

respiratory tract. At high concentrations, irritation and pulmonary effects can occur, which could facilitate the uptake of other contaminants. Some asthmatics have been shown to be a sensitive subpopulation to decrements in FEV1 upon acetaldehyde inhalation.⁵⁰ The agency is currently conducting a reassessment of risk from inhalation exposure to acetaldehyde.

Acrolein: Nonroad diesel engines accounted for about 17.5 percent of acrolein emissions in 1996. Of ambient acrolein levels due to mobile sources, 28 percent in urban and 18 percent in rural areas came from nonroad diesel.

Acrolein is extremely toxic to humans when inhaled, with acute exposure resulting in upper respiratory tract irritation and congestion. The Agency has developed a reference concentration for inhalation (RfC) of acrolein of 0.02 micrograms/m³.⁵¹

Although no information is available on its carcinogenic effects in humans, based on laboratory animal data, EPA considers acrolein a possible human carcinogen.

Polycyclic Organic Matter (POM): POM is generally defined as a large class of chemicals consisting of organic compounds having multiple benzene rings and a boiling point greater than 100 degrees C. Polycyclic aromatic hydrocarbons (PAHs) are a chemical class that is a subset of POM. POM are naturally occurring substances that are byproducts of the incomplete combustion of fossil fuels and plant and animal biomass (e.g., forest fires). They occur as byproducts from steel and coke productions and waste incineration. They also are a component of diesel particulate emissions. Many of the compounds included in the class of compounds known as POM are classified by EPA as probable human carcinogens based on animal data. In particular, EPA frequently obtains data on 7 of the POM compounds, which we analyzed separately as a class in the 1996 NATA. Nonroad diesel engines account for less than 1 percent of these 7 POM compounds with total mobile sources responsible for only 4 percent of the total; most of the 7 POMs come from area sources. For total POM compounds, mobile sources as a whole are responsible for only 1 percent. The mobile source emission numbers used to derive these inventories are based on

only particulate phase POM and do not include the semi-volatile phase POM levels. Were those additional POMs included (which is now being done), these inventory numbers would be substantially higher.

Even though mobile sources are responsible for only a small portion of total POM emissions, the particulate reductions from today's action will reduce these emissions.

Dioxins: Recent studies have confirmed that dioxins are formed by and emitted from diesels (both heavy-duty diesel trucks and non-road diesels although in very small amounts) and are estimated to account for about 1 percent of total dioxin emissions in 1995. Recently EPA issued a draft assessment designating one dioxin compound, 2,3,7,8-tetrachlorodibenzo-p-dioxin as a human carcinogen and the complex mixtures of dioxin-like compounds as likely to be carcinogenic to humans using the draft 1996 carcinogen risk assessment guidelines. EPA is working on its final assessment for dioxin.⁵² An interagency review group is evaluating EPA's designation of dioxin as a likely human carcinogen. Reductions from today's nonroad proposal will have minimal impact on overall dioxin emissions.

3. Ozone

a. What Are the Health Effects of Ozone Pollution?

Ground-level ozone pollution (sometimes called "smog") is formed by the reaction of volatile organic compounds (VOC) and nitrogen oxides (NO_x) in the atmosphere in the presence of heat and sunlight. These two pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources, including on-road and off-road motor vehicles and engines, power plants and industrial facilities, and smaller "area" sources.

Ozone can irritate the respiratory system, causing coughing, throat irritation, and/or uncomfortable sensation in the chest.^{53 54} Ozone can reduce lung function and make it more difficult to breathe deeply, and

breathing may become more rapid and shallow than normal, thereby limiting a person's normal activity. Ozone also can aggravate asthma, leading to more asthma attacks that require a doctor's attention and/or the use of additional medication. In addition, ozone can inflame and damage the lining of the lungs, which may lead to permanent changes in lung tissue, irreversible reductions in lung function, and a lower quality of life if the inflammation occurs repeatedly over a long time period (months, years, a lifetime). People who are of particular concern with respect to ozone exposures include children and adults who are active outdoors. Those people particularly susceptible to ozone effects are people with respiratory disease, such as asthma, and people with unusual sensitivity to ozone, and children. Beyond its human health effects, ozone has been shown to injure plants, which has the effect of reducing crop yields and reducing productivity in forest ecosystems.^{55 56}

The 8-hour ozone standard, established by EPA in 1997, is based on well-documented science demonstrating that more people are experiencing adverse health effects at lower levels of exertion, over longer periods, and at lower ozone concentrations than addressed by the one-hour ozone standard. (See, e.g., 62 FR 38861-62, July 18, 1997). The 8-hour standard addresses ozone exposures of concern for the general population and populations most at risk, including children active outdoors, outdoor workers, and individuals with pre-existing respiratory disease, such as asthma.

There has been new research that suggests additional serious health effects beyond those that had been known when the 8-hour ozone health standard was set. Since 1997, over 1,700 new health and welfare studies relating to ozone have been published in peer-reviewed journals.⁵⁷ Many of these studies have investigated the impact of ozone exposure on such health effects as changes in lung structure and biochemistry, inflammation of the

⁵⁰ Myou, S.; Fujimura, M.; Nishi K.; Ohka, T.; and Matsuda, T. (1993) Aerosolized acetaldehyde induces histamine-mediated bronchoconstriction in asthmatics. *Am Rev Respir Dis* 148(4 Pt 1): 940-3.

⁵¹ U.S. EPA (1993). Environmental Protection Agency, Integrated Risk Information System (IRIS), National Center for Environmental Assessment, Cincinnati, OH.

⁵² U.S. EPA (June 2000) Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds, External Review Draft, EPA/600/P-00/001Ag. This material is available electronically at <http://www.epa.gov/ncea/dioxin.htm>.

⁵³ U.S. EPA (1996). Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA/600/P-93/004aF. Docket No. A-99-06. Document Nos. II-A-15 to 17.

⁵⁴ U.S. EPA. (1996). Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-96-007. Docket No. A-99-06. Document No. II-A-22.

⁵⁵ U.S. EPA (1996). Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA/600/P-93/004aF. Docket No. A-99-06. Document Nos. II-A-15 to 17.

⁵⁶ U.S. EPA. (1996). Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-96-007. Docket No. A-99-06. Document No. II-A-22.

⁵⁷ New Ozone Health and Environmental Effects References, Published Since Completion of the Previous Ozone AQCD, National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, NC 27711 (7/2002) Docket No. A-2001-11. Document No. IV-A-19.