OREGON TRUCKING ASSOCIATIONS, INC.

The Senate Environment and Natural Resources Committee
Testimony of Bob Russell
Vice President Government Affairs
Senate Bill 824
April 1, 2015

I would like to start with the preamble of Senate Bill 824 because I believe that it is incomplete at best. The first sentence says, "Whereas Oregon ranks sixth highest in the nation for risk to human health from diesel pollution…" On its face, this statement is difficult to believe since Oregon is a relatively small state in terms of economic activity that requires the use of diesel engines. This claim comes from a 2005 study, entitled *An Analysis of Diesel Air Pollution and Public Health in America,* conducted by the Clean Air Task Force. This study was based on 1999 data. The data was then projected forward to reach the conclusion above. The problem is that the world of diesel engines has changed significantly since 2005 much less 1999.

Attached, is the document from the Clean Air Task Force that concludes that Oregon ranks sixth highest in nation for health risks from diesel emissions. There is a map, at page 3, that shows Oregon as one of the unhealthiest states in the nation. Compare that to the EPA map attached, at page 4, for PM-2.5 emissions, most commonly associated with diesel engines, that shows that Oregon has no nonattainment areas. Also attached, at page 5, is an EPA 2015 map for PM-10 emissions that shows that Lane County Oregon has a moderate emissions problem. PM-10 data also includes PM-2.5. Particulate matter emissions are often associated with health concerns. The data includes particulates from all sources including wood stoves, forest fires, the burning of grass straw, diesel emissions and even roadway particles chipped up from the use of studded tires.

Why the difference? Both DEQ and EPA acknowledge that particulate emissions have been significantly reduced in recent years. Newer wood stoves have particulate filters, grass straw burning has been significantly

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curtailed and 2007 and newer truck engines have 95% less particulate emissions compared to 2001 models. A recent Health Effects Institute study found that there is no link between the exhaust from 2007 engines and cancer. (See attached at page 6.) This study also acknowledges that the approximate 95% reduction in NOx emissions from 2010 and newer truck engines has further reduced the health impacts of diesel exhaust.

The trucking industry has made significant strides to reduce emissions from heavy-duty diesel trucks including the following regulations, incentives and research:

Regulatory

EPA required pollution control technology on 2010 and newer truck engines that reduce particulates and NOx by approximately 95% compared to engines built in 2001.

EPA regulation requiring truck manufacturers to improve heavy truck average MPG by 20% by 2018.

EPA Renewable Fuel Standard requiring increased use of biofuels including biodiesel.

Oregon Renewable Fuel Standard requiring diesel blends that include 5% biodiesel.

Oregon truck idling regulation that sets a basic standard of 5 minutes of idling or less in any given 60-minute period.

Oregon Low Carbon Fuel Standard that will reduce the carbon emissions form transportation fuels by 10% over a 10-year period.

Incentives

EPA SmartWay program that assists trucking companies to adopt fuel saving technologies.

Oregon Department of Energy 35% tax credits for the increased cost of purchasing natural gas powered trucks and for natural gas fueling stations.

Oregon PUC program that authorizes the agency to approve natural gas tariffs that provide a cost effective way to build natural gas fueling stations.

Research

EPA Super Truck Program that has provided $115 million in grants to truck manufacturers to develop heavy trucks that consume 50% less fuel.

These initiatives reduce emissions by reducing the amount of fuel consumed, changing the content of truck fuels and implementing technologies that reduce specific emissions. Taken together, these efforts have significantly reduced the threat to human health from diesel truck engines.

The remaining question is what is the number of heavy trucks operating in Oregon that have truck engines that are not equipped with the 2007 or newer emission reduction technologies? According to 2015 ODOT statistics, provided to Senator Dembrow, there are 314,001 heavy trucks operating on Oregon highways. Of these, 227,494 or 72%, have the 2007 or newer emission technologies. However, only 44.76% of the trucks operated by Oregon based trucking companies have 2007 or newer truck engines.

In an effort to deal with older diesel engines, the 2007 Legislature, with the support of the Oregon Trucking Associations, enacted House Bill 2172. This bill was known, at the time, as the Clean Diesel Bill. It established a fund to retrofit, repower or destroy older diesel engines. The priority was to reduce emissions from school busses. Unfortunately, this program has received very little support. The 2007 Legislature, through a budget note, transferred $500,000, from ODOT, to the Clean Diesel Fund. To my knowledge, this transfer has never been made. In addition, the Oregon Trucking Associations has repeatedly requested that DEQ include a Policy Option Package with its budget request to provide funding for the Clean Diesel Fund. To date, there have been no Policy Option Packages requesting money for the Clean Diesel Fund. DEQ's Clean Diesel Program has essentially operated with very small federal grants for the last 8 years. As a result, very little has been accomplished. This has been extremely disappointing particularly because of where we find ourselves today.

During the 2011 legislative session, House Bill 2081 was passed into law. This bill restricts truck idling. The basic standard is that it is unlawful to idle a truck for more than 5 minutes in any 60-minute period. This standard is more restrictive than idling regulations in California. The Oregon Trucking Associations also supported this bill. While the biggest deterrent to truck idling is the cost of fuel, our members believed that House Bill 2081 would provide an additional incentive, to our drivers, to curb unnecessary idling because they are subject to citation, by any law enforcement officer, for violations. SB 824 seeks to repeal the preemption of local governments from enacting their own anti-idling regulations. This would allow for a plethora of different idling regulations in Oregon. Most truck drivers

operating in Oregon are from other states. It is certainly possible to train them to comply with a single standard. However, if cities and counties adopt different standards, it will simply be impossible to expect our drivers to know about them much less comply.

The trucking industry currently transports approximately 75% of the tons of freight moving to, from and within Oregon. It is true that trucking moves Oregon's economy. Anything that damages Oregon's trucking industry will have a negative impact on Oregon's economy. In December of 2007, there were 9,578 Oregon based trucking companies. In December of 2014, there were 7,656. This represents a 21% reduction!!! Certainly, the Great Recession has decimated Oregon's trucking industry. Many companies that have survived have significantly reduced, if any, cash reserves. This Legislature has already passed Senate Bill 324 that will increase trucking's costs. It will not take much more to put many more Oregon based trucking companies out of business with potential disastrous results for Oregon's economy.

California has provided hundreds of millions on dollars in incentives to California's trucking industry before implementing new regulations to reduce diesel emissions from heavy trucks like those contemplated in SB 824. On the other hand, Oregon has not provided the needed support for the Clean Diesel Program that was established in 2007. The members of the Oregon Trucking Associations respectfully request that this Legislature amend and appropriate meaningful funds for the existing Clean Diesel Fund instead of enacting new regulations like those contained in Senate Bill 824.

Clean Air Task Force is a nonprofit organization dedicated to reducing atmospheric pollution through research, advocacy, and private sector collaboration.

Methane and Black Carbon

- Methane
- Black Carbon
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  - Diesel
  - Flaring
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- Arctic
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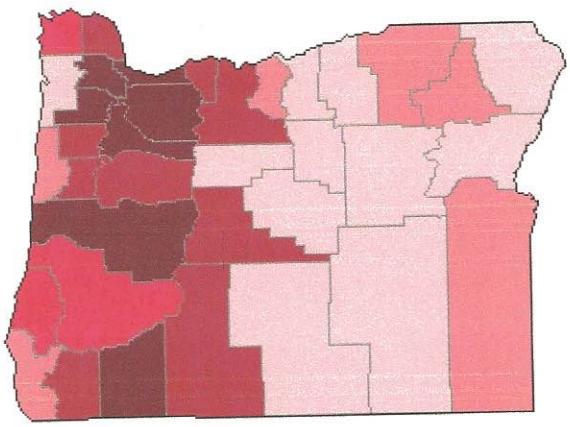
Diesel Soot Health Impacts

Diesel & Health in America:
Diesel Soot Health Impacts

Where You Live
Oregon
United States



Diesel Health Risk

Page 1

Lowest Impact  Highest Impact

How your risk may be higher depending on where you live and work

Annual Diesel Fine Particle Health Impacts (NATA 2005):

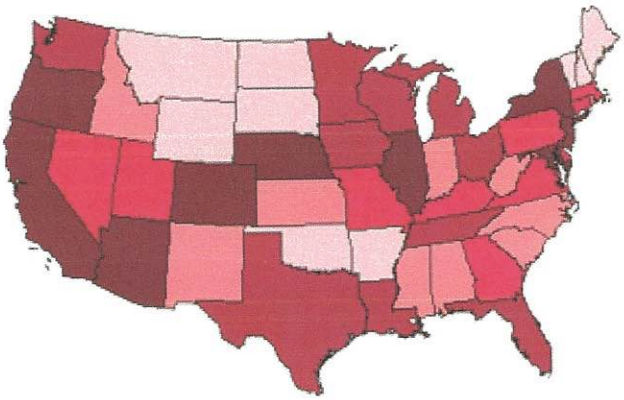
	Adults
176	Premature Deaths
145	Non-Fatal Heart Attacks
25,910	Work Loss Days (WLD)
151,520	Minor Restricted Activity Days (MRAD)
	Children
119	Asthma ER Visits
250	Acute Bronchitis
3,203	Lower Respiratory Symptoms
2,449	Upper Respiratory Symptoms
5,376	Asthma Exacerbation
	Monetized Value of Health Impacts
	$1.5 Billion

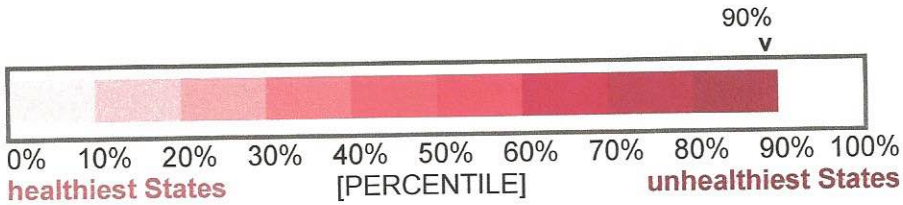
How the analysis was performed

How the Risk from Diesel Soot in Oregon Compares to the Other Lower 48 States and District of Columbia:

Ranking:*

6 of 49





What are percentiles?

Cancers per Million in Oregon:

192	55
Inhaled Diesel Soot	Other Inhaled Toxics

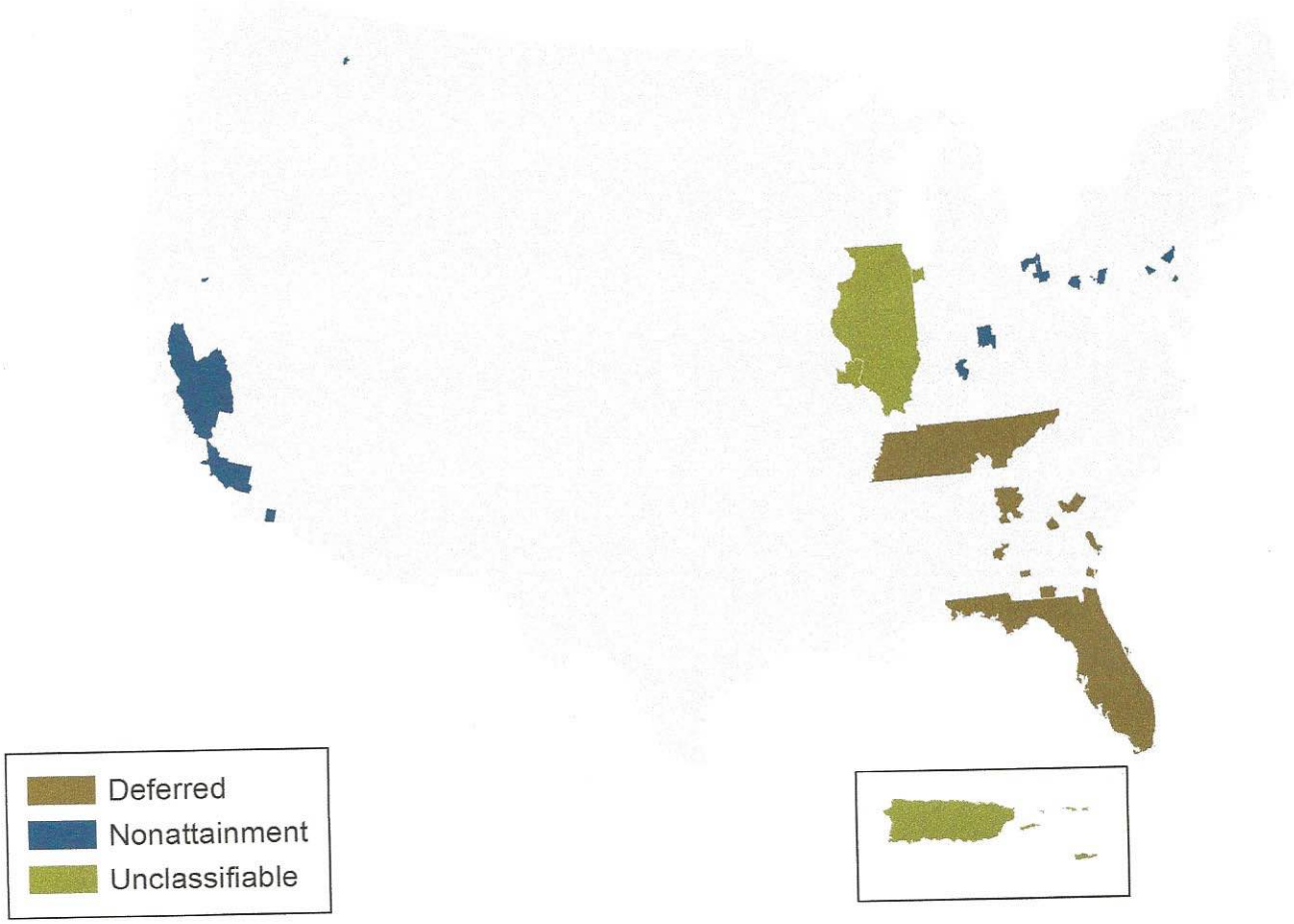
How did CATF compare the risk of diesel particulate to other air toxics?

Diesel Emissions (2005):

Pollutant	Annual Tons per Year	Highway (on road)	Heavy Equipment (non road)
NOx	49,991	30,956	19,034
PM2.5	2,895	1,120	1,775
PM10	3,113	1,282	1,830
CO	22,863	13,225	9,638
VOC	4,347	2,293	2,054
SO2	3,377	759	2,619

Page 3

2012 Annual PM2.5 Designations



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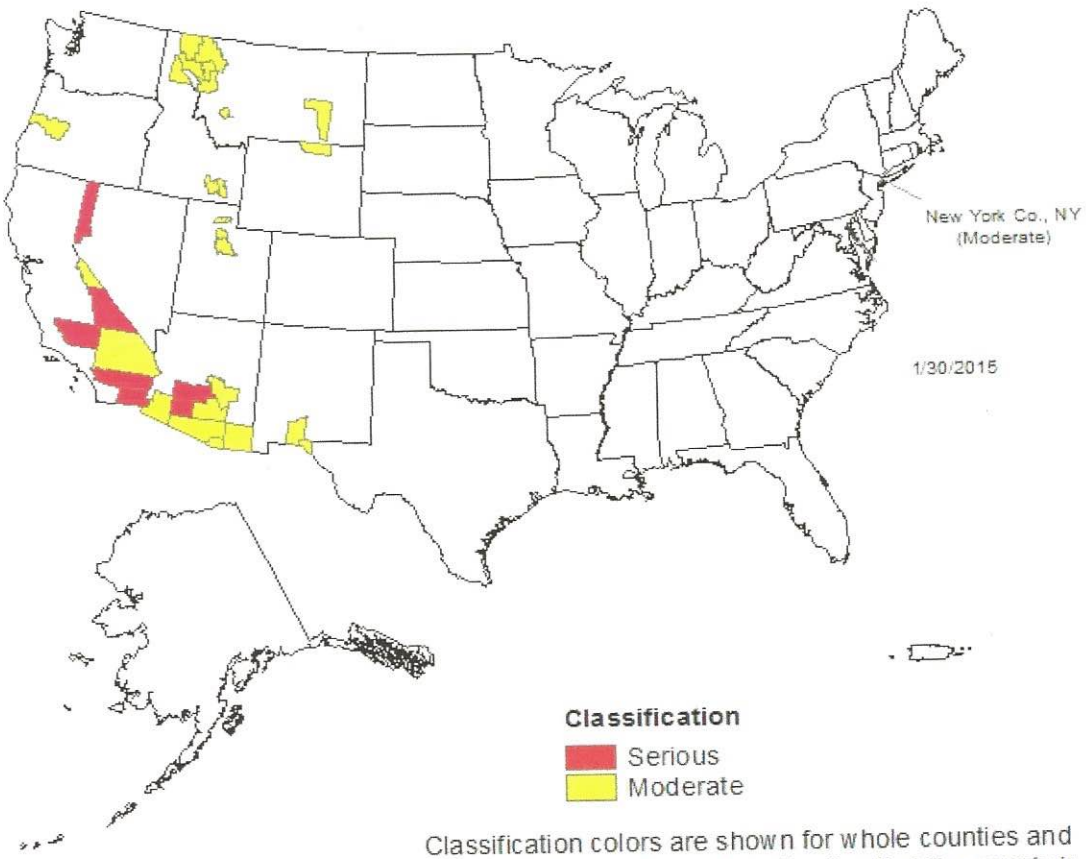
http://www.epa.gov/airquality/greenbook/mappm10.html
Last updated on January 30, 2015

Green Book

You are here: EPA Home Green Book PM-10 Area Map

View PDF map that can be zoomed

Counties Designated Nonattainment for PM-10



Classification colors are shown for whole counties and denote the highest area classification that the county is in

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HEI

STATEMENT

Synopsis of Research Report 184, Parts 1–4

HEALTH EFFECTS INSTITUTE

Effects of Lifetime Exposure to Inhaled New-Technology Diesel Exhaust in Rats

INTRODUCTION

This Statement summarizes HEI's independent evaluation, conducted by a specially convened Review Panel, of four studies conducted as a single phase (Phase 3B) of the Advanced Collaborative Emissions Study (ACES) program. The ACES Phase 3B studies investigated the health effects of chronic, lifetime exposures of rats (up to 30 months) and subchronic exposures (3 months) of mice to "new-technology diesel exhaust" (NTDE) — emissions from a heavy-duty diesel engine system compliant with 2007 U.S. Environmental Protection Agency (EPA) regulations. The studies were led by Drs. Jacob D. McDonald of the Lovelace Respiratory Research Institute (LRRI), Albuquerque, New Mexico, Jeffrey C. Bemis of Litron Laboratories, Rochester, New York, Lance M. Hallberg of the University of Texas Medical Branch, Galveston, Texas, and Daniel J. Conklin of the University of Louisville, Louisville, Kentucky.

BACKGROUND

In light of concerns identified over many decades about the potential health effects of diesel emissions, the U.S. EPA and the California Air Resources Board adopted stringent regulations for heavy-duty highway diesel engines, which were required to meet a new standard for particulate matter (PM) by 2007. A tighter standard for nitrogen oxides (primarily nitric oxide [NO] and nitrogen dioxide [NO_2]) came into effect in 2010. The regulatory agencies also mandated that sulfur in fuel be reduced substantially. To address these regulations and standards, motor vehicle and engine manufacturers introduced new technologies. These developments were expected to substantially reduce emissions from diesel engines.

To characterize the exhaust emissions from heavy-duty diesel engines that met the new standards and to assess the possible adverse health effects of exposure to these emissions, HEI, working in collaboration with the Coordinating Research

What This Study Adds

- This is the first study to conduct a comprehensive evaluation of lifetime inhalation exposure to emissions from heavy-duty 2007-compliant engines (referred to as "new-technology diesel exhaust," or NTDE).
- The study evaluated the long-term effects of multiple concentrations of inhaled NTDE, which has greatly reduced particle emissions compared with "traditional-technology diesel exhaust" (TDE) in male and female rats on more than 100 different biologic endpoints, including tumor development, and compared the results with biologic effects seen in earlier studies in rats after exposure to TDE.
- Lifetime inhalation exposure of rats exposed to one of three levels of NTDE from a 2007-compliant engine, for 16 hours per day, 5 days a week, with use of a strenuous operating cycle that more accurately reflected the real-world operation of a modern engine than cycles used in previous studies, did not induce tumors or pre-cancerous changes in the lung and did not increase tumors that were considered to be related to NTDE in any other tissue. A few mild changes were seen in the lungs, consistent with long-term exposure to NO_2, a major component of NTDE, which is being further substantially reduced in 2010-compliant engines.

This Statement, prepared by the Health Effects Institute, summarizes a research project funded by HEI and conducted by Drs. Jacob D. McDonald of the Lovelace Respiratory Research Institute, Albuquerque, New Mexico, Jeffrey C. Bemis of Litron Laboratories, Rochester, New York, and Lance M. Hallberg of the University of Texas Medical Branch, Galveston, Texas, and their colleagues, and Daniel J. Conklin and Maiying Kong of the University of Louisville, Louisville, Kentucky. The complete report, *Advanced Collaborative Emissions Study (ACES): Lifetime Cancer and Non-Cancer Assessment in Rats Exposed to New-Technology Diesel Exhaust* (© 2015 Health Effects Institute), can be obtained from HEI or our Web site (see last page).

ACES 184

Council, a nonprofit organization with expertise in emissions characterization, launched the multiphase ACES program. Phases 1 and 2 focused on emissions characterization, and Phase 3A established conditions for animal exposure. Phase 3B was designed to evaluate health outcomes in rats exposed to NTDE for up to 24 months, with the possibility of extension to 30 months, and in mice exposed for up to 3 months.

Through competitive processes, HEI funded several investigator teams in Phase 3B: a core study at LRRI, led by McDonald (who became principal investigator after the retirement of Dr. Joe L. Mauderly), and ancillary studies to evaluate endpoints not assessed in the core study. The overall hypothesis for ACES Phase 3B was that NTDE would *not* increase tumor formation or have substantial toxic health effects in rats and mice, although some biologic effects might occur.

This Statement summarizes results reported from the core study and the ancillary studies led by Bemis and Hallberg, which assessed genotoxic endpoints in the exposed animals, and by Conklin, which assessed inflammatory and thrombotic endpoints. Reports from the investigator teams were reviewed by a specially convened ACES Review Panel, comprising members of HEI's Health Review Committee and outside experts. The current report focuses on findings in rats over the entire study; findings from subchronic exposures of mice and rats (up to 3 months of exposure) have already been published in HEI Research Report 166.

APPROACH

McDonald and colleagues generated exhaust from a 2007-compliant heavy heavy-duty diesel engine (defined as an engine installed in a vehicle with gross vehicle weight rating above 33,000 lb; hereafter called "heavy-duty") equipped with emission controls. The engine was fueled with ultra-low-sulfur diesel fuel meeting current on-road specifications and was operated with a dynamometer.

The investigators exposed male and female 6-week-old Wistar Han rats (140 animals of each sex per exposure level) to one of three target dilutions of whole diesel exhaust — 4.2 (high), 0.8 (mid), or 0.1 (low) ppm NO_2 — or to filtered air as a control. Exposure levels were set based on NO_2 rather than PM, which had been used in previous studies of TDE, because the PM level in NTDE, identified in earlier phases of ACES, was so substantially reduced compared with TDE. Thus, calibrating exposures based on PM would have been problematic. In addition, the highest NO_2 exposure level was chosen to provide a comparison with the same cumulative exposure to NO_2 (the product of concentration and exposure duration) used in prior HEI-funded long-term inhalation studies in rats conducted by Mauderly and colleagues, in which minor biologic changes — but no cancer or pre-cancerous changes — were observed in the respiratory tract.

Exposures were conducted for 16 hours per day from approximately 1600 to 0800 hours for 5 days per week. The engine was run on a unique and strenuous operating cycle that represented more closely the behavior of modern engines than operating cycles used in older long-term studies of TDE. The emissions were characterized before they reached the animal exposure chambers as well as inside the chambers; in this way, the investigators could assess how the presence of the animals affected the composition of the exposure atmospheres.

Groups of male and female rats were euthanized at LRRI after 1, 3, 12, and 24 months of exposure, as well as at the terminal sacrifice — 28 months for males, 30 months for females. The LRRI investigators harvested blood and tissues for their analyses at these time points (10 animals of each sex per exposure group) and also sent aliquots of blood and appropriate tissue samples from 5 to 10 animals of each sex per exposure group to the ACES Phase 3B ancillary studies investigators. McDonald and colleagues evaluated animals histologically throughout the study for the presence of tumors and other types of lesions in the airways and in multiple tissues. In addition, they examined a vast array of biologic endpoints: hematologic (several cell types, plus coagulation), serum chemistry (including triglyceride and protein components), lung lavage (including numbers of cells and levels of multiple cytokines and markers of oxidative stress and tissue injury), and pulmonary function.

For the assessments of genotoxicity, Bemis and colleagues measured the number of reticulocytes — immature red blood cells — containing micronuclei in peripheral blood. Micronuclei can form as a result of a break in deoxyribonucleic acid (DNA) or from the disruption of chromosome segregation during cell division. Hallberg and colleagues assessed several markers of oxidative damage to cell components, which is believed to be involved in the induction of carcinogenesis. To detect damage to DNA, the Hallberg team used a comet assay on lung cells and measured 8-hydroxydeoxyguanosine levels in blood. As a measure of damage to lipids, they assessed levels of thiobarbituric acid

reactive substances in brain tissue. Conklin and Kong measured multiple plasma markers of inflammation and thrombosis, and whether chronic exposure had an effect on cardiac fibrosis or the remodeling of the aorta.

RESULTS AND CONCLUSIONS

In its independent review of the core ACES Phase 3B report by McDonald and colleagues, the HEI ACES Review Panel concluded that their study is the first to conduct a careful, comprehensive, and well-executed evaluation in rodents of lifetime inhalation exposure to NTDE from a 2007-compliant engine. Using appropriate statistical approaches to analyze the data from more than 100 endpoints in the broad areas of histology, serum chemistry, systemic and lung inflammation, and respiratory function, the investigators confirmed the a priori hypothesis, namely, that NTDE would *not* cause an increase in tumor formation or substantial toxic health effects in rats, although some biologic effects might occur.

Over the entire exposure period, the investigators attained NTDE exposure atmospheres within 20% of the target NO_2 levels. In their extensive analysis of the physical and chemical composition of the emissions, McDonald and colleagues found that the most abundant pollutants were carbon dioxide, carbon monoxide, NO, and NO_2. Concentrations of engine-generated PM were very low (< 11 $\mu g/m^3$) at all exposure levels (in the ultrafine range of 20–40 nm in diameter), as were concentrations of sulfur dioxide and semivolatile and volatile organic species. These findings confirm that the concentrations of components of NTDE differ strikingly from those of older engines, in which the concentrations of PM, as well as volatile and PM-associated organic species, are much higher.

Most biologic endpoints evaluated showed no NTDE-associated changes after exposure of rats for up to 28 months in males and 30 months in females. In particular, chronic exposure to NTDE did not induce tumors or pre-cancerous changes in the lung and did not increase tumors that were considered to be related to NTDE in any other tissue. Some mild histologic changes were found in the lung; however, these were not pre-cancerous lesions, previously described in long-term exposure studies of rats to TDE. Rather, the histologic changes — periacinar epithelial hyperplasia, bronchiolization, accumulation of macrophages, and periacinar interstitial fibrosis — were confined to a small region, the centriacinus, which is involved in gas exchange.

HEI convened a separate panel of expert pathologists, the Pathology Working Group (PWG), to evaluate the histopathology data collected. The PWG findings confirmed the major histopathologic observations reported by the investigators. Also, the PWG, by evaluating the findings of this study side by side with findings from prior long-term exposure studies, provided a context with which to compare and contrast the current study findings with those of other relevant long-term studies of exposure to TDE and oxidant gases. The overall conclusion was that chronic exposure of rats to NTDE did not produce tumors in the lung, in marked contrast to the effects of chronic exposure to TDE observed in multiple previous rat studies, in which lung tumors, as well as inflammation and the deposition of soot in the lung, were observed. Rather, the effects of NTDE in the lung more closely resembled changes noted after long-term exposures to gaseous oxidant pollutants, in particular NO_2, and to TDE from which particles have been filtered out. It is possible that components of NTDE other than NO_2 may have contributed to the effects reported, but the low levels of other components suggest that they would not be primarily responsible.

The ACES Review Panel concluded that the multiple toxicity endpoints evaluated — including lung and serum chemistry and respiratory function — were appropriate for evaluating a wide range of possible biologic effects. There were small decreases in some respiratory endpoints, in particular those concerned with expiratory flow, predominantly at the highest exposure level and more in females than males. The diffusing capacity of carbon monoxide (DL_{CO}, a measure of alveolar–capillary gas exchange) showed a small effect of exposure to NTDE. The Panel considered the small reductions in DL_{CO} to be consistent with the histopathologic findings of mild changes in the gas-exchange regions of the lung, indicating that the histologic changes might have had functional effects. In addition, some small changes in a few markers of oxidative stress and inflammation were detected in lung tissue, bronchoalveolar lavage fluid, and blood. The Panel identified a minor limitation to the study: some biochemical assays lacked positive controls (to determine that each was sensitive enough to detect any changes).

The Panel considered that the ancillary studies by Bemis et al., Hallberg et al., and Conklin and Kong were valuable extensions to the ACES core investigation. These generally well implemented studies took advantage of samples collected by McDonald

and colleagues at several exposure time points up to 24 months to assess multiple endpoints that are not normally part of chronic inhalation bioassays. The genotoxicity studies assessed well-accepted endpoints — the frequency of micronucleated reticulocytes (immature red blood cells) in blood in the report by Bemis et al., and DNA damage and lipid peroxidation in the report by Hallberg et al. Conklin and Kong assessed a wide range of plasma markers associated with systemic inflammation and thrombosis, as well as markers of more chronic effects, to identify possible cardiovascular effects of NTDE. The Panel agreed with the conclusions of Bemis and colleagues and of Hallberg and colleagues that no genotoxic effects could be detected that were associated with exposure for up to 24 months to NTDE. However, the Panel noted that the assays measured relatively short-term effects (lasting 1 month or less), which somewhat reduced confidence in the utility of these negative findings. In Conklin and Kong's study, NTDE had no effect on cardiac fibrosis or aortic remodeling and few effects, predominantly in females and of uncertain pathophysiologic significance, on the inflammatory and thrombotic pathway endpoints measured in plasma over 24 months of exposure.

Overall, these results indicate that rats exposed to one of three levels of NTDE from a 2007-compliant engine for up to 30 months, for 16 hours per day, 5 days a week, with use of a strenuous operating cycle that more accurately reflected the real-world operation of a modern engine than cycles used in previous studies, showed few exposure-related biologic effects. In contrast to the findings in rats chronically exposed to TDE, there was no induction of tumors or pre-cancerous changes in the lung and no increase in tumors that were considered to be related to NTDE in any other tissue. The effects that were observed with NTDE were limited to the respiratory tract and were mild and generally seen only at the highest exposure level. The histologic changes in the lungs were consistent with previous findings in rats after long-term exposure to NO_2 — a major component of the exposure atmosphere, which is being substantially further reduced in 2010-compliant engines.

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