

major contributor to ambient PM_{2.5} in the eastern U.S. and in some areas make greater contributions than carbonaceous PM_{2.5}.^{10 11}

Nonroad engines, and most importantly nonroad diesel engines, contribute significantly to ambient PM_{2.5} levels, largely through emissions of carbonaceous PM_{2.5}. Carbonaceous PM_{2.5} is a major portion of ambient PM_{2.5}, especially in populous urban areas. Nonroad diesels also emit high levels of NO_x which react in the atmosphere to form secondary PM_{2.5} (namely nitrate). Nonroad diesels also emit SO₂ and NMHC which react in the atmosphere to form secondary PM_{2.5} (namely sulfates and organic carbonaceous PM_{2.5}). For more details, consult the draft RIA for this proposed rule.

Diesel particles from nonroad diesel are a component of both coarse and fine PM, but fall mainly in the fine (and even ultrafine) size range. As discussed later, diesel PM also contains small quantities of numerous mutagenic and carcinogenic compounds associated with the particulate (and also organic gases). In addition, while toxic trace metals emitted by nonroad diesel engines represent a very small portion of the national emissions of metals (less than one percent) and a small portion of diesel PM (generally less than one percent of diesel PM), we note that several trace metals of potential toxicological significance and persistence in the environment are emitted by diesel engines. These trace metals include chromium, manganese, mercury and nickel. In addition, small amounts of dioxins have been measured in highway engine diesel exhaust, some of which may partition into the particulate phase; dioxins through out the environment are a major health concern (although the diesel contribution has not been judged significant at this point). Diesel engines also emit polycyclic organic matter (POM), including polycyclic aromatic hydrocarbons (PAH), which can be present in both gas and particle phases of diesel exhaust. Many PAH compounds are classified by EPA as probable human carcinogens.

For additional, detailed, information on PM beyond that summarized below,

see the draft Regulatory Impact Analysis.

a. Health Effects of PM_{2.5} and PM₁₀

Scientific studies show ambient PM (which is attributable to a number of sources, including nonroad diesel) is associated with a series of adverse health effects. These health effects are discussed in detail in the EPA Criteria Document for PM as well as the draft updates of this document released in the past year.^{12 13} In addition, EPA's final "Health Assessment Document for Diesel Engine Exhaust," (the Diesel HAD) also reviews health effects information related to diesel exhaust as a whole including diesel PM, which is one component of ambient PM.¹⁴

As described in these documents, health effects associated with short-term variation in ambient particulate matter (PM) have been indicated by epidemiologic studies showing associations between exposure and increased hospital admissions for ischemic heart disease, heart failure, respiratory disease, including chronic obstructive pulmonary disease (COPD) and pneumonia. Short-term elevations in ambient PM have also been associated with increased cough, lower respiratory symptoms, and decrements in lung function. Short-term variations in ambient PM have also been associated with increases in total and cardiorespiratory daily mortality. Studies examining populations exposed to different levels of air pollution over a number of years, including the Harvard Six Cities Study and the American Cancer Society Study suggest an association between exposure to ambient PM_{2.5} and premature mortality, including deaths attributed to lung cancer.^{15 16} Two studies further analyzing the Harvard Six Cities Study's air quality data have also established a

specific influence of mobile source-related PM_{2.5} on daily mortality¹⁷ and a concentration-response function for mobile source-associated PM_{2.5} and daily mortality.¹⁸ Another recent study in 14 U.S. cities examining the effect of PM₁₀ on daily hospital admissions for cardiovascular disease found that the effect of PM₁₀ was significantly greater in areas with a larger proportion of PM₁₀ coming from motor vehicles, indicating that PM₁₀ from these sources may have a greater effect on the toxicity of ambient PM₁₀ when compared with other sources.¹⁹ Additional studies have associated changes in heart rate and/or heart rhythm in addition to changes in blood characteristics with exposure to ambient PM.^{20 21} For additional information on health effects, see the draft RIA.

The health effects of PM₁₀ are similar to those of PM_{2.5}, since PM₁₀ includes all of PM_{2.5} plus the coarse fraction from 2.5 to 10 micrometers in size. EPA is also evaluating the health effects of PM between 2.5 and 10 micrometers in the draft revised Criteria Document. As discussed in the Diesel HAD and other studies, most diesel PM is smaller than 2.5 micrometers.²² Both fine and coarse fraction particles can enter and deposit in the respiratory system.

In addition to the information in the draft revised Criteria Document, the relevance of health effects associated with on-road diesel engine-generated PM to nonroad applications is supported by the observation in the Diesel HAD that the particulate characteristics in the zone around nonroad diesel engines is likely to be substantially the same as published air quality measurements made along busy roadways.

Of particular relevance to this rule is a recent cohort study which examined the association between mortality and

¹² U.S. EPA (1996.) Air Quality Criteria for Particulate Matter—Volumes I, II, and III, EPA, Office of Research and Development. Report No. EPA/600/P-95/001a-cF. This material is available electronically at <http://www.epa.gov/ttn/oarpg/ticd.html>.

¹³ U.S. EPA (2002). Air Quality Criteria for Particulate Matter—Volumes I and II (Third External Review Draft) This material is available electronically at <http://cfpub.epa.gov/ncea/cfm/partmatt.cfm>.

¹⁴ U.S. EPA (2002). Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F Office of Research and Development, Washington DC. This document is available electronically at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060>.

¹⁵ Dockery, DW; Pope, CA, III; Xu, X; *et al.* (1993) An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 329:1753-1759.

¹⁶ Pope, CA, III; Thun, MJ; Namboodiri, MM; *et al.* (1995) Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* 151:669-674.

¹⁷ Laden F; Neas LM; Dockery DW; *et al.* (2000) Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect* 108(10):941-947.

¹⁸ Schwartz J; Laden F; Zanobetti A. (2002) The concentration-response relation between PM(2.5) and daily deaths. *Environ Health Perspect* 110(10):1025-1029.

¹⁹ Janssen NA; Schwartz J; Zanobetti A.; *et al.* (2002) Air conditioning and source-specific particles as modifiers of the effect of PM₁₀ on hospital admissions for heart and lung disease. *Environ Health Perspect* 110(1):43-49.

²⁰ Pope CA III, Verrier RL, Lovett EG; *et al.* (1999) Heart rate variability associated with particulate air pollution. *Am Heart J* 138(5 Pt 1):890-899.

²¹ Magari SR, Hauser R, Schwartz J; *et al.* (2001) Association of heart rate variability with occupational and environmental exposure to particulate air pollution. *Circulation* 104(9):986-991.

²² U.S. EPA (1985). Size specific total particulate emission factor for mobile sources. EPA 460/3-85-005. Office of Mobile Sources, Ann Arbor, MI.

¹⁰ Rao, Venkatesh; Frank, N.; Rush, A.; and Dimmick, F. (November 13-15, 2002). Chemical speciation of PM_{2.5} in urban and rural areas (November 13-15, 2002) In the Proceedings of the Air & Waste Management Association Symposium on Air Quality Measurement Methods and Technology, San Francisco Meeting.

¹¹ EPA (2002) Latest Finds on National Air Quality, EPA 454/K-02-001.