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When You Can't Breathe, Nothing Else Matters

For nearly 100 years, the American Lung Association, Lung Association affiliates throughout the United States and the American Thoracic Society have worked together in the fight against lung disease.

AMERICAN LUNG ASSOCIATION®

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CLEAN AIR ACT ADVISORY COMMITTEE

MOBILE SOURCE TECHNICAL REVIEW SUBCOMMITTEE

NONROAD WORK GROUP

JANUARY 16, 2001

Good Morning. I am Deborah Shprentz, and I am speaking today on behalf of the American Lung Association Association (ALA). The American Lung Association is the nation's oldest voluntary health organization. ALA's mission is to prevent lung disease and promote lung health.

Our message to you today is very simple: there are compelling health reasons for the EPA to proceed with an additional round of emission restrictions for nonroad diesel engines. Relative to cars and trucks, nonroad diesel engines present a significant opportunity for additional emissions control.

Emissions from nonroad diesel engines are harmful to human health in many different ways, and are important contributors to several air quality problems. Of particular concern are:

1) The cancer effects of diesel particulate emissions;
2) The noncancer effects of diesel particulate emissions;
3) The contribution of diesel to fine particulate concentrations; and
4) Emissions of precursor pollutants such as nitrogen oxides that contribute to the formation of ground level ozone smog as well as fine particle pollution.

These problems are exacerbated by the enhanced personal exposure profile of many nonroad diesel engine categories, for instance farm and construction equipment, that may concentrate emissions in the breathing zone of farm workers, construction workers and passersby.

Cancer Effects

EPA's Clean Air Scientific Advisory Committee (CASAC) met in October 2000 and CASAC members voted unanimously to reach closure on a revised version of the EPA Health Assessment Document for Diesel Exhaust, culminating a review process that spanned almost two decades. Significantly, the Committee affirmed EPA's designation of diesel exhaust as a "likely human carcinogen."

In doing so, the EPA joins numerous other scientific and health organizations in concluding that diesel poses a significant cancer hazard to humans. These agencies include the International Agency for Research on Cancer (IARC), the U.S. National Toxicology Program (NTP), and the State of California, all of which have concluded that diesel emissions constitute a "known" or "probable" human carcinogen.

The epidemiological database is extensive. Dr. John R. Froines of UCLA School of Public Health, and Acting Chairman of the Scientific Review Panel for the State of California has noted that there are more human epidemiological studies for diesel exhaust – over 30 – than for any of the prior 21 toxic air contaminant reports reviewed by the

Scientific Review Panel.[1] According to the report of the California Scientific Review Panel, these studies on average found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer.[2]

In addition to the epidemiology studies, there are experimental laboratory studies of cancer effects. Taken together, the record demonstrates strong, consistent findings of carcinogenicity.

According to the National Toxicology Program, "Diesel exhaust particles are considered likely to account for the human lung cancer findings because they are almost all of a size allowing penetration to the entire lung, and because mutagenic and carcinogenic chemicals including polycyclic aromatic hydrocarbons and nitroarenes have been extracted from these particles with organic solvents, or with a lipid component of mammalian lung surfactant."[3]

While CASAC declined to endorse a quantitative estimate of cancer risk, The California Office of Environmental Health Hazard Assessment used epidemiological studies to estimate a lifetime unit risk of 1.3×10^{-4} to 2.4×10^{-3} per $\mu g/m^3$.[4] The Scientific Review Panel derived a "reasonable estimate" unit risk number of 3×10^{-4} per microgram per cubic meter of diesel particulate.

The unit risk number estimate generated by the California Scientific Review Panel indicates that diesel exhaust is a more potent carcinogen than other substances regulated as toxic air contaminants in California or listed as Hazardous Air Pollutants under the Clean Air Act, including formaldehyde, methylene chloride, and trichloroethylene.[5]

The South Coast Air Quality Management District's recent MATES-II study estimated air pollution cancer risks in the South Coast Basin using local monitoring data and the California unit risk number.[6] Basinwide, cancer risks from air pollution were estimated at 1,400 in one million, with diesel particulate responsible for 71 percent of the cancers.

A similar analysis was performed nationally by STAPPA and ALAPCO, the associations of state and local air administrators. Using the California unit risk number, the

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[1] Froines, John R., Ph.D. Acting Chairman, Scientific Review Panel, May 27, 1998 letter to John D. Dunlap III, Chairman, Air Resources Board.

[2] Findings of the Scientific Review Panel on The Report on Diesel Exhaust as adopted at the Panel's April 22, 1998 Meeting. In, California Environmental Protection Agency Air Resources Board, "Initial Statement of Reasons for Rulemaking," Staff Report on Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant, June 1998.

[3] National Toxicology Program, The Report on Carcinogens – 9th Edition, May 2000.

[4] California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Appendix III Part B Health Risk Assessment for Diesel Exhaust, As Approved by the Scientific Review Panel on April 22, 1998.

[5] SCAQMD, MATES-II Final Report, Appendix 1-A. List of Toxic Compounds and Their Associated Unit Risk Factors (URF), March 2000.

[6] South Coast Air Quality Management District, Multiple Air Toxics Exposure Study (MATES)– II Final Report, March 2000.

STAPPA/ALAPCO report estimated the cancer risk from diesel emissions from onroad and offroad engines at 125,000 cancer cases nationwide.[7]

Non-Cancer Effects

Human and animal evidence indicates that diesel exhaust can induce irritation to the eyes, the nose, and the throat, as well as trigger inflammatory responses in the airways and the lung following short-term exposures to high concentrations.[8] At last October's meeting, the CASAC panel restored the Reference Concentration for noncancer effects back to 5 $\mu g/m^3$, from 15 $\mu g/m^3$ in the draft under review.

Of particular concern, given the rising toll of asthma on our nation's youth, are recent studies which suggest that pre-exposure to diesel exhaust may enhance the response to aero-allergens.[9]

Fine Particle Pollution

Epidemiological studies from around the world have reported an association between particulate air pollution and reductions in lung function, respiratory symptoms, school absenteeism, increased use of asthma medications, doctor visits, emergency room visits, hospital admissions, and premature death at levels common in U.S. cities.

Diesel exhaust is a significant contributor to fine particle concentrations, particularly in urban areas. In 1997, EPA established health standards for fine particulate matter, $PM_{2.5}$, for the first time. Since then, there have been significant new research findings that lend support to the need for fine particle standards.

Long-term epidemiological studies indicating that fine particles increase the risk of early death have recently been validated by an independent reanalysis.[10] New studies show that chronic exposure to particulate pollutions shortens lives by one to three years.[11]

A study of the 90 largest U.S. cities found strong evidence linking daily increases in particle pollutions at contemporary levels to increases in daily death rates, and in hospital

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[7] State and Territorial Air Pollution Program Administrators and Association of Local Air Pollution Control Officials, Cancer Risk from Diesel Particulate: National and Metropolitan Area Estimates for the United States, March 15, 2000.

[8] U.S. Environmental Protection Agency, National Center for Environmental Assessment, Health Assessment Document for Diesel Exhaust, SAB Review Draft, EPA/600/8-90/057E, July 2000.

[9] Cohen, Aaron J. and Nikula, Kristen. The Health Effects of Diesel Exhaust: Laboratory and Epidemiologic Studies, in Air Pollution and Health, Academic Press, 1999.

[10] Krewski, D., Burnett, R.R., Goldberg, M.S., Hoover, K., Siemiatycki, J., Jerrett, M., Abrahamowicz, M., White, W.H, and Others. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute, July, 2000.

[11] Pope, C.A. III, Epidemiology of Fine Particulate Air Pollution and Human Health: Biological Mechanisms and Who's at Risk? Environ Health Persect 108 (suppl 4):713-723 (2000).

admissions of the elderly.[12] These results are consistent with a study of 12 European cities.

Particulate pollution has been tied to low heart rate variability, a risk factor for heart attacks. A chamber study with human subjects found that concentrated air particles can induce pulmonary inflammation and increase concentrations of fibrinogin in the blood, a risk factor for clotting and heart attacks.[13]

Particulate pollution worsens bronchitis in asthmatic children. Kids experience declines in lung flow and increased symptoms such as cough, phlegm production, and sore throat after particle exposure, but children with asthma are more susceptible to these effects than other children.

Children's emergency room visits for asthma increase on high particle pollution days.

People most sensitive to fine particle pollution are infants and children, especially those with asthma, the elderly, and people with pre-existing heart and lung conditions.

Studies of particulate matter and mortality have not identified a threshold or "safe" level of exposure.

There have been a number of recent studies pointing to significant health endpoints specifically from traffic related pollution. For example, a recent study by Laden et al differentiated the sources of particles in the Six City Study and specifically identified $PM_{2.5}$ from mobile sources as the most significant contributor to mortality relative to stationary combustion sources, and crustal sources.[14] A recent risk assessment performed by Abt Associates for the Clean Air Task Force reported quantitative estimates of on and off road sources of diesel emissions on human health.[15] The analysis reported that 15,400 premature deaths each year are attributable to the diesel contribution to fine particle concentrations. In addition, there are an estimated 11,100 cases of chronic bronchitis due to diesel emissions, thousands of hospitalizations due to chronic obstructive pulmonary disease, pneumonia, asthma, and cardiovascular causes, and over a million cases of minor illness such as acute bronchitis, upper and lower respiratory symptoms, and asthma attacks.

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[12] Samet, J.M., Zeger, S.L., Dominici, F., Curriero, F., Coursac, I., Dockery, D.W., Schwartz, J., and Zanobetti, A. The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity, Mortality and Air Pollution in the United States. Health Effects Institute Research Report 94, Part II, June 2000.

[13] Ghio, A.J., Kim, C., and Devlin, R.B. Concentrated Ambient Air Particles Induce Mild Pulmonary Inflammation in Healthy Human Volunteers. Am. J. Respir. Crit. Care Med. 2000 162: 981-988.

[14] Laden, Francine, Neas, Lucas M., Dockery, Douglas W., and Schwartz, Joel. Association of Fine Particulate Matter from Different Sources with Daily Morality in Six U.S. Cities. Environ Health Perspect 108:941-947 (2000).

[15] Abt Associates Inc. with ICF Consulting and E.H. Pechan Associates, Inc., The Particulate-Related Health Benefits of Reducing Power Plant Emissions, Prepared for Clean Air Task Force, October 2000.

Ozone Smog

Ozone is the principle component of ground-level smog. It is formed when hydrocarbon and nitrogen oxide pollution combine in the atmosphere in the presence of sunlight. Ozone is a powerful oxidizing agent that damages lung tissue. Diesel engines are a major source of nitrogen oxide emissions and the importance of diesel's contribution, from both onroad and offroad sources, to ozone smog is well-recognized. These nitrogen oxide emissions also contribute to the fine particle pollution problem in many areas.

Recent research with laboratory animals, clinical subjects, and human populations has identified a cascade of adverse health effects from ozone at levels common in the United States. Effects include increased respiratory symptoms, damage to cells of the respiratory tract, declines in lung function, increased susceptibility to respiratory infections, and increased risk of hospitalization and early death.

EPA revised the National Ambient Air Quality Standards for ozone in 1997 because the health evidence showed that adverse health effects were experienced at lower concentrations than the earlier standards established in 1978. Well documented effects of ozone include:

• Short-term exposures to ozone can cause a decline in lung function, including rapid breathing, decreased lung volumes and flow, and increased twitchiness of the airways. Exposure early in life may lead to acceleration in the decline of lung function that is a normal process of aging.

• Respiratory symptoms can include coughing, throat irritation, shortness of breath, and pain on taking a deep breath. Asthmatics can experience wheezing, a hallmark of an asthma attack.

• When children with a tendency toward allergies and asthma are exposed to ozone, they may be more responsive to allergens that can trigger an asthma attack.

• Ozone triggers an inflammation response in the cells lining the lungs, causing them to rupture and leak. Repeated exposures may lead to structural changes in the respiratory tract including increased production of fibrous tissue associated with lung scarring.

• Ozone compromises the lungs' natural defense mechanisms, increasing susceptibility to respiratory infections such as colds, flu, and pneumonia.

• Short-term exposures are also associated with an increase in daily mortality, and increased hospital admissions and emergency department visits for respiratory causes. A recent risk assessment estimated that ozone sends 53,000 people to the hospital, 159,000 to the emergency room, and triggers 6,200,000 asthma attacks in the Eastern half of the United States each summer.[16]

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[16] Clean Air Task Force, Out of Breath, Health Effects from Ozone in the Eastern United States. Oct. 1999.

Four groups of people are especially sensitive to ozone: children, people with respiratory disease, health adults who work or exercise outdoors, and people with special sensitivity who are otherwise healthy.

Conclusion

As EPA moves to clean up diesel fuel and tighten emissions standards for heavy-duty vehicles, it is necessary to consider more stringent fuel quality and emissions limits for diesel nonroad engines. Emissions from nonroad diesel engines are a significant contributor to serious adverse health effects from air pollution in urban and rural areas, and remain a relatively uncontrolled source of the particulate, ozone and toxic air pollution that cause these health concerns.