

agricultural, construction, and utility nonroad equipment operations. This work will measure infiltration of elemental carbon and other PM components to indoor environments. The project also evaluates lung function changes in the asthmatic children during fluctuations in exposure concentrations and compositions. This information may allow an evaluation of adverse health effects associated with exposures to elemental carbon and other PM components from on-road and nonroad sources. Some initial results may be available in late 2003.

b. Gaseous Air Toxics

Nonroad diesel engine emissions contain several substances known or suspected as human or animal carcinogens, or that have noncancer health effects. These other compounds include benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, dioxin, and polycyclic organic matter (POM). For some of these pollutants, nonroad diesel engine emissions are believed to account for a significant proportion of total nation-wide emissions. All of these compounds were identified as national or regional "risk" drivers in the 1996 NATA. That is, these compounds pose a significant portion of the total inhalation cancer risk to a significant portion of the population. Mobile sources contribute significantly to total emissions of these air toxics. As discussed later in this section, this proposed rulemaking will result in significant reductions of these emissions.

Benzene: Nonroad diesel engines accounted for about 3 percent of ambient benzene emissions in 1996. Of ambient benzene levels due to mobile sources, 5 percent in urban and 3 percent in rural areas came from nonroad diesel.

The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia at high, prolonged air exposures) by all routes of exposure, and exposure is associated with additional health effects including genetic changes in humans and animals and increased proliferation of bone marrow cells in mice.^{39 40 41 42} EPA states

in its IRIS database that the data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. Respiration is the major source of human exposure and at least half of this exposure is attributable to gasoline vapors and automotive emissions. A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.^{43 44}

1,3-Butadiene: Nonroad diesel engines accounted for about 1.5 percent of ambient butadiene emissions in 1996. Of ambient butadiene levels due to mobile sources, 4 percent in urban and 2 percent in rural areas came from nonroad diesel.

EPA earlier identified 1,3-butadiene as a probable human carcinogen in its IRIS database and recently redesignated it as a known human carcinogen (but with a lower carcinogenic potency than previously used).⁴⁵ The specific mechanisms of 1,3-butadiene-induced carcinogenesis are unknown, however, it is virtually certain that the carcinogenic effects are mediated by genotoxic metabolites of 1,3-butadiene. Animal data suggest that females may be more sensitive than males for cancer effects; nevertheless, there are insufficient data from which to draw any conclusions on potentially sensitive subpopulations. 1,3-Butadiene also causes a variety of reproductive and developmental effects in mice; no human data on these effects are available. The most sensitive effect was ovarian atrophy observed in a lifetime bioassay of female mice.⁴⁶

Formaldehyde: Nonroad diesel engines accounted for about 22 percent of ambient formaldehyde emissions in

1996. Of ambient formaldehyde levels due to mobile sources, 37 percent in urban and 27 percent in rural areas came from nonroad diesel. These figures are for tailpipe emissions of formaldehyde. Formaldehyde in the ambient air comes not only from tailpipe (of direct) emissions but is also formed from photochemical reactions of hydrocarbons.

EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.⁴⁷ Epidemiological studies in occupationally exposed workers suggest that long-term inhalation of formaldehyde may be associated with tumors of the nasopharyngeal cavity (generally the area at the back of the mouth near the nose), nasal cavity, and sinus.⁴⁸ Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes. Sensitive individuals may experience these adverse effects at lower concentrations than the general population and in persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack. The agency is currently conducting a reassessment of risk from inhalation exposure to formaldehyde.

Acetaldehyde: Nonroad diesel engines accounted for about 34 percent of acetaldehyde emissions in 1996. Of ambient acetaldehyde levels due to mobile sources, 24 percent in urban and 17 percent in rural areas came from nonroad diesel. Also, acetaldehyde can be formed photochemically in the atmosphere. Counting both direct emissions and photochemically formed acetaldehyde, mobile sources were responsible for the major portion of acetaldehyde in the ambient air according to the National-Scale Air Toxics Assessment for 1996.

Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen and is considered moderately toxic by the inhalation, oral, and intravenous routes.⁴⁹ The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and

stimulating activity of granulocyte/macrophage colony-stimulating factor *in vitro*, Proc. Natl. Acad. Sci. 89:3691-3695, 1992.

⁴² U.S. EPA (1998). Carcinogenic Effects of Benzene: An Update, National Center for Environmental Assessment, Washington, DC, 1998.

⁴³ Aksoy, M. (1989). Hematotoxicity and carcinogenicity of benzene. Environ. Health Perspect. 82: 193-197.

⁴⁴ Goldstein, B.D. (1988). Benzene toxicity. Occupational medicine. State of the Art Reviews. 3: 541-554.

⁴⁵ U.S. EPA (2002). Health Assessment of 1,3-Butadiene. Office of Research and Development, National Center for Environmental Assessment, Washington Office, Washington, DC. Report No. EPA/600/P-98/001F.

⁴⁶ Bevan, C; Stadler, JC; Elliot, GS; *et al.* (1996) Subchronic toxicity of 4-vinylcyclohexene in rats and mice by inhalation. Fundam. Appl. Toxicol. 32:1-10.

⁴⁷ U.S. EPA (1987). Assessment of Health Risks to Garment Workers and Certain Home Residents from Exposure to Formaldehyde, Office of Pesticides and Toxic Substances, April 1987.

⁴⁸ Blair, A., P.A. Stewart, R.N. Hoover, *et al.* (1986). Mortality among industrial workers exposed to formaldehyde. J. Natl. Cancer Inst. 76(6): 1071-1084.

⁴⁹ U.S. EPA (1988). Integrated Risk Information System File of Acetaldehyde. This material is available electronically at <http://www.epa.gov/iris/subst/0290.htm>.

³⁹ U.S. EPA (2000). Integrated Risk Information System File for Benzene. This material is available electronically at <http://www.epa.gov/iris/subst/0276.htm>.

⁴⁰ International Agency for Research on Cancer, IARC monographs on the evaluation of carcinogenic risk of chemicals to humans, Volume 29, Some industrial chemicals and dyestuffs, International Agency for Research on Cancer, World Health Organization, Lyon, France, p. 345-389, 1982.

⁴¹ Irons, R.D., W.S. Stillman, D.B. Colagiovanni, and V.A. Henry, Synergistic action of the benzene metabolite hydroquinone on myelopoietic