl Sagan said that "extraordinary claims require extraordinary evidence." The RIA falls far short of that standard.

"No substitutes" is a prime pillar supporting the RIA's *a priori* case for a zero price elasticity.[125] Yet, a substantial source of substitutes leaps instantly to mind: the stock of

have a large impact on their total production costs. Diesel equipment is only factor of production for their output of construction, agricultural, or manufactured goods. The average decrease in the quantity of all engines produced as a result of the regulation is estimated to be about 0.013 percent. This decrease ranges from 0.010 percent for engines less than 25 hp to 0.016 percent for engines 175 to 600 hp.

[125] The *RIA* states (Chapter 10, p. 9): "The demand for nonroad diesel equipment is ineleastic because of the following: (1) Nonroad diesel equipment and fuel expenditures are a relatively small share of total production costs for the products and services that use this equipment and fuel as inputs. (2) There are limited substitutes for nonroad diesel equipment and fuel." Obviously, the second point refers to the lack of substitutes for new nonroad engines, even at the margin.

existing diesel engines. One could reasonably expect—lacking empirical evidence to the contrary—that a 23 percent price increase for a new diesel engine would motivate at least some profit-conscious companies to retain their existing engines for a longer period of time through more intensive maintenance and repair. Were that to occur, engine manufacturers could easily witness a sudden, substantial reduction in new engine sales in 2013, despite the RIA's assurances of a barely discernable blip in industry sales.

Furthermore, EPA's estimate of a "mere" 23 percent increase in new engine prices is itself dependent upon the accuracy of the Agency's engineering cost estimates. However, as already discussed, some of those estimates appear to apply to relatively large firms that sell engines in several countries around the world, not just in the United States. EPA's estimates imply that relatively small firms could not survive if market prices for new engines rise by only that amount that approximates the entire industry's average cost increase. Hence, accompanying whatever change occurs in total engine sales by the industry as a whole, may be considerable change in the identity of firms supplying the engines actually sold, with larger firms gaining market share at the expense of smaller firms.

E. EPA's Regulatory Cost Accounting For the Proposed Regulation Does Not Provide Much Relevant Information to Policy Makers

EPA's cost accounting adopts the perspective of a directly-regulated firm that asks, "How quickly will I be able to recover my up-front costs under the assurance that those—and all subsequent—costs will be passed along to my customers in the form of higher prices?" Even an accurate answer to this question would be of little use to policy makers because that is not the question they want answered. Instead, they want to know what will be the proposed regulation's opportunity cost from society's point of view. The RIA asserts that its engineering cost estimates provide an accurate measure of total social cost, but that claim—like similar claims by EPA in previous regulatory analyses—are not entertained seriously by professional economists. Finally, the accuracy of EPA's engineering cost estimates themselves is open to serious question.

VIII. Conclusion and Recommendation

Ideally, EPA would rethink not just the Proposed Rule, but more importantly the regulatory context in which it is embedded. The epidemiologic evidence suggests the annual PM2.5 standard is much more stringent than necessary to protect public health and attaining the standard will not confer anywhere near the health benefits EPA imagines. The cost of attainment will likely far exceed the benefits. For ozone, EPA has itself already concluded that the 8-hour ozone standard would impose net costs, while outside analysts have concluded the net costs are far larger than EPA originally estimated.

If EPA were to relax these standards, say to 20 $\mu g/m^3$ for PM2.5, and somewhere between 90 and 95 ppb for 8-hour ozone, most of the related problems would resolve themselves. Nonattainment would be uncommon and remaining non-attainment areas would need fewer reductions to reach attainment. Stringent national rules would then be unnecessary. The NOx disbenefit issue would be defused, because VOC reductions would probably suffice to reach ozone attainment in most areas. The inventory problem would still be with us, but with fewer emission reductions needed, the distortions caused by errant inventories would be less costly. And these less stringent standards would still protect public health.

Since EPA is unlikely to change course on the standards, the Agency should take much more seriously the issue of whether NOx reductions will increase ozone in some or many areas. To the extent NOx reductions are detrimental to ozone, the Proposed Rule will exacerbate an already problematic situation. EPA should likewise revisit the emission inventories used for modeling future air quality. The evidence suggests these inventories overstate future levels of ozone precursors.

EPA should also revamp the Proposed Rule's health benefit assumptions. EPA was only able to achieve its large benefit estimates by ignoring shortcomings in its favored studies, and selectively excluding countervailing evidence.

EPA's regulatory cost accounting does not provide a meaningful estimate of the Proposed Rule's societal cost—the standard against which an unbiased total benefit estimate should be compared. EPA should provide a realistic analysis of the Proposed Rule's social costs.

Finally, EPA should rethink how it communicates health risks to the public, not only for the Proposed Rule, but in general. Rather than resorting to scare stories, EPA should place risk claims in context to give the public a realistic idea of both the harm from current air pollution levels, and the potential benefits of a given emission reduction proposal.

Appendix

The following documents support this Public Interest Comment. Please refer to them at their relevant Web locations:

J. Schwartz, "Particulate Air Pollution: Weighing the Risks," Competitive Enterprise Institute, April 2003, http://www.cei.org/pdf/3452.pdf.

J. Schwartz, "No Way Back: Why Air Pollution Will Continue to Decline," American Enterprise Institute, July 2003, http://www.aei.org/docLib/20030804_4.pdf.

J. Schwartz, "Proposed Rule to Implement the 8-Hour Ozone National Ambient Air Quality Standard, Public Interest Comment," Mercatus Center, George Mason University, August 2003, http://www.mercatus.org/pdf/materials/366.pdf.

APPENDIX I

RSP CHECKLIST

PROPOSED NONROAD DIESEL ENGINE AND FUEL STANDARDS

Element	Agency Approach	RSP Comments
1. Has the agency identified a significant market failure?	EPA does not address this issue in the Proposed Rule, but the idea of market failure is implicit in much of air pollution regulation. **Grade: NA**	To the extent additional emission reductions from nonroad engines are desirable, markets will not provide these reductions without some kind of outside intervention. Key issues are the extent to which nonroad emissions ought to be further reduced, and whether national regulations are necessary.
2. Has the agency identified an appropriate federal role?	EPA cites its authority to regulate nonroad mobile sources under the Clean Air Act, and believes national regulations would be less expensive than a patchwork of regional approaches. **Grade: C**	To the extent additional nonroad emission reductions are desirable, it isn't clear that federal regulations are necessary. Farm and construction equipment, for example, probably doesn't move around the country as much as automobiles, and regional approaches might therefore be effective. Since most areas of the country can attain federal pollution standards without the reductions from this rule, regional approaches might be considerably less expensive.
3. Has the agency examined alternative approaches?	EPA examines only a limited range of alternatives involving minor tweaks to its preferred approach. **Grade: F**	

Element	Agency Approach	RSP Comments
4. Does the agency attempt to maximize net benefits?	EPA does not estimate the social costs of the proposal, and does not examine alternatives to its general command-and-control approach. **Grade: D**	EPA does not estimate the social costs of the proposal and makes implausible cost-related assumptions. As a result, the social costs of the proposal are unknown and likely higher than suggested by EPA's ad hoc cost analysis. EPA also greatly overestimates health benefits of the proposal and ignores the risk that NOx reductions will increase ozone levels.
5. Does the proposal have a strong scientific or technical basis?	EPA believes the proposal is based on rigorous science regarding pollution health effects, particularly premature mortality, and regarding pollution reduction strategy. **Grade: F**	EPA has chosen to ignore cogent evidence that NOx reductions will increase ozone levels in many areas of the U.S. On health effects, the Agency bases its benefit claims on a naïve and selective reading of the epidemiologic literature. EPA's cost analysis bears little or no relationship to the social costs of the Proposed Rule and makes implausible assumptions that likely underestimate the rule's likely costs.
6. Are distributional effects clearly understood?	EPA does not address distributional effects. **Grade: F**	
7. Are individual choices and property impacts understood?	EPA does not address the potential effects of the Proposed Rule on individual choices or property rights. **Grade: F**	