Before the Senate Energy and Environment Committee
March 8, 2017

Testimony of Bob Russell
Vice President Government Affairs
Oregon Trucking Associations

There has been a great deal of discussion about the impact of diesel engine exhaust on human health. The Environmental Protection Agency began regulating the exhaust from heavy truck engines, in earnest, beginning in 2004 when engine manufacturers were required to reduce NOX emissions. Then, in 2007 EPA required a 98% reduction in diesel engine particulate matter emissions. In 2010 an additional reduction in NOX was required reducing emissions of this element by a total of 95%. (See Attachment 1, Perry Hystad Presentation.) A 2015 study by the respected Health Effects Institute concludes that there is no longer a link between 2007 model diesel engine exhaust and cancer. (See Attachment 2)

So, how many heavy trucks, over 26,000 pound, are operating in Oregon that have the 2007 model year engines or newer? ODOT has provided the following statistics as of March 1, 2017:

	Companies	Trucks	2007>	%
In State	7,872	46,599	24,617	53%
Out of State	18,321	292,489	248,750	85%
Totals	26,193	339,088	273,367	80%

Based on the number of older trucks left in the fleet, what is an appropriate response? While many states provide incentives to help fleets move into the newer engines, California is the only state that has developed a regulatory response. California's response is understandable given the quality of their air. The attached map shows that large portions of California are designated as non-attainment areas for particulate matter emissions most closely associated with diesel engines. (See attachment 3) On the other hand, Oregon has no non-attainment areas for particulate matter emissions. (See attachment 4) As of last month, California continues to have significant parts of the state in non-attainment even though they have employed both significant incentives and adopted stringent regulations to reduce emissions from diesel engines. (See attachment 5)

Oregon adopted its original; incentives based Clean Diesel Program in 2007. Since that time, Oregon has not used any state funds for this program. The small amount of funds that have been made available, \$4.3 million, have all come from the federal government. In contrast, California has spent hundreds of millions of dollars on incentives to replace older diesel engines. Washington has also implemented a strong incentives based program having provided over \$80 million to industry. For Oregon, the most likely source of incentives going forward is the Volkswagen Settlement Fund. While the trucking industry, has made tremendous progress towards eliminating the engines with higher rates of emissions from the fleet, it will be difficult to significantly expedite the rate of conversion without sizable incentives from the State. Given the current budget situation, we recognize that this is unlikely.

So, how should Oregon proceed to reduce emissions from heavy duty truck engines? We believe that <u>any program</u> should be based on the following principles:

- **The program should be targeted geographically to address the areas of the state with high levels of emissions.**

- **The program should be based on monitor data rather than computer based models.** Oregon lacks a system of monitors to actually determine the quality of our air. (See attachment 6) The Legislature should provide the necessary funding so that DEQ can determine when a contaminant curtailment program is necessary but the data is also essential to determine if these programs are effective. The current reliance on computer based models is only as good as the assumptions that go into them. For example, the model used by DEQ to conduct the Portland Air Toxics Study completely missed the significant emissions coming from two art glass manufacturers operating in the City.

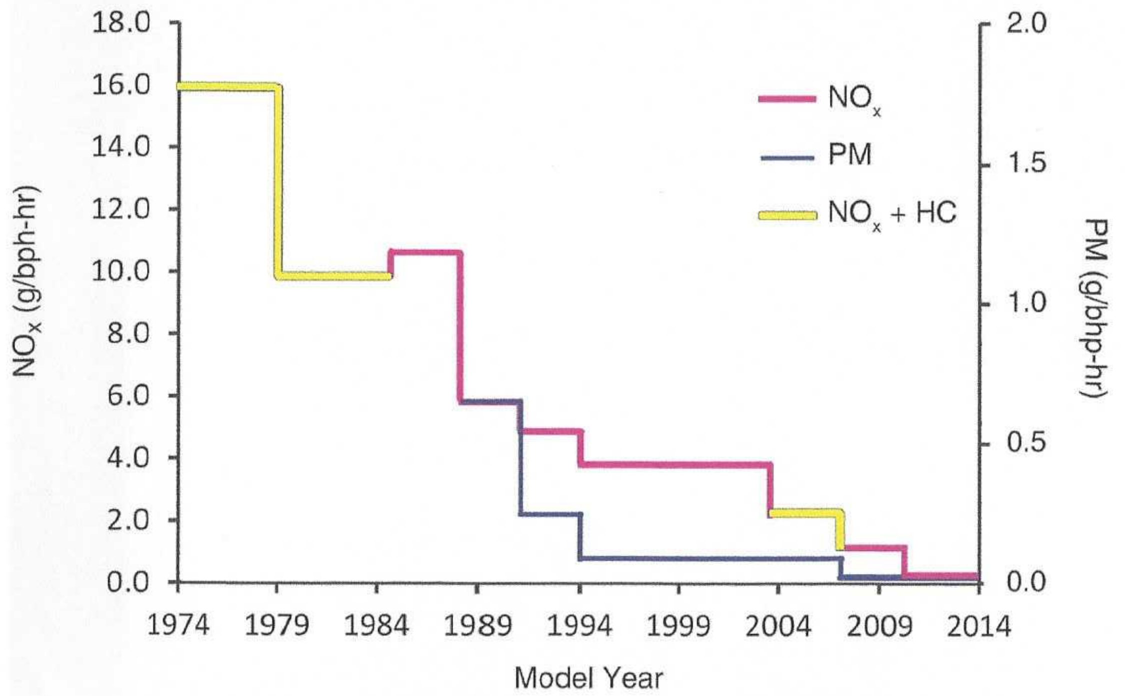
- **Program resources should be expended in such a way that they provide the largest cost to benefit ratio in order to meet the defined objectives of the program.** This includes expenditures of the Volkswagen Settlement Fund.

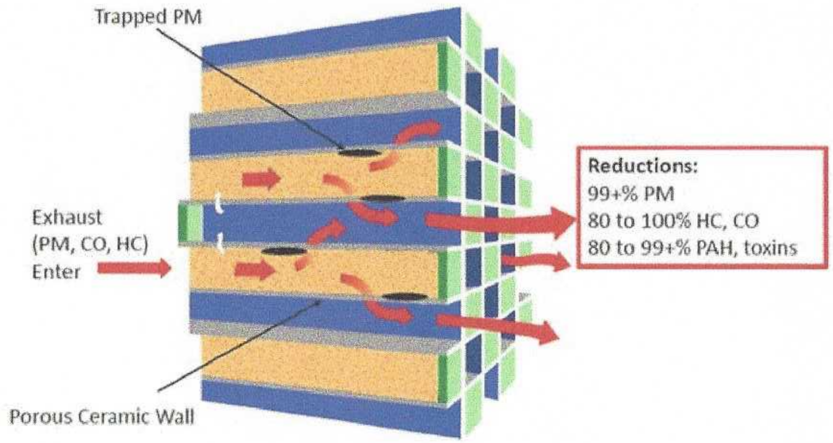
- **Maintain the statewide application of Oregon's existing truck idling regulations.**

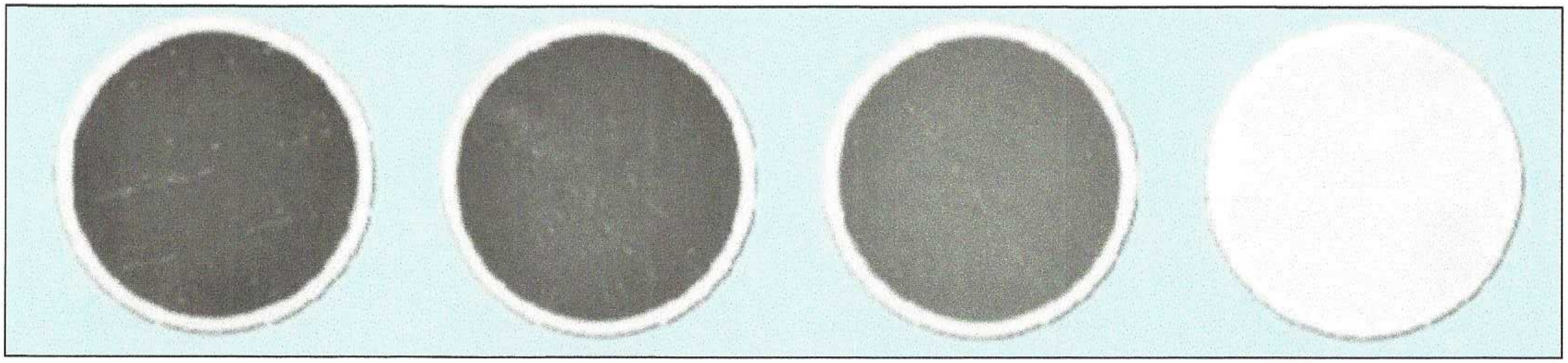
The members of the Oregon Trucking Associations recognize that reducing emissions from heavy trucks is an ongoing discussion. We look forward to our continued participation in these discussions and sincerely hope that emissions can continue to be reduced in a manner that does not unreasonably burden the industry.

Thank you for the opportunity to testify today.

Reductions in Diesel Emissions Over Time







Uncontrolled Diesel Exhaust | Diesel Oxidation Catalyst | Partial Filter | Diesel Particulate Filter (since 2007)

References: Meca (2017), EPA (2013), HEI (2015)

Attachment 1

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Attachment 2

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).

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

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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

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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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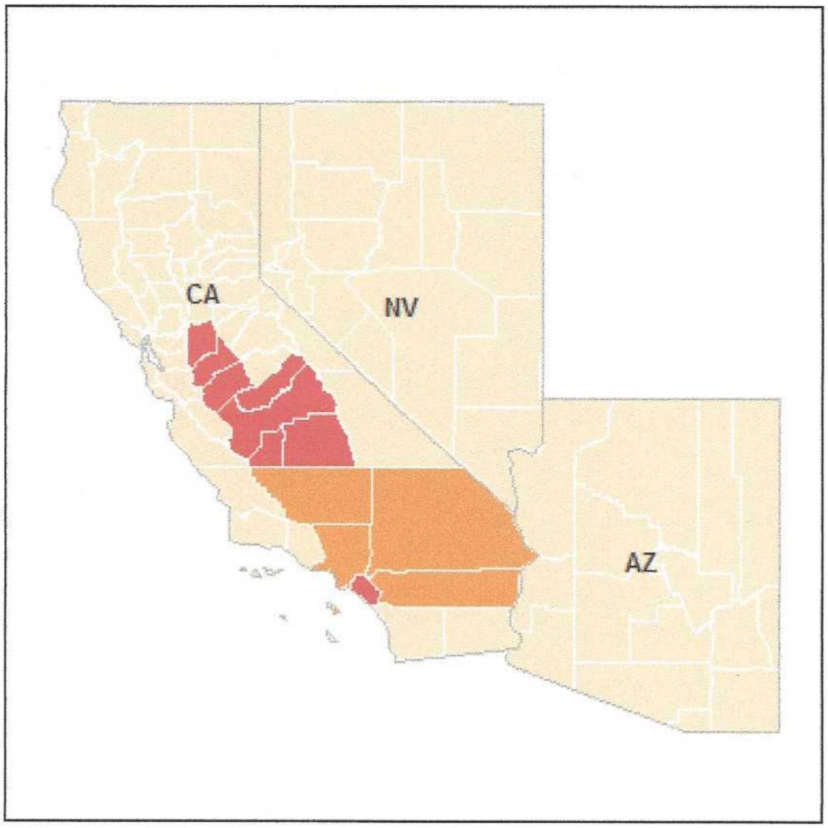
Attachment 3

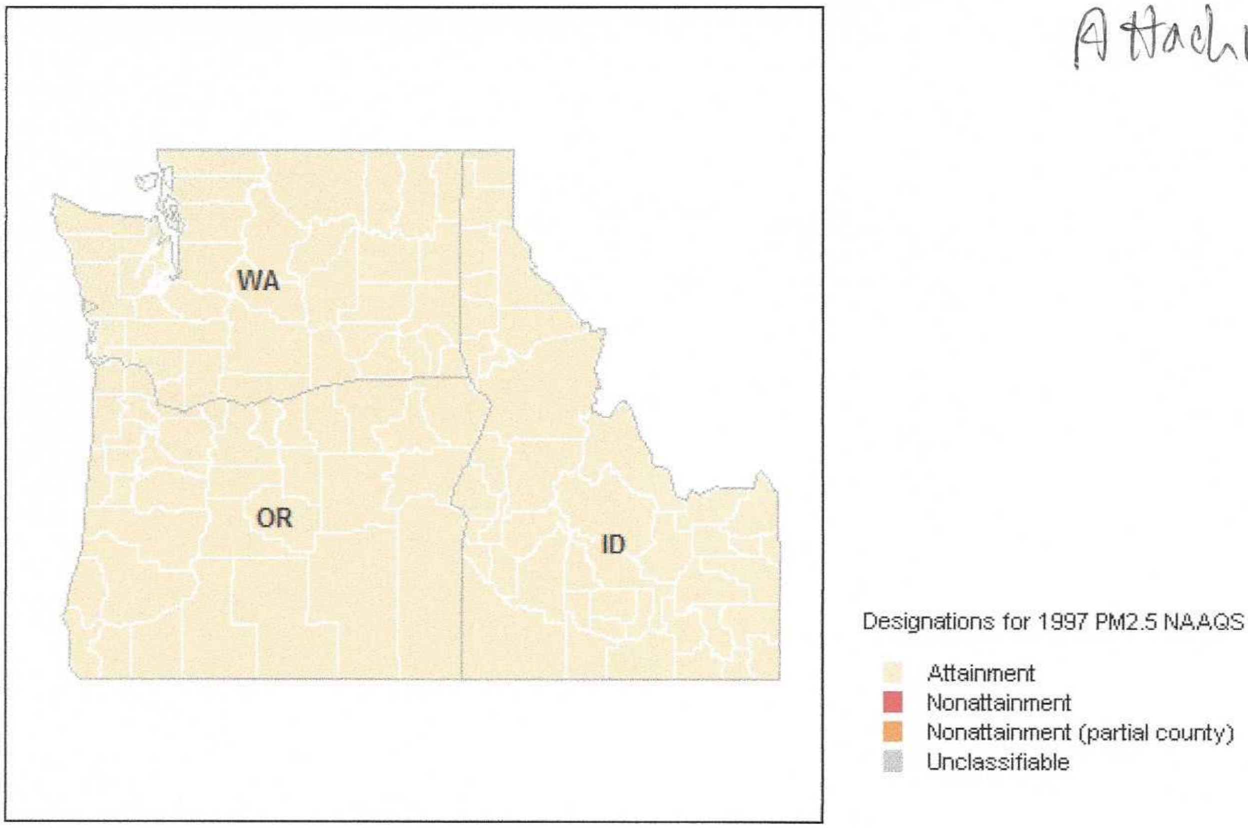


This table identifies all counties EPA has designated as nonattainment. In some cases EPA designated partial counties. These are identified by a (P). If a county is not listed below, EPA has designated it as unclassifiable/attainment.

State	Area Name	Counties
Arizona	Entire state is attainment/unclassifiable	
California*	Los Angeles-South Coast Air Basin, CA	Los Angeles (P) Orange Riverside (P) San Bernardino(P)
	San Joaquin Valley, CA	Fresno Kern (P) Kings Madera Merced San Joaquin Stanislaus Tulare
Hawaii	Entire state is attainment/unclassifiable	
Nevada	Entire state is attainment/unclassifiable	

*See correspondence related to the April, 2005 supplemental notice for information about changes to designations since the December, 2004 action.

Attachment 4



This table identifies all counties EPA has designated as nonattainment. In some cases EPA designated partial counties. These are identified by a (P). If a county is not listed below, EPA has designated it as unclassifiable/attainment.

State	Area Name	Counties
Alaska	Entire state is attainment/unclassifiable	
Idaho	Entire state is attainment/unclassifiable	
Oregon	Entire state is attainment/unclassifiable	
Washington	Entire state is attainment/unclassifiable	

Region 10 Recommendations and EPA Responses

Area Navigation

- Fine Particle ($PM_{2.5}$) Designations Home

- Area Designations for 1997 Fine Particle ($PM_{2.5}$) Standards Home
- Regulatory Actions
- Where You Live
  - State Designations
  - Tribal Designations
  - $PM_{2.5}$ Conditions
  - Air Quality Forecast

Attchmnt 5

EPA United States Environmental Protection Agency

You are here: EPA Home > PM-2.5 (2012) Designated Area/State Information

PM-2.5 (2012) Designated Area/State Information

As of February 13, 2017

PM-2.5 (2012) Notes

Display: Nonattainment and Maintenance Areas / Nonattainment Areas / Maintenance Areas

Area Name *Click underlined column heading to change report order.* Click area name for state/county list	State	Current Status	Current Classification or at Redesignation click * for classification history	No. Counties	2010 Population	EPA Region
Allegheny County, PA	PA	Nonattainment	Moderate	1	1,223,348	03
Cleveland, OH	OH	Nonattainment	Moderate	2	1,581,478	05
Delaware County, PA	PA	Nonattainment	Moderate	1	558,979	03
Imperial County, CA	CA	Nonattainment	Moderate	1	154,061	09
Lebanon County, PA	PA	Nonattainment	Moderate	1	133,568	03
Los Angeles-South Coast Air Basin, CA	CA	Nonattainment	Moderate	4	15,716,242	09
Plumas County, CA	CA	Nonattainment	Moderate	1	5,843	09
San Joaquin Valley, CA	CA	Nonattainment	Moderate	8	3,842,165	09
West Silver Valley, ID	ID	Nonattainment	Moderate	1	7,497	10
			Total Areas	**Total Counties**	**Total Population (2010)**	
Nonattainment			9	20	23,223,181	
Maintenance			0	0	0	
Nonattainment and Maintenance			9	20	23,223,181	

PM-2.5 (2012) Notes

County subtotals and grand totals may not equal sum of the counties. Part counties are only counted one time within groupings. Multi-state areas are counted only once in area totals, and are counted in maintenance totals when all states in the area have been redesignated.

Discover. Connect. Ask.

Attachment 6

DEQ Air Toxics Monitoring Summary 2017

Full Spectrum Stations to Monitor Trends and Assess Communities

Current monitoring:

DEQ currently has the capacity to conduct air toxics monitoring at six locations.

Two sites are National Air Toxics Trends monitoring stations, one in North Portland and one in La Grande that are funded by U.S. EPA. These are full spectrum stations where DEQ monitors for over one hundred pollutants including: metals, hexavalent chromium, volatile organic compounds, carbonyls, polycyclic aromatic hydrocarbons and black carbon to evaluate particulate. These sites are considered trend sites and do not move year to year.

Four sites are rotating annual full spectrum monitoring stations supported by state General Fund. One site is currently located in Southeast Portland near Precision Castparts Corporation and another is located in Gresham for the same suite of toxics. The two monitors funded in February 2016 are being built and will be sited based on a prioritization approach DEQ is developing to determine where to place available monitors statewide in the future. These four sites are designed to move to the next priority site after collecting data for a year (which allows monitoring to account for seasonal weather patterns and comparing the data to Oregon's Ambient Air Quality Benchmark Concentrations).

2017-2019 DEQ Budget Request:

DEQ is requesting equipment and staff for six new trend sites. This funding would allow DEQ to expand the number of trend sites from the two federally funded sites in Portland and La Grande to a new statewide total of eight sites. The areas of interest would be three additional Portland locations, Eugene, Medford, and Bend. At each location DEQ would be able to monitor for the full spectrum of air toxic pollutants of concern. This would allow for long term trends of air toxics to be identified and would include analysis for metals, hexavalent chromium, volatile organic compounds, carbonyls, polycyclic aromatic hydrocarbons, and black carbon. *2017-2019 budget request: 8 positions, 6.0 FTE, $720K equipment; total request $2.2M*

DEQ is also requesting equipment and staff for 30 new particulate monitors that would be placed throughout the state. The presence of high levels of particulate is often indicative of the presence of air toxics. This would be a cost effective method of identifying possible air toxics hot spots and at the same time provide Oregonians with more real time information about air quality by posting particulate results on the Air Quality Index. *2017-2019 budget request: 2 positions, 1.75 FTE, $200K equipment; total request $600K*

Response Monitoring

Current monitoring:

As a follow-up to the results of U.S. Forest Service moss analytical data, DEQ is assessing hot spots in four Portland Metro areas.

- DEQ has completed metals and hexavalent chromium monitoring at four locations surrounding Uroboros. The monitoring data was released and posted to the Cleaner Air Oregon website.
- DEQ has completed monitoring at two locations for metals and hexavalent chromium near Bullseye Glass. Two other monitors for metals and two monitors for hexavalent chromium remain at locations near Bullseye Glass. DEQ will continue this monitoring for one full year from when monitoring began, through the end of March 2017.
- DEQ has removed two monitors for metals and two monitors for hexavalent chromium surrounding Precision Castparts Corporation. DEQ will continue monitoring metals and hexavalent chromium at one location to compile one full year of data.
- DEQ completed monitoring at two locations that had monitors for metals and monitors for hexavalent chromium in the Cully neighborhood.

Currently, some of the equipment being used in the moss follow-up monitoring is on loan from EPA. During the April Emergency Board session, DEQ received funding for 12 metals monitors and 12 hexavalent chromium monitors. DEQ is working through the necessary steps to procure the equipment and will return the borrowed equipment to EPA upon their arrival. The USFS moss study identified additional areas of interest that will have some follow-up monitoring in the form of additional moss collections to deployment of air monitors that will continue to take place into 2017.

2017-2019 DEQ Budget Request:

DEQ did request equipment and personnel for one continuous metals monitoring site that can be used to identify sources of metals emissions. A continuous metals monitor can take a sample anywhere from every hour to every four hours and can detect changes in concentrations of metals throughout the day, which can lead to better identification of potential sources. The continuous metals monitor would allow DEQ to have a better understanding of metals concentrations in real time and with a better understanding of concentrations throughout the day instead of a 24 hour composite. *2017-2019 budget request: 1 position, 1.00 FTE, $200K equipment; total request $450K. This was not included in the GRB*

DEQ did request additional staff to expand the use of moss studies as a screening tool to identify areas of concern for metals pollutants. *2017-2019 budget request: 1 position, .50 FTE; total request $107K. This was not included in the GRB*

Odor/Complaint investigations

Recently, DEQ deployed monitoring resources in order to investigate odor complaints and determine whether odors are associated with air pollutants that might impact human health. DEQ is currently involved in monitoring at two locations, Hayden Island and The Dalles which include equipment for specific classes of compounds and meteorological stations.

- DEQ completed monitoring at Hayden Island for volatile organic compounds and hydrogen sulfide.
- DEQ completed monitoring in The Dalles at three locations for polycyclic aromatic hydrocarbons.

DEQ does not have staff or equipment dedicated to this type of monitoring. The resources and monitors being used are borrowed from the full spectrum stations, which has led to a delay in starting the new annual sites.

Meteorology Stations

DEQ has meteorology stations associated with every air toxics network for monitoring wind speed and direction in order to help evaluate the sources of air toxics.