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Taking the Clean Air Task Force to Task for Junk Science: Diesel Exhaust and Health Effects

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In February 2005, the Clean Air Task Force (“CATF”) published a report entitled “Diesel and Health in America: The Lingering Threat,” (“CATF Report”). CATF’s purpose in publishing this report is to force EPA, as well as state and local air pollution control

agencies, to expand upon the already broad scope of EPA’s Clean Diesel rules. Specifically, while EPA’s Clean Diesel rules prescribe costly¹ control technologies for new diesel engines, CATF asserts that these rules do not go far enough. Instead, CATF proposes that the

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¹ EPA has estimated the costs of its Clean Diesel rules to be approximately \$135 billion over the period 2005–2030. See EPA, “Final Regulatory Analysis: Control of Emissions from Non Road Diesel Engines” (May 2004) at p. ES-9, (estimating approximately \$45 billion in costs during the 2005–2030 period) and EPA, “Draft Regulatory Impact Analysis For the Proposed Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements Rule,” (May 2000) at p. iii (estimating approximately \$90 billion in costs during the 2005–2030 period).

nation's existing stock of diesel engines should be retrofitted with additional controls. To justify this prohibitively expensive² proposal, CATF has portrayed the diesel industry as purveyors of "the most widespread air pollution risk in the U.S."³ The CATF Report is just one of a series of recent harsh attacks on the diesel industry.⁴ We can also expect the CATF Report to spawn new toxic tort litigation, with its claims of thousands of diesel-induced deaths each year.

Although the CATF Report is replete with inaccuracies and is not based on reliable science, we address here only the two most egregious errors in the CATF Report: (1) CATF's allegation that diesel exhaust is highly toxic because it contains trace amounts of several potentially hazardous substances, and (2) CATF's allegation that diesel exhaust caused massive deaths in specified geographic areas in 1999 and that, if CATF's proposal to retrofit existing engines is not adopted, diesel exhaust will cause 21,000 deaths in the year 2010 alone and will cause 100,000 deaths between 2005–2030. As demonstrated below, these allegations are false.

■ DIESEL PARTICULATE MATTER IS MERELY A COMPONENT OF PM 2.5—AND NOTHING MORE

Diesel exhaust is a complex substance that consists of two primary components: a vapor phase and a particle phase.⁵

Although it has been subjected to scientific scrutiny,⁶ the vapor phase of diesel exhaust is not of serious concern to toxicologists, industrial hygienists, or epidemiologists.⁷ Rather it is the particle phase of diesel exhaust that is considered a potential hazard. Certainly it is the particle phase that is the primary focus of the CATF report. The particle phase is generally referred to in the scientific literature as "diesel particulate matter" ("DPM"). DPM is one of many types of small particles that comprise what EPA refers to as PM_{2.5}, *i.e.*, particles suspended in the ambient air with a diameter of no more than 2.5 micrometers. However, DPM is certainly not the only substance that makes up PM_{2.5}. In fact, EPA has estimated that DPM constitutes at most 36 percent of all ambient PM_{2.5}, and perhaps no more than 10 percent of ambient PM_{2.5}.⁸ An industry group, the Diesel Technology Forum, has estimated that DPM constitutes only 4.4 percent of PM_{2.5}.⁹

DPM is composed of small carbon particles that have adsorbed minute quantities of a number of different organic and inorganic substances,¹⁰ presumably either as the byproducts of the combustion process¹¹ or as a result of the fuel chemistry employed in the diesel refining process.¹² Of chief concern¹³ among these adsorbed substances are polycyclic aromatic hydrocarbons ("PAHs").¹⁴ Although some

² Although CATF goes to great lengths to describe alleged diesel-induced adverse health conditions, there is scarcely any discussion of the costs of its proposed retrofit of existing diesel engines. The only ink that CATF devotes to the costs of its proposal is to estimate these costs on a per-vehicle basis, so there is no way to ascertain the relative costs and benefits of CATF's proposal as a whole over the 2005–2030 period. It appears, however, that the costs of compliance with CATF's retrofitting proposal are nearly as much as the cost of EPA's Clean Diesel rules. CATF estimates the cost of retrofitting at \$1,000–\$15,000 per engine (CATF, "An Analysis of Diesel Air Pollution and Public Health in America" at pp. 36-37). CATF also estimates there are 13 million existing diesel engines. *Id.* at p. 28. If we assume that the 13 million diesel engine retrofits necessary to meet the CATF proposal are uniformly distributed between the \$1,000 and the \$15,000 retrofit, so that the average cost of a retrofit is \$8,000, that yields a cost of \$104,000,000. This would represent a staggering cost to the diesel industry of \$239,000,000,000 to achieve CATF's goals of both retrofitting existing diesel engines and to comply with EPA's Clean Diesel Rules.

³ CATF Report, p. 9

⁴ See *e.g.* U.S. PIRG Education Fund, "Dangers of Diesel: How Diesel Soot and Other Air Toxics Increase America's Risk of Cancer" (October 2002); Natural Resources Defense Council, "Cleaning Up Today's Dirty Diesels: Retrofitting and Replacing Heavy Duty Vehicles in the Coming Decade" (November 2004).

⁵ EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 5-86.

⁶ *Id.* at p. 5-85.

⁷ *Id.* at pp. 3-1, 6-1, 7-126.

⁸ *Id.* at p. 9-19.

⁹ http://www.dieselforum.com/statements/feb_22_2005-2.html

¹⁰ *Id.* at p. 6-1.

¹¹ *Id.* at p. 2-12.

¹² *Id.* at pp. 2-28 to 2-29.

¹³ In reviewing the scientific literature of the composition of diesel exhaust, EPA addresses a number of studies of the vapor phase and its constituents, and DPM and its constituents. But after reviewing all of these studies, EPA focuses almost exclusively on one group of constituents—polycyclic aromatic hydrocarbons in DPM. See EPA Health Assessment Document for Diesel Engine Exhaust (2002), at pp. 7-126 to 7-137; 9-13.

¹⁴ CATF Report, p. 10. Although CATF also lists a number of other potentially hazardous substances (specifically, formaldehyde, acetaldehyde, butadiene, acrolein, and benzene), in fact these substances are found in the vapor phase, and not in the DPM. As noted earlier, the vapor phase of diesel exhaust is not considered to pose serious health effects issues. Moreover, these substances are present in very small amounts and, as importantly, because they are in the vapor phase, they readily degrade in the atmosphere. See EPA Health Assessment Document for Diesel Engine Exhaust, at p. 2-86 (Table 2-20).

PAHs potentially can be toxic if present at sufficiently high levels, they clearly are not present at such levels in DPM.¹⁵ However, the CATF Report suggests that merely because DPM adsorbs such substances, DPM is acutely dangerous. A sampling of CATF's assertions about the dangers posed by these adsorbed substances include these statements: (a) "Diesel exhaust contains... carcinogens"¹⁶; (b) "[C]arbon particles from mobile sources... adsorb... toxic gases produced by diesel engines—such as cancer-causing PAHs (polycyclic aromatic hydrocarbons)—onto their surfaces making them even more dangerous."¹⁷; (c) "Diesel exhaust can contain 40 hazardous air pollutants as listed by EPA, 15 of which are listed by [the International Agency for Research on Cancer] as known, probable, or possible human carcinogens."¹⁸

While it is undisputed that a number of such potentially hazardous substances are present in diesel exhaust, mere presence is not the relevant scientific issue; instead, the issue is "what is the dose?" For it is foundational in all toxicology that the dose makes the poison—yet CATF provides no dose information for these adsorbed substances. In fact, nowhere in the CATF Report is there any information about how much of these allegedly dangerous substances are present in DPM. Had CATF bothered to report the amount of these substances in diesel exhaust, it would be clear to the readers that the amounts of these substances are at such low levels that they would not even raise the eyebrow of a toxicologist, an industrial hygienist, or an epidemiologist. Certainly, the mere presence of such small amounts of these substances is not of any concern to the regulatory agencies charged with responsibility for safe working environments,

much less for the environment at large. For example, the concentration of these substances in diesel exhaust does not even approach the Permissible Exposure Limits prescribed by the Occupational Safety and Health Administration for any of these substances in the workplace.¹⁹ Likewise, these substances are not present at a level even close to exceeding EPA standards for hazardous air pollutants.²⁰ In its Health Assessment Document for Diesel Engine Exhaust (2002), EPA prepared a table demonstrating just how little of these substances are present in DPM.²¹ That table displays adsorbed organic compounds emitted, not per meter, but per mile(!). It is reproduced here as Table 1.

It is difficult to comprehend why CATF would put such misleading information in the CATF Report, but we suspect it was placed there to create sufficient fear among those who read the CATF Report to force adoption of CATF's proposal to retrofit existing diesel engines with additional controls. We witnessed a similar effort undertaken by our opposing counsel in the nation's first diesel exhaust cancer case tried to a jury verdict, *Missouri Pacific R.R. Co. vs. Navarro*, 90 S.W. 3d 747 (Tex. App.—San Antonio 2002) ("*Navarro*"). There, the plaintiffs' lawyers paraded angry red charts describing diesel exhaust as a witch's brew of benzene, butadiene, formaldehyde, PAHs, and other potentially toxic substances in an effort to convince a jury in Laredo, Texas, that because diesel exhaust contains such substances, it must be terribly lethal.

Like the argument made by those plaintiffs' lawyers in *Navarro*, the music behind the words in the CATF Report is that diesel exhaust is really a "toxic cocktail" simply because

¹⁵ EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 2-74 to 2-80. Of the total mass of DPM, PAHs represented only 0.5 percent in one 1993 test. *Id.* at p. 2-75. Moreover, newer diesel engines and fuels have dropped the amount of PAHs in DPM dramatically, so that they are now present at considerably lower levels. *Id.* at p. 2-65. EPA has not established a limit for PAHs, but OSHA has set a limit for PAHs of 0.2 mg/m³ of air. ATSDR, "Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs)" (August 1995). PAHs in DPM are orders of magnitude below this limit. EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 2-72 to 2-75, where PAHs are measured in micrograms, not per meter, but per mile. See also Table I herein.

¹⁶ CATF Report, p. 1

¹⁷ CATF Report, p. 1.

¹⁸ CATF Report, p. 4.

¹⁹ 29 CFR 1910.1000.

²⁰ EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 2-74 to 2-80. See also 64 Fed. Reg. 38709 (July 19, 1999), where EPA noted that its mobile source air toxics program was focused in four areas: (1) redesign of gasoline (removing lead from gasoline and reformulating gasoline), (2) advance catalysts and computer-controlled fuel systems, (3) new diesel fuel rules, and (4) control of DPM. In the same 1999 notice, EPA noted that it would investigate any health risks associated with these adsorbed substances (64 Fed. Reg. at 38714; 38725), which it did in its 2002 Health Assessment Document for Diesel Engine Exhaust and where EPA prescribed particulate controls, but no controls on any of these adsorbed substances. *Id.* at pp. 2-15 to 2-17. Moreover, EPA's Clean Diesel rules prescribe no specific controls for these adsorbed substances. *Id.* The lack of such controls is even more understandable in light of the fact that the amount of such adsorbed compounds in DPM has dropped precipitously over the last 25 years, so that the adsorbed organic compounds of DPM now constitute only 20 percent of what they did in 1980. *Id.* at p. 2-65.

²¹ EPA Health Assessment Document for Diesel Engine Exhaust (2002) at p. 2-73.

it contains a number of potentially toxic substances, even if each such substance is present at such an insignificant level that it does not pose any kind of risk. The CATF Report contains several backdoor claims of synergism: (a) “Diesels churn out a hazardous mix of gaseous and particle pollutants.”²²; (b) “Diesel exhaust contains... a host of air toxics.”²³; (c) “We know that diesel exhaust is a hazardous mixture of gases and particles including carcinogens, mutagens, respiratory irritants and inflammatory agents and other toxins that cause a range of diverse health effects.”²⁴ The undisputed truth, however, is that there is no scientific evidence that these substances act in any synergistic manner, and the CATF Report cites to no articles in support of a claim of synergism.²⁵ Absent a synergistic effect, each substance in diesel exhaust must be evaluated on its own, independent of the presence of any other substance—and in the case of DPM, these substances are present at such low levels that they can pose no danger under any accepted regulatory standard.

Moreover, the mere fact that these substances are present in DPM does not mean that these substances are bioavailable. CATF, however, treats this very complex issue of bioavailability²⁶ as if it were quite simple and, predict-

ably, CATF claims that these substances are bioavailable: “Diesel particles act like magnets for toxic organic chemicals and metals. The smallest of these particles (ultrafine particles) can penetrate deep into the lung and enter the bloodstream, carrying with them an array of toxins.”²⁷ Again, the CATF Report is flatly contradicted by sound science. Two specific studies²⁸ have tested CATF’s assertion, and both refute it. Both studies involved rats because, unlike other species,²⁹ rats seem predisposed to develop lung tumors after being forced to take into their lungs incredibly large quantities of very small particles.³⁰ Both research studies tested precisely why the rats developed the tumors, and both found no evidence that the substances adsorbed onto the DPM were implicated in the formation of the lung tumors. Rather, both studies concluded that it is simply inhaling very small particles, in incredibly large quantities, that can be correlated with the development of the rat lung tumors.

It is also significant that practically all of the toxicological studies of the substances adsorbed onto DPM have uniformly found it necessary to extract these tightly bound adsorbed substances from the DPM through the use of organic solvents not found in the rats (or, for that matter, in the human

²² CATF Report, p. 1.

²³ CATF Report, p. 1.

²⁴ CATF Report, p. 4.

²⁵ Moreover, there are no studies cited in the 700 pages of EPA’s Health Assessment Document for Diesel Engine Exhaust (May 2002) that suggest any synergistic effect among these adsorbed substances.

²⁶ While not conceding that these adsorbed substances are not bioavailable, EPA recognizes the complexity of this issue and repeatedly refers to the conflict in these studies. EPA does refer to some studies that could be construed to support very limited bioavailability (see e.g. EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 7-126 to 7-137; 9-13), but nothing in any of these studies could be construed as demonstrating the magnitude, much less the certainty, of the bioavailability that the CATF Report treats as an undisputed fact.

²⁷ CATF Report, p. 4

²⁸ Mauderly et al., “Comparison of the Carcinogenicity of Diesel Exhaust and Carbon Black in Rat Lungs” available at <http://www.healtheffects.org/pubs/st68l.htm>; Nikula et al., “Comparative Pulmonary Toxicities and Carcinogenicities of Chronically Inhaled Diesel Exhaust and Carbon Black in F344 Rats,” *Fundamental and Applied Toxicology* 25:80-94 (1995).

²⁹ Mauderly et al., “Diesel Exhaust is not a Pulmonary Carcinogen in CD-1 Mice Exposed Under Conditions Carcinogenic to F344 Rats,” *Fundamental and Applied Toxicology* 30:233-242 (1996); Mauderly et al., “Lung Overload: The Dilemma and Opportunities for Resolution,” *Inhalation Toxicology* 8 (Suppl. 1):1-28 (1996).

³⁰ Mauderly et al., “Diesel Exhaust is a Pulmonary Carcinogen in Rats Exposed Chronically by Inhalation,” *Fundamental and Applied Toxicology* 9:208-221 (1987); Valberg et al., “Comparative Mutagenic Dose of Ambient Diesel Engine Exhaust,” *Inhalation Toxicology* 11:215-228 (1999).

body). These organic solvent materials include acetone,³¹ dimethylformamide,³² acetonitrile,³³ dichloromethane,³⁴ methylene chloride,³⁵ cyclohexane and methanol,³⁶ and toluene:dichloromethane:methanol(1:1:1).³⁷ Likewise, most animal inhalation studies have “typically inserted the particles directly into the animals’ tracheas, which isn’t the same as inhaling them,”³⁸ for it bypasses the natural defenses the animals would otherwise use to fend off the adverse effects of such constituents. Our cross-examination of the plaintiffs’ experts in the *Navarro* trial ultimately yielded this truth: There are no studies that establish biological plausibility for the substances adsorbed onto the DPM to be released³⁹ in the

human body at levels that could pose a health threat. Simply put, rats (and humans) do not contain ready stores of these solvents, nor do they produce similarly functioning biological fluids, that would be required to extract these tightly bound substances adsorbed onto the DPM. As a consequence, these substances largely remain adsorbed onto the DPM and so are not released into the rat (or human) body at dangerous levels. Consequently, the only biological mechanism that can explain these two rat inhalation studies is that small particle size, and small particle size alone, can be correlated with the formation of these rat tumors.⁴⁰ In any event, the CATF Report certainly presents no evidence to support its asser-

³¹ Kotin, et al, “Aromatic Hydrocarbons,” *Industrial Health* 11:113-120 (1955).

³² Muzyka, et al, “On the Carcinogenic Risk Evaluation of Diesel Exhaust: Benzene in Airborne Particles and Alterations of Heme Metabolism in Lymphocytes as Markers of Exposure,” *Sci Total Environ* 217(1-2):103-11 (1998a); Muzyka, et al, “Particle-Bound Benzene from Diesel Engine Exhaust,” *Scand J Work Environ Health* 24(6):481-5 (1998b).

³³ Rappaport, et al, “Isolation and Identification of a Direct-Acting Mutagen in Diesel-Exhaust Particulates,” *Environ. Science & Tech.* 14:1505-1509 (1980).

³⁴ Rappaport, et al, “Isolation and Identification of a Direct-Acting Mutagen in Diesel-Exhaust Particulates,” *Environ. Science & Tech.* 14:1505-1509 (1980); Dukovich, et al, “The Mutagenic and SOS-Inducing Potential of the Soluble Organic Fraction Collected from Diesel Particulate Emissions,” *Environmental Mutagenesis* 3:253-264 (1981); King, et al, “Evaluation of the Release of Mutagens From Diesel Particles in the Presence of Physiological Fluids,” *Environmental Mutagenesis* 3(e):109-121 (1981); Brooks, et al, “Deposition and Biological Availability of Diesel Particles and Their Associated Mutagenic Chemicals,” *Environmental International* 5:263-267 (1982); Clark, et al, “Mutagenicity of Diesel Exhaust Particle Extracts: Influence of Fuel Composition in Two Diesel Engines,” *Fundamental and Applied Toxicology* 2:38-43 (1982); Li, et al, “Diesel-Exhaust-Particle Extract Enhancement of Chemical-Induced Mutagenesis in Cultured Chinese Hamster Ovary Cells: Possible Interaction of Diesel Exhaust with Environmental Carcinogens,” *Mutation Research* 103:349-355 (1982); Rappaport, et al, “High-Performance Liquid Chromatography with Reductive Electrochemical Detection of Mutagenic Nitro-Substituted Polynuclear Aromatic Hydrocarbons in Diesel Exhausts,” *Journal of Chromatography* 240:145-154 (1982); Albert, et al, “Comparative Potency Method for Cancer Risk Assessment: Application to Diesel Particulate Emissions,” *Soc. for Risk Anal.* 3:101-117 (1983); Lewtas, “Evaluation of the Mutagenicity and Carcinogenicity of Motor Vehicle Emissions in Short-Term Bioassays,” *Env. Hlth. Perspectives.* 47:141-152 (1983); Lewtas, et al, “A Comparative Potency Method for Cancer Risk Assessment: Clarification of the Rationale, Theoretical Basis, and Application to Diesel Particulate Emissions,” *Risk Analysis* 3:33-137 (1983); Ong, et al, “Genotoxicity Studies of Rodents Exposed to Coal Dust and Diesel Emission Particulates,” *Env. Res.* 37:399-409 (1985).

³⁵ Barfknecht, et al, “Human Cell Mutagenicity of Polycyclic Aromatic Hydrocarbon Components of Diesel Emissions,” *Dev. Toxicol. Environ. Sci.* 10:277-294 (1982); Pereira, “Genotoxicity of Diesel Exhaust Emissions in Laboratory Animals,” *Dev. Toxicol. Environ. Sci.* 10:265-276 (1982); Lewtas (eds), *TOXICOLOGICAL EFFECTS OF EMISSIONS FROM DIESEL ENGINES* (Elsevier Science Publishing Co., Inc. 1982) pp. 265-276; Pereira, “In Vivo Detection of Mutagenic Effects of Diesel Exhaust by Short Term Mammalian Bioassays,” *Environ. Int.* 5:439-443 (1981).

³⁶ King, et al, “Evaluation of the Release of Mutagens From Diesel Particles in the Presence of Physiological Fluids,” *Environmental Mutagenesis* 3:109-121 (1981).

³⁷ King, et al, “Evaluation of the Release of Mutagens From Diesel Particles in the Presence of Physiological Fluids,” *Environmental Mutagenesis* 3:109-121 (1981); Nakagawa, et al, “Identification of Disintropyrenes in Diesel-Exhaust Particles: Their Probable Presence as the Major Mutagens,” *Mutation Research* 124:201-211 (1983).

³⁸ Kaiser, “Mounting Evidence Indicts Fine Particle Pollution,” *Science* 307:1858-1861 (2005).

³⁹ Or, as the scientific literature calls it, “desorbed” from the particle. EPA found a few isolated studies suggesting that some limited desorption occurred, but nothing on the scale necessary to release a potentially toxic dose of any of such substances. See EPA Health Assessment Document for Diesel Engine Exhaust (May 2002) at pp. 7-127 to 7-130.

⁴⁰ Perhaps the most complex scientific issue involving diesel exhaust is whether it causes lung cancer in humans. EPA has (we think incorrectly) designated diesel exhaust as a “likely” human carcinogen, based largely on earlier rat tumor studies and a 1988 epidemiology study of railroad workers (Garshick et al., “A Retrospective Cohort Study of Lung Cancer and Diesel Exhaust Exposure in Railroad Workers,” *Am Rev Respir Dis* 135(6):1242-1248 [1988]). That epidemiology study has been the subject of a robust written dialogue over the last 15 years between its lead author, Eric Garshick, and another very well-regarded scientist, Kenny Crump. Garshick recently published a new article on the same subject, in which his team concluded that the relative risk (the numerical ratio denoting the mathematical correlation between exposure to a substance and an adverse health outcome) of developing lung cancer from diesel exhaust exposure was only 1.40; to further complicate any such association, he noted that lung cancer mortality was inversely related to years worked, *i.e.*, the more years one is exposed to diesel exhaust, the less likely one is to develop lung cancer. Moreover, when these numbers are adjusted for smoking, the relative risk is only 1.17 to 1.27. Garshick’s team asserts that such a lowering of the relative risk from 1.40 to 1.17-1.27 assumes “no interaction between diesel exposure and smoking, but there are insufficient data to assess this possibility.” Stated otherwise, Garshick’s team’s assertion of a relative risk of 1.40 assumes that smoking and diesel exhaust act synergistically to increase the likelihood of lung cancer, without citing to any scientific literature to support this position. See Garshick et al, “Lung Cancer in Railroad Workers Exposed to Diesel Exhaust,” *Environmental Health Perspectives* 112 (15):1539-1543 (November 2004). Because the sole purpose of this note is to address the two most egregious claims among the CATF Report’s many erroneous assertions, the issue of human lung cancer and DPM (which CATF scarcely mentions in the CATF Report) will not be addressed further here. Nevertheless, it will be important to follow EPA’s decision-making in the light of this recent Garshick study, particularly its notation that smoking adjusted data leads to essentially no difference in the relative risks between DPM and lung cancer and that between PM_{2.5} and lung cancer. *Id.* Finally, it should be emphasized that it is only lung cancer studies that have formed the basis for EPA’s finding that DPM is a “likely” human carcinogen. Other human cancers have not demonstrated such a positive statistical correlation. For example, there is no causal connection between DPM and multiple myeloma (Wong, “Is There Causal Relationship Between Exposure to Diesel Exhaust and Multiple Myeloma,” *Toxicol. Rev.* 22(2) 91-102 [2003]) or between DPM and acute myelogenous leukemia (Boffetta, “Risk of Acute Myeloid Leukemia After Exposure to Diesel Exhaust: A Review of the Epidemiological Evidence,” *J. Occup. Environ. Med.* 46(10):1076-1083 [October 2004]).

tion that these substances—even if they were present at a level (which they are not) that might present a hazard—would ever be bioavailable in the quantities necessary to produce an adverse health outcome.

In effect, then, although CATF has erroneously characterized it otherwise, when inhaled, the current state of the science is that DPM is plainly and simply a component of PM_{2.5}. No less, but no more.⁴¹ While studies of PM_{2.5} will continue to be undertaken to understand the health risks that PM_{2.5} in the ambient air may pose, DPM appears at this time to be, at worst, a contributor to the ambient levels of PM_{2.5}.⁴² Certainly there is no reliable science to support CATF's assertion that DPM has been proven⁴³ to be a uniquely hazardous type of ambient PM_{2.5}.

■ THERE IS NO SCIENTIFIC SUPPORT FOR CATF'S CLAIMS REGARDING DIESEL-INDUCED PREMATURE MORTALITIES

Central to the CATF Report are its erroneous claims that because of diesel exhaust, 100,000 lives will be lost during the period 2005–2030 and 21,000 lives will be lost in year 2010 alone, and that it can pinpoint precisely how many deaths occurred in specific locales during 1999. Yet after even a thorough reading of the CATF Report, it is difficult to find precisely how CATF arrived at these claimed diesel-induced death tolls.⁴⁴ Instead, to find how these calculations were made, one must refer to a technical report prepared by CATF's consultant, Abt Associates, Inc., entitled "Diesel Emissions: Particulate Matter-Related Health Damages," prepared originally in December 2004 and revised in March 2005. We encourage CATF to prepare a second revision to its report that repudiates these erroneous claims, and we hope the discussion below will convince CATF to disavow

them. CATF made its prediction of diesel-induced deaths by employing two scientifically indefensible, and internally inconsistent, theories. One concerns CATF's estimates of diesel-induced premature mortality for 1999 and 2010. The second concerns CATF's estimates of diesel-induced premature mortality for the period 2005–2030. Both are addressed below.

CATF's Estimates of Premature Mortality for 1999 and 2010.

To estimate diesel-induced premature mortality both for year 1999 and for year 2010, CATF modeled theoretical concentrations of ambient PM_{2.5} in different locales in 1999 and predicted what those concentrations would be in 2010 in those same locales. CATF's consultant used these modeled estimates of PM_{2.5} emissions in conjunction with an ill-defined "concentration/response function"⁴⁵ (*i.e.*, as the concentration of a substance—here, PM_{2.5}—increases, the response is an increasing likelihood of an adverse health outcome, which CATF asserts here to be premature mortality) to estimate the number of people whose deaths CATF claims were diesel-induced in specific locales in year 1999.⁴⁶ Simply stated, the more PM_{2.5} emitted in an area according to CATF's modeling, the more deaths CATF predicted. CATF employed the same methodology to predict that 21,000 people would die because of diesel exhaust in year 2010.⁴⁷ As demonstrated below, CATF is flatly wrong both in its estimate of 21,000 deaths in year 2010 and in its individual locale-specific estimates of diesel-induced deaths in year 1999.

Exactly how CATF could produce such exaggerated claims of diesel-induced premature mortality is difficult to ascertain. But with some intensive searching, at least part of the answer can be found in "Non-Road and On-Road Diesel Emissions: Particulate Matter-Related Health Damages," a CATF docu-

⁴¹ Xia, et al, "Quinones and Aromatic Chemical Compounds in Particulate Matter Induce Mitochondrial Dysfunction: Implications for Ultrafine Particle Toxicity," *Environmental Health Perspectives* 112:1347-1358 (2004). See also, Garshick, et al, "Lung Cancer in Railroad Workers Exposed to Diesel Exhaust," *Environmental Health Perspectives* 112(15): 1539-1543 (November 2004), where the authors note that positive, smoking adjusted, statistical associations between DPM and human lung cancer "are similar to smoking adjusted RRs [*i.e.*, relative risk, meaning the degree of association between exposure to a substance and an adverse health outcome] attributable to fine particulate air pollution on lung cancer in prospective population-based cohorts (Dockery, et al, 1993; Pope, et al, 2002)..."

⁴² And if EPA is correct in its estimates of the impact of its Clean Diesel rules, diesel exhaust will contribute less and less to ambient PM_{2.5} levels over the next 25 years.

⁴³ EPA's 2002 classification of diesel exhaust as a "likely" human carcinogen notwithstanding, the 2004 Garshick Study (see footnote 40, *supra*) and the recent rat tumor studies implicate, if anything, PM_{2.5} as a whole, and not specifically DPM.

⁴⁴ CATF Report, p.3

⁴⁵ The concentration/response function that CATF employed is 0.0445. This mysterious concentration/response function used for these estimates is fatally flawed, scientifically unreliable, and produces dramatic overpredictions. It is examined in detail in the succeeding discussion.

⁴⁶ These are displayed in the CATF Report, pp. 7-8.

⁴⁷ CATF, "An Analysis of Diesel Air Pollution and Public Health in America," p. 7.

ment that purportedly supports the CATF Report. There, CATF states that it is relying on an epidemiology study⁴⁸ (“Pope Study”) that tested possible statistical correlations of ambient concentrations of PM_{2.5} (measured in 10 ug/m³ increments) with adverse health outcomes.⁴⁹ While the Pope Study did find a positive statistical link between increasing concentrations of ambient PM_{2.5} (*i.e.*, not just DPM) and premature mortality,⁵⁰ it is a very low mathematical correlation, or relative risk (the degree of association between exposure and disease),⁵¹ of only 1.06.⁵² This is very nearly the result that would occur from pure chance (1.00). Certainly, however, a relative risk of 1.06 does not establish a causal link between the two.⁵³ And in fairness to the authors of the Pope Study, they did not use the word “causation,” to describe this correlation. The failure of the Pope Study to establish a statistically significant association, much less causation, is understandable in light of the fact that only two years earlier, another epidemiology study (“Lipfert Study”) found absolutely no correlation at all between PM_{2.5} and premature mortality.⁵⁴

Unfortunately, CATF chose both to disregard the findings of the Lipfert Study that there is no causation and to misuse the Pope Study’s limp statistical correlation as if it does establish causation. Unlike CATF, our courts require reliable scientific proof of causation and would never confuse a statistical correlation of only 1.06 with causation. In fact, absent compelling evidence to the contrary, epidemiological studies suggesting anything less than a doubling of the risk (that is a relative risk ratio of at least 2.00) is not likely to be admitted into evidence in the courts of Texas,⁵⁵ or elsewhere.⁵⁶ Using the Pope Study to attempt to support causation, as CATF has done, is the worst kind of junk science. As the *Navarro* court made clear in the diesel exhaust cancer case that we tried, it is inappropriate for a plaintiff’s expert witness to assert there to be a causal relationship between diesel exhaust and a specific health outcome by relying on epidemiological studies whose authors did not find such a causal link.⁵⁷ It goes without saying that, were CATF to be subjected to the scrutiny of the courts, it would never be allowed to testify about such insupportable theories.

⁴⁸ See Pope et al, “Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution,” *Journal of the American Medical Association* 287:1132-1144 (2002).

⁴⁹ *Id.*

⁵⁰ The Pope Study’s term is actually not “premature mortality” (which CATF employs), but rather “all cause mortality.” However, for purposes of this analysis, the meanings appear to address the same adverse health outcome, *i.e.*, a death the authors say would not occur, but for excessive concentrations of PM_{2.5}.

⁵¹ “Relative risk” is a numerical ratio that expresses the association between exposure to a substance and an adverse health outcome. A relative risk of 2.00 indicates that the adverse health outcome is twice as common in the exposed subjects as in the unexposed subjects. See Green et al., *Reference Guide on Epidemiology* in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (Fed. Jud. Center 2d ed. 2000), pp. 348-349. A relative risk of 2.00 or greater is often equated with the “more likely than not” standard that governs medical causation issues in toxic tort litigation. On the other hand, a relative risk of 1.00 means that the substance at issue has no apparent relationship to an adverse health outcome, *i.e.*, it is purely by chance that the two may be associated.

⁵² See Pope Study, p.1136.

⁵³ Importantly, the subjects of the Pope Study were at least 30 years old on the date they were enrolled in the Study in 1982. The Pope Study assumes that the further one goes back in time, the more PM_{2.5} was present in the ambient air. If that is true, the subjects of the Pope Study had substantially more exposure to PM_{2.5} between their date of birth (1952 or earlier) and their enrollment in 1982 in the study (more than 30 years) than was their PM_{2.5} exposure between their date of enrollment and the date that the Pope Study was prepared for publication (less than 20 years). Yet the Pope Study apparently made no effort to estimate PM_{2.5} in these metropolitan areas for the period between the subjects’ birth and their enrollment in the study. If the amount of PM_{2.5} was higher before 1982 than after 1982, as the Pope Study seems to suggest (as does EPA; see EPA Health Assessment Document for Diesel Engine Exhaust [2002] at p. 2-37), and if the impact of such PM_{2.5} is additive, as the Pope Study also seems to suggest, then there is even less basis for a correlation than the very weak one that the Pope Study found. In fact, if the decline rate in the ambient concentration of PM_{2.5} was steeper during the period between the subjects’ date of birth and their enrollment than was the decline rate during the period between the date of enrollment and the date of preparation of the Pope Study, the entire “correlation” may be attributable solely to events during this earlier period that dates back to 1952 or earlier. Moreover, in a reanalysis of the same data, the Health Effects Institute concluded that these observed deaths were associated with a mix of pollutants that included PM_{2.5}, but were not associated with PM_{2.5} alone. See Krewski et al., “Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality,” at p. 207 (Table 46), Health Effects Institute (2000). It should also be noted that one recent epidemiology study seems to conflict with the Pope Study’s assumption that the impact of PM_{2.5} is additive. Garshick, et al., “Lung Cancer in Railroad Workers Exposed to Diesel Exhaust,” *Environ Health Perspect* 112(15) 1539-1543 (November 2004). There, lung cancer mortality was inversely related to total years worked with a railroad, in spite of the fact that such subjects were obviously exposed to more PM_{2.5} the longer they worked there.

⁵⁴ See Lipfert et al, “The Washington University—EPRI Veterans’ Cohort Mortality Study: Preliminary Results,” *Inhalation Toxicology* 12 (Supp. 4):41-73 (2000).

⁵⁵ See *Merrell Dow Pharmaceuticals v. Havner*, 953 S.W.2d 706, 718-719 (Tex. 1997)

⁵⁶ *Daubert v. Merrill Dow Pharmaceuticals, Inc.*, 43 F.3d 1311 (9th Cir. 1995); *Jones v. Ortho Pharmaceutical Corp.*, 163 Cal. App. 3d 396, 403-404 (1985); *Deluca v. Merrill Dow Pharmaceuticals, Inc.*, 911 F.2d 941, 958 (3d Cir. 1990); *Hall v. Baxter Healthcare Corp.*, 947 F. Supp. 1387, 1403 (D. Or. 1996); *Manko v. United States*, 636 F. Supp. 1419, 1434 (W.D. Mo. 1986), *aff’d* in part 830 F.2d 831 (8th Cir. 1987); *Marder v. G.D. Searle & Co.*, 630 F. Supp. 1087, 1092 (D. Md. 1986), *aff’d* without op. sub nom., *Wheelahon v. G.D. Searle & Co.*, 814 F.2d 655 (4th Cir. 1987); *In re “Agent Orange” Prod. Liab. Litig.*, 597 F. Supp. 740, 835-37 (E.D.N.Y. 1984), *aff’d*, 818 F.2d 145 (2d Cir. 1987); *Cook v. United States*, 545 F. Supp. 306, 308 (N.D. Cal. 1982); *Landrigan v. Celotex Corp.*, 605 A.2d 1079, 1087 (N.J. 1992).

⁵⁷ See *Navarro* at 90 S.W. 3d 758.

In any event, it was only by choosing to treat the Pope Study as if it established causation that CATF could reach its clearly erroneous claims that diesel exhaust will cause the deaths of 21,000 people in year 2010, or that a massive number of deaths in specific locales during year 1999 could be attributed to diesel exhaust. Specifically, to the extent that CATF's emission estimates predicted an ambient PM_{2.5} of 10 ug/m³ or more in a particular locale in year 1999, CATF derived a concentration response function⁵⁸ (misusing the Pope Study) that predicted a 4 percent increase in premature mortality for each increment of modeled 10 ug/m³ PM_{2.5}. This remarkable and illogical leap enabled CATF to ratchet up its estimated death toll for each locale. Likewise, CATF applied the same concentration response function to all of its modeled emission estimates for each locale in year 2010 and arrived at a cumulative nationwide predicted diesel-induced death toll of 21,000 for year 2010.

There are several reasons why CATF is incorrect. First, a very weak statistical correlation (1.06) does not causation make. Second, even CATF's absurd death toll claims are dramatically higher than any premature mortality arguably suggested in the Pope Study. The Pope Study addressed all PM_{2.5} and not just DPM. Because DPM constitutes only 4.4-36 percent of ambient PM_{2.5}, CATF's predictions of premature mortality are at least three (and perhaps as much as 23) times higher than

the Pope Study could possibly predict. Third, the "concentration response" function that CATF employs to make these claims of diesel-induced deaths is fallacious. While the errors in the math used to create CATF's concentration/response function are addressed in the succeeding assessment of CATF's prediction of 100,000 diesel-induced deaths in the 2005–2030 time period, there are also several conceptual infirmities in CATF's approach. CATF's concentration/response function is derived through a combination of (a) misusing the Pope Study, as if it established causation, which it does not, (b) using CATF's modeled estimates of PM_{2.5} emissions in year 1999 in specific locales as if they were actual monitoring results, which they are not, (c) treating all PM_{2.5} as if it were DPM, which it is not, and (d) assuming that there is no safe threshold for PM_{2.5}, which has no support in reliable science.⁵⁹ Unless all four of these factors are correct, the concentration response function is wrong. Since all four factors are wrong, CATF's concentration response function lacks any scientific plausibility.

Finally, it is noteworthy that PM_{2.5} concentrations are higher in urban areas than in rural areas.⁶⁰ According to CATF, this means that urban areas will have greater premature mortality than rural areas because of diesel exhaust. But it seems at least as plausible to attribute the Pope Study's positive statistical correlation (between increasing PM_{2.5}

⁵⁸ CATF combined its modeled PM_{2.5} estimates with its erroneous interpretation of the Pope Study, to create this concentration/response function. Whenever CATF modeled a 10 ug/m³ increase in PM_{2.5} emissions in a specific locale, CATF assumed a 4 percent increase in premature mortality. After totaling all such PM_{2.5} modeled emissions nationwide and all such predicted increases in premature mortality nationwide for year 1999, CATF divided the total modeled emissions into the total predicted deaths to arrive at its "concentration/response function."

⁵⁹ In fact, in the rat tumor studies, the quantities of small particles the rats were forced to inhale to develop tumors is staggering. In one meta-analysis of the rat tumor studies, the authors concluded, in effect, that there was a threshold for DPM because no rats developed tumors except when they were subjected to lung "overload" conditions, specifically greater than a 600 ug/m³ continuous lifetime exposure, which would equate to a human continuous lifetime exposure threshold concentration of 1400 ug/m³. Valberg, et al., "Meta-Analysis of Rat Lung Tumors from Lifetime Inhalation of Diesel Exhaust," *Environmental Health Perspectives* 107:693-699 (1999). By contrast, the ambient average concentrations of DPM to which the general population is exposed is 0-3 ug/m³. *Id.* See also EPA Health Assessment Document for Diesel Exhaust (2002) at p. 2-106, (Table 2-26), where EPA notes that the annual average concentration of DPM was 2.1 ug/m³ in 1996. Even then, "an animal study may or may not provide reasonably reliable support for an opinion on causation in human beings. Differences between human beings and other species, including differences in absorption, distribution, and metabolism of substances, may affect toxicity." (Goldstein & Henifin, *Reference Guide on Toxicology* in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, at p. 419.) Moreover, the "high doses typically used in animal experiments compared with the much lower levels to which human beings typically may be exposed make it necessary to consider the relationship between dose and response, the shape of the dose-response curve at lower levels of exposure, and the possibility that exposure may not cause a disease when the exposure is below a threshold level." (*Id.* at pp. 409, 410). The Pope Study results were "based on average [PM_{2.5} concentrations of] 10 to 30 ug/m³. By 2000, the range across these cities was about 5 to 20 ug/m³, a reduction of nearly one-half." Schwartz, "Particulate Air Pollution—Weighing the Risks," *Competitive Enterprise Institute*, at p. 16 (2003). In future updates of the Pope Study, it will be important to watch for whether similar reductions in ambient PM_{2.5} concentration (resulting from both mobile and stationary source emission reductions over the next several decades) will effect a concomitant drop in premature mortality, or if some threshold is (or has already been) reached. That there clearly must be a threshold for DPM is evidenced in Reed, et al., "Health Effects of Subchronic Exposure to Environmental Levels of Diesel Exhaust," *Inhal. Toxicol.* 16(4):177-193 (April 2004), where realistic concentrations of contemporary diesel emissions were associated with essentially no adverse health effects in rats or mice.

⁶⁰ This is displayed well in the map in the CATF Report, p. 14.

and premature mortality) to non-diesel factors or what epidemiologists refer to as confounding.⁶¹ For example, the *New York Times* recently noted that geriatricians have long marveled at the longevity of the residents of McIntosh County, North Dakota. It has a higher proportion of residents who are at least 85 years old than any county in the U.S. The author conducted a number of interviews to understand why these residents have this remarkable longevity. The conclusions reached were that, in addition to relatively clean air, there are a number of factors contributing to the longevity of McIntosh County residents: “going slow, patience, low-cost, low-stress economy, ...decades of heavy lifting outdoors, keeping an eye out for one another, long stable marriages, [and] an absence of sharp differences in income and wealth....”⁶² While we do not suggest that a *New York Times* article is a substitute for a scientifically rigorous epidemiology study, it at least raises a series of considerations that could very well explain this disparity between the premature mortality statistics of urban and rural residents. Thus, the weak statistical link of 1.06 that the Pope Study found between increasing concentrations of PM_{2.5} and premature mortality might very well be sheer coincidence.⁶³

CATF’s Estimates of Premature Mortality for 2005–2030.

The second erroneous assertion that CATF made regarding premature mortalities relates to its claim that if EPA, as well as state and local environmental agencies, will not adopt CATF’s proposal to retrofit existing diesel engines with prohibitively expensive controls, 100,000 more people will die during the period 2005–2030.⁶⁴ Despite making this bold assertion, however, the CATF Report does not supply the calculations necessary to support its claim that these government employees (as well as the diesel industry) bear responsibility for these alleged deaths. Nevertheless, we checked CATF’s math, and like its misuse of the Pope Study and the clearly erroneous concentration/response function it devised, CATF’s

numbers don’t add up. Unlike CATF, which did not provide its calculations in the CATF Report, our calculations are set forth in the Appendix, Table 2. We not only invite CATF to check our math, we also encourage CATF to provide the same kind of transparency for the claims that it makes. While the details as to why CATF is wrong are set forth in that Appendix, we highlight below what CATF did and why it is wrong about its claim of 100,000 diesel-induced deaths:

- a) CATF constructed three scenarios for the period 2005–2030: (1) a “baseline” in which neither the EPA Clean Diesel rules, which contemplate strict diesel control technology on new diesel engines, nor CATF’s proposal, which contemplates retrofitting existing diesel engines with additional controls, would be implemented; (2) what CATF pejoratively calls a “business-as-usual” scenario in which the very restrictive EPA Clean Diesel rules will be implemented, but the CATF proposal will not be implemented; and (3) a scenario consistent with the “CATF Goal” of implementation of both the EPA Clean Diesel rules and the CATF proposal to retrofit all 13 million existing diesel engines.
- b) Using each of these scenarios, CATF predicted that even if EPA’s Clean Diesel rules are fully implemented (scenario 2), the failure to adopt CATF’s retrofitting proposal is so dramatic that during the period 2005–2030, 100,000 lives will be lost. To make this calculation of 100,000 “saved lives,” CATF adopted the same erroneous concentration response function⁶⁵ that it derived from its 1999 estimated death and emissions data, and its misuse of the Pope Study, discussed in the immediately preceding section.
- c) While we question the scientific support for EPA’s 2002 decision to classify diesel exhaust as a likely human carcinogen, it is at least worth noting that even EPA is dramatically at odds with CATF’s findings.

⁶¹ Confounding has been defined as “a confusion of effects. Specifically, the apparent effect of the exposure of interest is distorted because the effect of an extraneous factor is mistaken for or mixed with the actual exposure effect (which may be null).” Rothman & Greenland, *MODERN EPIDEMIOLOGY* (1998) at p. 120.

⁶² Kilborn, “North Dakota Town’s Payoff for Hard Lives is Long Life,” *New York Times* (July 31, 2003).

⁶³ Here, instead of DPM or PM_{2.5} emissions, the reported differences in premature mortality may be attributable solely to a rural vs. urban lifestyle. At a minimum, such confounding should be addressed in future studies.

⁶⁴ CATF Report, p. 2

⁶⁵ CATF also calls this a “premature mortality factor” in the technical reports that purportedly support the CATF Report.

EPA, which stands alone among federal regulatory agencies⁶⁶ on its “likely” carcinogenicity finding, has made estimates that are dramatically less dire than CATF’s predictions. When EPA’s estimate of premature mortalities per ton of diesel emissions⁶⁷ is compared to CATF’s, it yields a dramatically lower concentration/response function than does CATF. In fact CATF’s concentration/response function predicts 14 times more deaths (*i.e.*, CATF’s estimate of 100,000 additional deaths) between 2005–2030 than does EPA’s data. CATF’s estimate is based on CATF’s misuse of the Pope Study, *i.e.*, attributing a 4 percent increase in diesel-induced premature mortality to each 10 ug/m³ increase in PM_{2.5} in a particular locality. As explained earlier, the Pope Study cannot be used to support causation as CATF has chosen to use it.

- d) CATF’s estimate of 100,000 premature deaths is additionally inflated by at least 50 percent because it is in error in one of the cardinal assumptions made regarding the typical life of a diesel engine, which CATF estimates to be 30 years.⁶⁸ According to the Diesel Technology Forum, a trade group, CATF’s 30-year claim is based on an outdated Department of Energy study that was completed 15 years ago.⁶⁹ Instead of a useful life of 30 years, EPA now typically estimates that a fleet turns over every 13 to 15 years.⁷⁰ Moreover, 160,000 retrofits of existing diesel engines have already occurred, and many more are in progress nationwide.⁷¹ As importantly, CATF proposes that all 13 million existing diesel engines be retrofitted, making no distinction between the age of the engine. It is one thing to retrofit an engine manufactured in the late 1980s, but altogether another to retrofit an

engine manufactured in the early 2000s. Industry estimates are that diesel emissions were reduced 80 percent over that 15-year span.⁷² Consequently, CATF’s proposal is not likely to create anything close to the emission reductions that it claims will occur.

- e) This is further reflected in CATF’s modeled estimates of theoretical emissions resulting from EPA’s Clean Diesel Rules. Predictably, CATF’s estimates are far more exaggerated than EPA’s. Specifically, EPA estimates that its Clean Diesel rules will achieve 55 percent greater PM_{2.5} reductions during the years 2005–2030 than CATF would credit to these rules.⁷³ Moreover, if EPA is correct with its emission reduction estimates (6,188,000 tons of PM_{2.5} emission reductions during this period), in light of the fact that CATF’s total baseline diesel emission estimates for the years 2005–2030 are 7,095,021 tons, the theoretical limit of any emission reductions that CATF’s proposal could achieve is only 907,021 tons. This is only 51 percent of the total emission reductions that CATF claims would result from its proposal (1,784,301 tons). Accordingly, if EPA is correct about the effectiveness of its Clean Diesel rules, this in turn means that, on a per ton of pollutant basis, CATF’s proposal is actually twice as costly as the CATF Report claims.
- f) Finally, it is noteworthy that CATF’s prediction of 21,000 premature deaths for year 2010 is wholly inconsistent with its estimate of 100,000 premature deaths during the 2005–2030 period. CATF’s estimate of 100,000 premature deaths during the 2005–2030 period is predicated on CATF’s concentration/response function that it derived from 1999 data. Instead of using this concentration/response function (0.0445), CATF’s

⁶⁶ Other federal agencies with responsibility for making such determinations—OSHA, MSHA, ATSDR—have uniformly declined to give such a designation to diesel exhaust. Two agencies have merely placed DPM on a suspect list. In 1988, NIOSH classified DPM as a “potential” human carcinogen, but it has done nothing further with DPM. And, largely as a result of EPA’s classification, the National Toxicology Program of DHHS has described it as “reasonably anticipated to be a human carcinogen.”

⁶⁷ EPA estimates that its Clean Diesel rules will reduce premature mortalities by 20,300.

⁶⁸ CATF Report at p. 2.

⁶⁹ Nguyen, “Advocacy Group Stirs Up Diesel Issue” at http://fleetowner.com/news/topstory/truck_diesel_emission_study_clean_air_task_force_022305/

⁷⁰ *Id.*

⁷¹ http://www.dieselforum.com/statements/feb_22_2005-2.html.

⁷² *Id.* In 2002, EPA was estimating that fully 60 percent of diesel Class 7 and 8 trucks were 10 years old or less. EPA Health Assessment Document for Diesel Engine Exhaust (2002) at p. 2-7. While EPA is of the opinion that the useful life of a locomotive engine is 40 years, such engines are rebuilt several times during their lives, and during each such rebuilding, emission reduction changes are instituted so that the locomotives emit less DPM than before their engines were rebuilt. *Id.* at p. 2-9.

⁷³ EPA estimates that its Clean Diesel rules will eliminate 6,188,000 tons of PM_{2.5} emissions during this time period, while CATF would only credit EPA’s rules with emission reductions of 2,887,221 tons.

prediction of 21,000 diesel-induced deaths for year 2010 would produce a concentration/response function that is even more outlandish (0.0810).⁷⁴ To demonstrate the absurdity of CATF's position, while the concentration/response function that CATF elsewhere claims to be correct (0.0445) is 14 times larger than the concentration/response function that reflects EPA's estimates of premature mortality, this concentration/response function of 0.0810 would produce predictions of death that are nearly 25 times that which EPA would predict.⁷⁵ These claims of 21,000 premature deaths, then, are inconsistent with both CATF's other predictions and with EPA's estimates as well.⁷⁶

■ CONCLUSION

CATF, in its zeal to force EPA, state, and local air pollution control agencies to require retrofitting of existing diesel engines, has abandoned the rigors of sound science. We hope that this White Paper will encourage responsible environmental officials to reject CATF's suggestion. We also hope that CATF will revise the CATF Report to eliminate both the junk science and the irresponsible attacks on the diesel industry. The reality is that the diesel industry has done much, and will be doing a lot more, to reduce diesel emissions over the next 25 years. If even more is to be asked of the diesel industry, it should be because reliable science requires it, not because of CATF's junk science.

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⁷⁴ This number is derived by using CATF's own methodology: dividing CATF's estimated deaths from diesel exhaust in 2010 (21,000) by the tons of emissions that CATF predicts for 2010 (259,330).

⁷⁵ It is not as if EPA is somehow allied with the diesel industry; EPA is the only federal government agency that has gone so far as to classify diesel exhaust as a "likely" human carcinogen.

⁷⁶ Likewise CATF's prediction of 21,000 premature deaths for year 2010 is wholly inconsistent with another CATF estimate of premature deaths, as presented in Appendix, Table 2. Using CATF's own predictions, the highest estimate of premature mortality for the year 2010 in Table 2 is 12,667. (This number is found in the column entitled, "CATF Predicted Total Baseline Premature Mortality from CATF's Estimates of Emissions w/o either EPA Clean Diesel Rules or CATF's Proposed Additional Controls, Adjusted for Population," and discussed in paragraph T in the Appendix.) That CATF estimate, based on CATF's own "baseline" estimate of emissions (that would include neither the implementation of EPA's Clean Diesel rules nor CATF's proposal to retrofit existing diesel engines) and CATF's own concentration response function, predicts not 21,000 deaths, but rather 12,667. Moreover, as we have noted above, CATF's estimate of 12,667 premature deaths in 2010 is predicated on CATF's concentration/response function (0.0445) that yields a predicted premature mortality that is 14 times larger than EPA's data would yield. (EPA's data would yield a factor of 0.0032, using the identical CATF methodology for estimating premature mortality, as discussed in paragraph Z of the Appendix.) CATF's claim of 21,000 diesel-induced deaths in 2010 is inconsistent with the very concentration-response function (0.0445) that CATF uses to claim that if its proposal to retrofit existing diesel engines is not adopted, 100,000 people will lose their lives in the period of 2005–2030.

TABLE 1 EMISSION RATES OF PAH (MG/MI) FROM LD AND HD DIESEL VEHICLES

PAH	Light-duty diesel	Heavy-duty diesel
Naphthalene	5.554 ± 0.282	2.451 ± 0.154
2-Menaphthalene	3.068 ± 0.185	2.234 ± 0.152
1-Menaphthalene	2.313 ± 0.134	1.582 ± 0.103
Dimethylnaphthalenes	5.065 ± 0.333	2.962 ± 0.488
Biphenyl	0.743 ± 0.041	0.505 ± 0.037
2-Methylbiphenyl	0.203 ± 0.015	0.049 ± 0.024
3-Methylbiphenyl	1.048 ± 0.063	0.401 ± 0.036
4-Methylbiphenyl	0.447 ± 0.028	0.144 ± 0.021
Trimethylnaphthalenes	6.622 ± 0.563	1.940 ± 0.221
Acenaphthylene	0.422 ± 0.024	0.059 ± 0.087
Acenaphthene	0.06 ± 0.008	0.030 ± 0.040
Phenanthrene	1.411 ± 0.072	0.084 ± 0.011
Fluorene	0.442 ± 0.038	0.066 ± 0.022
Methylfluorenes	1.021 ± 0.091	0.071 ± 0.055
Methylphenanthrenes	1.115 ± 0.064	0.124 ± 0.069
Dimethylphenanthrenes	0.637 ± 0.047	0.090 ± 0.096
Anthracene	0.246 ± 0.025	0.052 ± 0.016
9-Methylanthracene	0.013 ± 0.002	0.434 ± 0.082
Fluoranthene	0.213 ± 0.014	0.044 ± 0.026
Pyrene	0.245 ± 0.020	0.071 ± 0.017
Methyl(pyrenes/fluoranthenes)	0.548 ± 0.045	0.022 ± 0.082
Benzoaphthothiophene	0.002 ± 0.002	0.001 ± 0.027
Benzo[a]anthracene	0.020 ± 0.005	0.066 ± 0.046
Chrysene	0.029 ± 0.005	0.009 ± 0.021
Benzo[b+j+k]fluoranthene	0.056 ± 0.005	0.009 ± 0.022
Benzo[e]pyrene	0.019 ± 0.003	0.010 ± 0.014
Benzo[a]pyrene	0.013 ± 0.004	0.013 ± 0.044
Indeno[1,2,3-cd]pyrene	0.010 ± 0.003	0.001 ± 0.037
Dibenzo[a]anthracene	0.002 ± 0.003	0.000 ± 0.053
Benzo[b]chrysene	0.001 ± 0.002	0.001 ± 0.027
Benzo[ghi]perlyne	0.018 ± 0.004	0.013 ± 0.048
Coronene	0.006 ± 0.006	0.001 ± 0.095

From EPA Health Assessment
 Document for Diesel Engine Exhaust (2002)
 at p. 2-73 (Table 2-14)

TABLE 2

A	B	C	D	E	F	G	H	I	J	K	L	M	N
Year	CATF Estimate of Population (millions)	CATF's "Baseline" Estimate of Emissions w/o EPA Clean Diesel Rules or CATF's Proposed Additional Controls (PM _{2.5} Tons) ^b	CATF's Estimate of Emissions w/EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM _{2.5} Tons) ^c	CATF's Estimate of Net Emission Reductions w/EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM _{2.5} Tons)	EPA's Estimate of Net Emission Reductions Solely from EPA's Nonroad Diesel Rules (PM _{2.5} Tons) ^d	EPA's Estimate of Net Emission Reductions Solely from EPA's Heavy-Duty Diesel Truck and Bus Rules (PM _{2.5} Tons) ^e	EPA's Estimate of Net Emission Reductions Solely from EPA's Clean Diesel Rules (PM _{2.5} Tons)	EPA's Estimate of Net Emissions w/EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM _{2.5} Tons)	CATF's Estimate of Net Emissions Using Both CATF's Estimate of EPA Clean Diesel Rules and CATF's Proposed Additional Controls (PM _{2.5} Tons) ^f	CATF's Estimate of Net Reduction in Emissions Solely Through Adoption of CATF's Proposed Additional Controls (PM _{2.5} Tons) ^g	CATF's Estimate of Net Reduction in Emissions Using both EPA's Clean Diesel Rules and CATF's Proposed Additional Controls (PM _{2.5} Tons) ^h	CATF's Predicted Premature Mortality Factor /1000 Tons (Per 1999 Data) ⁱ	CATF Predicted Premature Mortality from EPA Baseline Emissions w/EPA Clean Diesel Rules or CATF's Proposed Additional Controls /1000 Tons
1999	279 ^a												
2000	282 ^a												
2001	284 ^a												
2002	287 ^a												
2003	289 ^a												
2004	292 ^a												
2005	294	293,297	288,783	4,514	29,000	109,000	238,000	55,297	288,783	0	-50,783	0.045	13,062
2006	297	294,646	275,758	8,888	29,000	109,000	238,000	46,646	281,474	14,285	-23,474	0.045	12,677
2007	299	277,022	259,632	17,390	29,000	109,000	238,000	39,022	229,650	29,992	8,350	0.045	12,337
2008	301	270,724	243,606	27,118	29,000	109,000	238,000	32,724	197,827	45,780	40,773	0.045	12,057
2009	304	265,113	235,860	29,253	29,000	109,000	238,000	27,113	166,003	69,857	71,997	0.045	11,807
2010	306	259,330	227,004	32,326	29,000	109,000	238,000	21,330	129,665	97,339	108,335	0.045	11,550
2011	309	259,475	217,883	41,592	29,000	109,000	238,000	21,475	116,465	101,398	121,515	0.045	11,556
2012	311	259,659	206,683	52,976	29,000	109,000	238,000	103,306	103,306	103,377	134,694	0.045	11,584
2013	314	260,343	194,974	65,369	29,000	109,000	238,000	22,343	90,126	104,848	147,874	0.045	11,595
2014	316	261,279	183,094	78,185	29,000	109,000	238,000	23,279	76,947	106,147	161,063	0.045	11,636
2015	319	255,068	171,252	83,816	29,000	109,000	238,000	17,068	63,767	107,485	187,287	0.045	11,380
2016	322	256,814	153,768	97,046	29,000	109,000	238,000	18,914	59,813	99,956	0	0.045	11,437
2017	324	258,844	148,579	110,265	29,000	109,000	238,000	20,844	55,858	92,721	0	0.045	11,519
2018	327	260,923	138,227	122,696	29,000	109,000	238,000	22,923	51,904	86,323	0	0.045	11,620
2019	329	263,037	128,138	134,899	29,000	109,000	238,000	25,037	47,949	80,189	0	0.045	11,715
2020	332	265,372	118,683	146,689	29,000	109,000	238,000	27,372	43,995	74,668	0	0.045	11,819
2021	334	268,669	114,446	154,223	29,000	109,000	238,000	30,669	43,995	70,461	0	0.045	11,965
2022	337	271,988	110,583	161,405	29,000	109,000	238,000	33,988	43,995	66,988	0	0.045	12,113
2023	340	275,710	107,428	168,282	29,000	109,000	238,000	37,710	43,995	63,433	0	0.045	12,279
2024	342	279,083	104,156	174,927	29,000	109,000	238,000	41,083	43,995	60,161	0	0.045	12,430
2025	345	282,532	101,120	181,412	29,000	109,000	238,000	44,532	43,995	57,25	0	0.045	12,583
2026	348	285,991	98,365	187,626	29,000	109,000	238,000	47,991	43,995	54,370	0	0.045	12,737
2027	350	289,780	96,274	193,506	29,000	109,000	238,000	51,780	43,995	52,279	0	0.045	12,906
2028	353	293,241	94,222	199,019	29,000	109,000	238,000	55,241	43,995	50,227	0	0.045	13,080
2029	356	296,708	92,336	204,372	29,000	109,000	238,000	58,708	43,995	48,341	0	0.045	13,214
2030	358	300,563	90,946	209,617	29,000	109,000	238,000	62,663	43,995	46,951	0	0.045	13,366
TOTAL		7,095,021	4,207,800	2,887,221	3,354,000	2,834,000	6,188,000	907,021	2,423,502	1,784,301	907,021	0.045	315,983

a On page 6 of the Technical Support paper for the CATF Report, reference is made to "ASPEN 1999 Data." Although no population estimates are provided for 1999, the population projections contained in CATF's report suggest that the U.S. population increases at five million persons every two years, and has displayed such increase as an increase of three million followed by an increase of two million in the succeeding year. This convention was followed here to estimate that the population in 1999 was 279 million.

b CATF's table refers to this as "EPA Baseline."

c CATF's table refers to this as "EPA Control (PM_{2.5} Tons)."

d Source: USEPA Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420104040.htm>). EPA has only provided the estimated reductions in annual averages over the period of 2005–2030, and so that value (129,000 tons) was inserted in this table for each year in this time period.

e Source: USEPA Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420104040.htm>). Although the 109,000 ton annual reduction projected by EPA is for all Particulate Matter (and not just PM_{2.5}), EPA has stated "The REMSAD results indicate that the predicted change in PM concentration is composed almost entirely of reductions in fine particulates (PM_{2.5}), with little or no reduction in coarse particles (PM₁₀ less PM_{2.5}). Therefore, the observed changes in PM₁₀ are composed primarily of changes in PM_{2.5}." (See Heavy-Duty Standards/Diesel Fuel RIA, December 2000; EPA 420-R-00-026, at VII-22) As a consequence for purposes of this analysis, all of EPA's projected particulate matter reductions are assumed to be PM_{2.5}. Moreover, EPA has only provided the estimated reductions in annual averages over the period of 2005–2030, and so that value (109,000 tons) was inserted in this table for each year in this time period.

f CATF's table refers to this as "CATF Goal."

g CATF's table refers to this as "Tons Below BAU."

h Although CATF has proposed additional reductions of 2,423,503 tons during the period 2005–2030, in fact, EPA's calculations suggest that there will be only 907,021 tons of PM_{2.5} emissions remaining in the 2005–2030 time period as a result of implementation of its Clean Diesel rules. Accordingly, the maximum net reduction that, theoretically, could be achieved is 907,021. Consequently, in year 2015, the calculated net reduction is less than the difference between EPA's claimed reduction produced by its Clean Diesel rules and CATF's proposed additional controls. Moreover, commencing in 2016, zeros were entered for each year thereafter, because the 907,021 theoretical emission limit would have been reached in 2015.

i On page 6 of the CATF Report, CATF asserts that based on the ASPEN 1999 data, it could calculate a ratio of deaths per 1000 tons of PM_{2.5}, which it determined to be 0.045. That is the source of the value in this column.

Table continued on next page

TABLE 2 cont'd.

Year	A	O	P	Q	R	S	T	U	V	W	X	Y	Z
	CATF Predicted Premature Mortality from CATF's Estimates of Emissions w/ Solely EPA Clean Diesel Rules/ 1000 Tons	CATF Predicted Premature Mortality from CATF's Estimates of Emissions w/both EPA Clean Diesel Rules and CATF's Proposed Additional Controls/ 1000 Tons	CATF Predicted Difference in Premature Mortality Between CATF's Estimates of EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Unadjusted for Population	CATF Predicted Difference in Premature Mortality Between CATF's Estimates of EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Adjusted for Population	CATF Predicted Difference in Premature Mortality Between CATF's Estimates of EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Adjusted for Population	CATF Predicted Total Baseline Premature Mortality from CATF's Estimates of Emissions w/ either EPA Clean Diesel Rules or CATF's Proposed Additional Controls, Adjusted for Population	CATF Predicted Total Premature Mortality from CATF's Estimates of Emissions from EPA Clean Diesel Rules, Adjusted for Population	CATF Predicted Total Premature Mortality from CATF's Estimates of Emissions from both EPA Clean Diesel Rules and CATF Proposed Additional Controls, Adjusted for Population	EPA's Predicted Reduced Total Premature Mortality from Solely EPA's Estimates of Emissions from Nonroad Diesel Rule	EPA's Predicted Total Reduced Premature Mortality from EPA's Estimates of Emissions of Heavy-Duty Diesel Truck and Bus Rule	EPA's Predicted Total Reduced Premature Mortality from EPA's Estimates of Emissions of Clean Diesel Rules	EPA's Predicted Premature Mortality Factor/1000 Tons	
1999													
2000													
2001													
2002													
2003													
2004													
2005	12,861	12,861	0.000	0.000	0.000	0.000	13,764,523	13,552,680	13,552,680				
2006	12,281	11,645	0,636	636,151	677,193	13,494,841	13,073,468	12,396,274					
2007	11,563	10,228	1,335	1,335,276	1,430,995	13,221,833	12,391,585	10,960,841					
2008	10,849	8,810	2,039	2,038,810	2,193,576	13,007,689	11,704,711	9,565,135					
2009	10,504	7,933	3,111	3,111,746	3,389,922	12,865,031	11,445,482	8,855,580					
2010	10,110	5,775	4,335	4,335,082	4,754,606	12,667,194	11,089,203	6,333,597					
2011	9,704	5,188	4,516	4,516,863	5,001,429	12,796,534	10,747,020	5,745,591					
2012	9,205	4,601	4,604	4,603,990	5,132,046	12,890,507	10,260,867	5,128,821					
2013	8,683	4,014	4,670	4,669,502	5,255,282	13,049,137	9,772,655	4,517,373					
2014	8,154	3,427	4,727	4,727,354	5,354,279	13,179,466	9,235,649	3,881,370					
2015	7,627	2,840	4,787	4,786,943	5,473,243	12,988,317	8,720,323	3,247,079					
2016	7,115	2,664	4,452	4,451,988	5,137,675	13,200,208	8,212,056	3,074,381					
2017	6,617	2,488	4,129	4,129,415	4,795,450	13,376,843	7,684,377	2,888,927					
2018	6,166	2,312	3,844	3,844,474	4,505,869	13,519,662	7,216,175	2,709,286					
2019	5,707	2,135	3,571	3,571,291	4,211,307	13,613,964	6,729,458	2,518,150					
2020	5,286	1,959	3,328	3,328,299	3,958,176	14,063,694	6,269,742	2,331,566					
2021	5,087	1,959	3,138	3,137,800	3,756,123	14,324,196	6,101,735	2,345,611					
2022	4,925	1,959	2,966	2,965,558	3,582,054	14,531,399	5,948,733	2,366,679					
2023	4,784	1,959	2,825	2,825,047	3,442,710	14,983,654	5,830,457	2,387,748					
2024	4,639	1,959	2,679	2,679,325	3,284,394	15,236,382	5,686,128	2,401,793					
2025	4,503	1,959	2,544	2,544,114	3,145,948	15,559,404	5,568,910	2,422,882					
2026	4,381	1,959	2,421	2,421,418	3,020,283	15,866,851	5,484,183	2,443,930					
2027	4,268	1,959	2,328	2,328,293	2,920,798	16,089,844	5,378,774	2,457,976					
2028	4,196	1,959	2,237	2,236,906	2,830,207	16,523,636	5,309,251	2,479,044					
2029	4,112	1,959	2,163	2,162,911	2,747,083	16,861,083	5,247,196	2,500,112					
2030	4,050	1,959	2,091	2,091,006	2,665,063	17,176,108	5,197,241	2,514,158					
TOTAL	187,398	107,933	79,465	79,465,353	92,669,673	369,353,981	219,855,917	121,865,245	8,300,000	20,300,000	0.003		

■ APPENDIX

Here is how to read our Table 2. The information that was directly provided by CATF is shaded in Table 2. Most of the remaining information in Table 2 is either based on CATF's estimates or on EPA's estimates, and each is specified therein. Going from the first column on the far left to the last column on the far right, this is how we constructed Table 2:

A. Year. CATF provided information for only years 2005–2030 (CATF's "An Analysis of Diesel Air Pollution and Public Health in America," pp. 5-6). However, CATF bases its dire prediction of 100,000 "saved lives" on the use of data from 1999, and so it was necessary to have population data from 1999 in order to test CATF's math. Accordingly, we have expanded the table to encompass data back to year 1999.

B. CATF Estimate of Population (millions). CATF provided population estimates for every year from 2005–2030. However, CATF's predictions are dependent on the population in 1999, which it did not provide in either the CATF Report or any technical documents that CATF has made publicly available purportedly supporting the CATF Report. While CATF did not provide the source for its population estimates for 2005–2030, CATF did follow a definite convention in its population estimates: CATF estimated that the population would increase by five million every two years and predicted further that this five million increase would occur by an increase of three million in each even year and by two million in each odd year. Accordingly, in order to have consistency with CATF's population estimates, we backtracked from 2005 to 1999 by following CATF's convention, which yields a population estimate for 1999 of 279 million.

C. CATF's "Baseline" Estimate of Emissions w/o EPA Clean Diesel Rules or CATF's Proposed Additional Controls (PM_{2.5} Tons). CATF prepared an estimate of the tons of PM_{2.5} that would be emitted during the time period 2005–2030, if neither EPA's Clean Diesel rules nor CATF's proposed existing engine retrofit proposal were implemented. CATF refers to this estimate as a "baseline." (CATF's "An Analysis of Diesel Air

Pollution and Public Health in America," p. 5). The numbers appearing in this column are exactly the same as those CATF used in its predictions.

D. CATF's Estimate of Emissions w/ EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM_{2.5} Tons). CATF also prepared an estimate of the tons of PM_{2.5} that would be emitted during the time period 2005–2030, if EPA's Clean Diesel rules were implemented but CATF's proposed existing engine retrofit proposal was not adopted. CATF refers to this estimate as a "Business as Usual," or "EPA Control" scenario. (CATF's "An Analysis of Diesel Air Pollution and Public Health in America," p. 5). The numbers appearing in this column are exactly the same as those CATF used in its predictions.

E. CATF's Estimate of Net Emission Reductions w/EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM_{2.5} Tons). Although CATF did not include the calculations in this column in its report, the data in this column are derived solely from CATF's estimates. Specifically, the numbers appearing in this column represent the reductions in emissions that CATF predicts will occur from the implementation of EPA's Clean Diesel rules. It is included in Table 2 because CATF's predictions are wildly at odds with EPA's own predictions of the emission reductions that will be achieved from the application of the Clean Diesel rules. EPA's predictions are set forth in the succeeding three columns to the right in Table 2.

F. EPA's Estimate of Net Emission Reductions Solely from EPA's Nonroad Diesel Rules (PM_{2.5} Tons). Significantly, the data in this column, as well as the succeeding three columns, were not even mentioned in CATF's Report. The data is not difficult to acquire; the source of the data is EPA itself. Specifically, EPA has published a fact sheet predicting the net emission reductions that will result from its nonroad diesel rules, which is one part of its Clean Diesel rules. In that fact sheet, EPA has estimated that, during the period 2005–2030, the implementation of these rules will yield annual reductions of 129,000 tons. See USEPA

Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420f04040.htm>). Because EPA has only provided the estimated reductions in annual averages over the period of 2005–2030, that value (129,000 tons) was inserted in this table for each year in this time period. Over this span, EPA's total predicted reduction is 3,354,000 tons.

G. EPA's Estimate of Net Emission Reductions Solely from EPA's Heavy-Duty Diesel Truck and Bus Rules (PM_{2.5} Tons).

Also included in the same EPA fact sheet is a prediction of the net emission reductions that will result from the implementation of EPA's heavy-duty diesel truck and bus rules, another part of EPA's Clean Diesel rules. Specifically, EPA has estimated that, during the period 2005–2030, compliance with these rules will yield annual reductions of 109,000 tons. See USEPA Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420f04040.htm>). Although the 109,000 ton annual reduction projected by EPA is for all Particulate Matter and not just PM_{2.5}, EPA has stated that the modeled results it adopted “indicate that the predicted change in PM concentrations is composed almost entirely of reductions in fine particulates (PM_{2.5}), with little or no reduction in coarse particles (PM₁₀ less PM_{2.5}). Therefore, the observed changes in PM₁₀ are composed primarily of changes in PM_{2.5}.” (See Heavy-Duty Standards/Diesel Fuel RIA, December 2000; EPA 420-R-00-026, at VII-22). As a consequence, for purposes of this analysis, all of EPA's projected particulate matter reductions related to this rule are assumed to be PM_{2.5}. Again, because EPA has only provided the estimated reductions in annual averages over the period of 2005–2030, that value (109,000 tons) was inserted in this table for each year in this time period. Over this span, EPA's total predicted reduction is 2,834,000 tons.

H. EPA's Estimate of Net Emission Reductions Solely from EPA's Clean Diesel Rules (PM_{2.5} Tons). The numbers in this column are simply the result of adding together the net emission reductions EPA predicts from both the nonroad sources and the heavy-duty diesel truck and bus sources that are set forth

in the preceding two columns to the left. EPA is predicting that the net emissions from these two portions of its Clean Diesel rules will produce total net emission reductions of 6,188,000 tons over the period of 2005–2030. In effect, CATF's predictions of the effectiveness of EPA's controls is dramatically lower than EPA's—less than one-half, or to be more precise, CATF prediction (2,887,221 tons) is only 46.66 percent of EPA's prediction.

I. EPA's Estimate of Net Emissions w/ EPA Clean Diesel Rules and w/o CATF's Proposed Additional Controls (PM_{2.5} Tons).

The numbers in this column represent the difference between (1) CATF's prediction of the “baseline” emissions that would occur during the period of 2005–2030 if neither EPA's Clean Diesel rules nor CATF's proposed existing engine retrofit proposal were to happen, and (2) EPA's estimate of the net reduction in emissions that would result from the implementation of its Clean Diesel rules. The total difference between these two estimates is only 907,021 tons. This is very important for considering the potential benefits that might be obtained from adoption of CATF's proposal to retrofit existing diesel engines with additional controls. If EPA's prediction of the emission reductions to be achieved from its Clean Diesel rules is correct, it means that less than one million tons of PM_{2.5} (much of which is not produced from diesel engines) will be emitted over the period of 2005–2030. That is an average of only 34,885 tons per year. Stated otherwise, the theoretical maximum annual average reduction that could be achieved from CATF's proposal of retrofitting existing diesel engines is only 34,885 tons of DPM.

J. CATF's Estimate of Net Emissions Using Both CATF's Estimate of EPA Clean Diesel Rules and CATF's Proposed Additional Controls (PM_{2.5} Tons).

CATF also prepared an estimate of the tons of PM_{2.5} that would be emitted, during the time period 2005–2030, if both EPA's Clean Diesel rules are implemented and if CATF's proposed existing engine retrofit proposal were adopted. CATF refers to this estimate as the “CATF Goal” (CATF's “An Analysis of Diesel Air

Pollution and Public Health in America,” p. 5). The numbers appearing in this column are exactly the same as CATF used in its predictions.

K. CATF’s Estimate of Net Reduction in Emissions Solely Through Adoption of CATF’s Proposed Additional Controls (PM_{2.5} Tons). CATF also prepared an estimate of the tons of PM_{2.5} emissions that would be eliminated, during the period 2005–2030, if CATF’s proposed existing engine retrofit proposal were adopted. CATF refers to this estimate as “Tons Below BAU,” or tons below business as usual, *i.e.*, the reductions it claims will result from its proposal in addition to those reductions that CATF claims EPA’s Clean Diesel rules will produce (CATF’s “An Analysis of Diesel Air Pollution and Public Health in America,” p. 5). The numbers appearing in this column are exactly the same as those CATF used in its predictions.

L. CATF’s Estimate of Net Reduction in Emissions Using Both EPA’s Estimate of EPA Clean Diesel Rules and CATF’s Proposed Additional Controls (PM_{2.5} Tons). Although CATF estimated its proposed additional reductions would produce 1,784,301 tons of emission reductions during the period 2005–2030, in fact, EPA’s calculations suggest that there will be only 907,021 tons of PM_{2.5} emissions that will remain in the 2005–2030 time period as a result of implementation of its Clean Diesel rules. Accordingly, if EPA is correct, the maximum net reductions that, theoretically, could be achieved through CATF’s retrofit of existing diesel engines proposal is 907,021 tons. Consequently, in Table 2, the calculated net reduction for year 2015 is less than the difference between EPA’s claimed reduction produced by its Clean Diesel rules and CATF’s proposed additional controls. Moreover, commencing in 2016, zeros were entered for each year thereafter because the 907,021 theoretical emission limit would have already been reached in 2015. This means that, if EPA is correct in its estimates of the emission reductions that will result from its Clean Diesel rules, by no later than 2016, CATF’s proposal would cease to produce meaningful emission reductions.

M. CATF’s Predicted Premature Mortality Factor/1000 Tons (Per 1999 Data). CATF asserts that based on some “modeled ASPEN 1999 directly emitted diesel particulate matter,” it can calculate a ratio of deaths per 1000 tons of PM_{2.5} emissions. Solely for purposes of constructing Table 2 to check CATF’s math, we have simply accepted CATF’s estimates of 1999 data—specifically, that in 1999 there were 357,352 tons of PM_{2.5} emitted and that there were 15,915 premature mortalities attributed to diesel exhaust in 1999 (CATF’s “An Analysis of Diesel Air Pollution and Public Health in America,” p. 6). To calculate this factor, CATF has divided the total premature mortalities it characterizes as attributable to diesel exhaust by the total PM_{2.5} emissions and arrived at a “factor” of 0.0445 deaths/1000 tons of PM_{2.5} emitted. Accordingly, this concentration response function, or premature mortality factor (0.0445), appears in each cell of this column.

N. CATF Predicted Premature Mortality from EPA Baseline Emissions w/o EPA Clean Diesel Rules or CATF’s Proposed Additional Controls/1000 Tons. The numbers in this column reflect simple multiplication of CATF’s purported concentration response function (0.0445) times the “baseline” emissions (and divided by 1000) that CATF projects would occur if neither EPA Clean Diesel rules nor CATF’s retrofit of existing diesel engines proposal were adopted. Both the factor and the baseline emission projections are set forth in CATF’s “An Analysis of Diesel Air Pollution and Public Health in America,” pp. 5-6.

O. CATF Predicted Premature Mortality from CATF’s Estimates of Emissions w/ Solely EPA Clean Diesel Rules/1000 Tons. Likewise, the numbers in this column reflect simple multiplication of CATF’s purported concentration response function (0.0445) times CATF’s estimates of the emissions (and divided by 1000) that CATF projects would occur if only EPA’s Clean Diesel rules (but not CATF’s retrofit of existing diesel engines proposal) were adopted. Both the factor and the estimated emissions are set forth in CATF’s “An Analysis of Diesel Air Pollution and Public Health in America,” pp. 5-6.

P. CATF Predicted Premature Mortality from CATF's Estimates of Emissions w/both EPA Clean Diesel Rules and CATF's Proposed Additional Controls/1000 Tons. Again, the numbers in this column reflect simple multiplication of CATF's purported concentration response function (0.0445) times CATF's estimates of the emissions (and divided by 1000) that CATF projects would occur if both EPA's Clean Diesel rules and CATF's retrofit of existing diesel engines proposal were adopted. Both the factor and the estimated emissions are set forth in CATF's "An Analysis of Diesel Air Pollution and Public Health in America," pp. 5-6.

Q. CATF Predicted Difference in Premature Mortality Between CATF's Estimates of EPA Clean Diesel Rules and CATF's Proposed Additional Controls/1000 Tons. The numbers in this column are simply the difference between the two preceding columns, *i.e.*, CATF's predictions of mortality (divided by 1000 tons) between its projections of PM_{2.5} emissions if EPA's Clean Diesel rules are implemented and its projections if both EPA's Clean Diesel rules and CATF's retrofit of existing diesel engines proposal are implemented.

R. CATF Predicted Difference in Premature Mortality Between CATF's Estimates of Emissions w/ EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Unadjusted for Population. The four columns immediately to the left of this one reflect CATF's predicted mortalities per 1000 tons of PM_{2.5} emissions. To test CATF's claim of 100,000 "saved lives," it is necessary to convert the numbers in the immediately preceding columns. Accordingly, the numbers in this column are the result of multiplying the numbers in the preceding column by 1000. The numbers in this column are not adjusted for the population increases that CATF predicts will occur during the 2005–2030 period; this occurs in the succeeding columns.

S. CATF Predicted Difference in Premature Mortality Between CATF's Estimates of Emissions from EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Adjusted for Population. This column calculates CATF's predictions of "saved lives," *i.e.*, the

mortalities that CATF claims will not occur if its retrofit of existing diesel engines proposal is adopted. The numbers stated here, for each year during the period 2005–2030, are based on both CATF's estimates of PM_{2.5} emissions in each such year and CATF's projections of population increases during this same time. Although CATF states that adoption of its proposal would produce 100,000 "saved lives" (CATF Report, p. 2), in fact, even its math yields only "95,000" (see CATF's "An Analysis of Diesel Air Pollution and Public Health in America," p. 5). Our calculations are that CATF's formula yields neither 100,000 nor 95,000, but instead 92,689. (Table 2, total in column labeled "CATF Predicted Difference in Premature Mortality Between CATF's Estimates of Emissions from EPA Clean Diesel Rules and CATF's Proposed Additional Controls, Adjusted for Population"). This is set forth in the total at the bottom of this column.

T. CATF Predicted Total Baseline Premature Mortality from CATF's Estimates of Emissions w/o either EPA Clean Diesel Rules or CATF's Proposed Additional Controls, Adjusted for Population. This column calculates CATF's predictions of the premature mortalities that will occur under CATF's estimates of the "baseline" emissions, *i.e.*, if neither EPA's Clean Diesel rules nor CATF's retrofit of existing diesel engines proposal is implemented. The numbers in this column reflect the product of multiplying the numbers in the column entitled "CATF Predicted Premature Mortality from EPA Baseline Emissions w/o EPA Clean Diesel Rules or CATF's Proposed Additional Controls/1000 Tons" (and described in paragraph N, above) by 1000 and adjusting these numbers as well to reflect CATF's estimated population increases during the period of 2005–2030.

U. CATF Predicted Total Premature Mortality from CATF's Estimates of Emissions from EPA Clean Diesel Rules, Adjusted for Population. Similarly, this column calculates CATF's predictions of the premature mortalities that will occur under CATF's estimates of the emissions that would occur if EPA's Clean Diesel rules (but not CATF's retrofit of existing diesel engines proposal)

is implemented. The numbers in this column reflect the product of multiplying the numbers in the column entitled “CATF Predicted Premature Mortality from CATF’s Estimates of Emissions w/ Solely EPA Clean Diesel Rules/1000 Tons” (and described in paragraph O above) by 1000 and adjusting these numbers as well to reflect CATF’s estimated population increases during the period of 2005–2030.

V. CATF Predicted Total Premature Mortality from CATF’s Estimates of Emissions from both EPA Clean Diesel Rules and CATF Proposed Additional Controls, Adjusted for Population.

Likewise, this column calculates CATF’s predictions of the premature mortalities that will occur under CATF’s estimates of the emissions that would occur if both EPA’s Clean Diesel rules and CATF’s retrofit of existing diesel engines proposal are implemented. The numbers in this column reflect the product of multiplying the numbers in the column entitled “CATF Predicted Premature Mortality from CATF’s Estimates of Emissions w/both EPA Clean Diesel Rules and CATF’s Proposed Additional Controls/1000 Tons” (and described in paragraph P above) by 1000 and adjusting these numbers as well to reflect CATF’s estimated population increases during the period of 2005–2030.

W. EPA’S Predicted Total Reduced Premature Mortality from Solely EPA’s Estimates of Emissions from Nonroad Diesel Rule.

This column contains only one number, expressed as a total. It is the estimate that EPA has made of the number of premature mortalities that will be reduced during the period 2005–2030 if its Nonroad Diesel rules are implemented. See USEPA Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420f04040.htm>).

X. EPA’S Predicted Total Reduced Premature Mortality from Solely EPA’s Estimates of Emissions of Heavy-Duty Diesel Truck and Bus Rule.

This column contains only one number, expressed as a total. It is the estimate that EPA has made of the number of pre-

mature mortalities that will be reduced during the period 2005–2030 if its Heavy-Duty Diesel Truck and Bus rules are implemented. See USEPA Clean Diesel Programs Facts and Figures (<http://www.epa.gov/cleandiesel/420f04040.htm>).

Y. EPA’s Predicted Total Reduced Premature Mortality from EPA’s Estimates of Emissions of Clean Diesel Rules.

This column also contains only one number: It is simply the sum of the numbers in the two immediately preceding columns.

Z. EPA’s Predicted Premature Mortality Factor/1000 Tons.

As noted in paragraph M, above, and as reflected in the column entitled “CATF’s Predicted Premature Mortality Factor/1000 Tons (Per 1999 Data), CATF asserts that it was able to calculate a concentration response function per 1000 tons of PM_{2.5} emissions by dividing the total premature mortalities it characterizes as attributable to diesel exhaust by the total PM_{2.5} emissions. CATF arrived at a “factor” of 0.0445 deaths/1000 tons of PM_{2.5} emitted. EPA’s estimates, on the other hand, yield a dramatically lower factor than that used by CATF. Following CATF’s own formulation, by dividing 20,300 (the total premature mortalities that EPA estimates will be reduced from 2005–2030 if its Clean Diesel Rules are implemented; see paragraph Y above) by 6,188,000 (the emissions that EPA estimates will be eliminated from 2005–2030 if its Clean Diesel Rules are implemented; see paragraph H above), the factor is 0.0033. Thus, CATF has selected an estimate of premature mortality that is nearly 1,477 times as high as that used by EPA in its estimate. Even using CATF’s outlandish emission estimates, its estimate of the “saved lives” that would result from the adoption of its retrofit of existing diesel engines proposal would not be 100,000, but would instead be only 6,827.58 (*i.e.*, 92,689.6725, from paragraph S above). If EPA’s emission reduction estimates are used with this factor, CATF’s “saved lives” claim over this period would instead be 2,555.29.

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