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SAB Review Draft

Health Assessment Document for Diesel Emissions

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PREFACE

This draft health risk assessment document was prepared by the National Center for Environmental Assessment (NCEA), which is the health risk assessment program in EPA's Office of Research and Development. The assessment has been prepared for EPA's Office of Mobile Sources which requested advice regarding the potential health hazards associated with diesel engine use. As diesel exhaust emissions also affect air toxics and ambient particulate matter, other EPA air programs also have an interest in this assessment. The previous draft of this assessment was released for public comment in February 1998, and the Agency's Clean Air Scientific Advisory Committee (CASAC) met in public session in May 1998 to review the draft. This November 1999 draft is a revision of that 1998 draft, but also builds on the 1990-1999 history of the development of this diesel health risk assessment.

The scientific literature search for this assessment is generally current through January 1999, though a few more recent publications on key topics also have been included.

This November 1999 draft assessment will be reviewed by CASAC in December 1999, and concurrently, public comments will be accepted for a limited time. Following the receipt of comments from CASAC and the public, NCEA plans to finalize the assessment.

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This document was preceded by three earlier drafts: a Workshop Review Draft (EPA/600/8-90/057A, July 1990), an External Review Draft (EPA/600/8-90/057B, December 1994), and an SAB Review Draft (EPA/600/8-90/057C, February 1998). The Science Advisory Board's Clean Air Scientific Advisory Committee (CASAC) reviewed the 1994 draft in public sessions in May 1995 and the 1998 draft in May 1998. Public comment periods also were conducted concurrently with the CASAC reviews. In addition, many reviewers both within and outside the Agency provided assistance at various review stages.

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1. EXECUTIVE SUMMARY

The Health Assessment Document for Diesel Emissions represents the Agency's first comprehensive review of health effects from exposure to exhaust from diesel engines. In-depth research on diesel exhaust (DE) started in the 1970s, and EPA began regulating emission levels for certain types of diesel engines during the same period. EPA wanted to be aware of the current health issues as it continues with Clean Air Act regulatory programs, hence the need for this assessment. In nine chapters, this health assessment addresses key themes or questions such as (1) the health effects of concern for humans, (2) the best insight as to the mode of action and measure of dose/exposure for the toxic response(s), (3) what dose-response analysis suggests about the possible impact/risk to a human population, and (4) the overall nature of the hazard and the related confidence or uncertainties.

Diesel exhaust is a complex mixture of particles and gases with hundreds of chemical compounds, including many organic compounds, present on the particles and in the gases. The particles have an elemental carbon core, with individual particles being very small (a mean aerodynamic diameter of about 0.2 µm) and thus highly respirable. The small particles have a large surface area upon which many organic compounds are adsorbed. The particle organics generally contribute 10%-30% of particle weight and, for example, contain various types of polyaromatic hydrocarbons (PAHs). The gases have both inorganic and organic constituents (e.g., sulfur dioxide, nitrogen oxides, benzene, ethylene, toluene, aldehydes, olefins, and low-molecular-mass PAHs). Both the particles and the numerous organic compounds of DE have toxicological properties that are capable of influencing a toxic response in humans, though the role of either or both in producing a toxic effect in humans is unknown.

DE particles contribute to ambient particulate matter, e.g., $PM_{2.5}$. Compared to other sources of ambient PM, the elemental carbon core is nearly unique to DE, as are a few of the adsorbed organic compounds. The DE gases are more ubiquitous in an urban environment.

Diesel engines may be on-road (vehicle engines) or off-road (many types of engines powering equipment, machinery, railroad locomotives, and ships). Quantitatively, amounts of specific emission constituents vary by type of engine and even within the same engine type. Qualitatively, the basic composition is fairly consistent, for example, an elemental carbon core particle with PAHs adsorbed to the particle and also present in the gases. Over the years, the mass of particles emitted in engine exhaust has been reduced, as have the accompanying organics.

For years researchers have measured DE concentrations using particle mass per unit volume, i.e., µg/m^3 of diesel particulate matter. This assessment adopts µg/m^3 as a dosimeter and further assumes that the important toxicologic agents in DE will be proportional to µg/m^3. This leads to some uncertainty, but the best dosimeter will not be known until the mode of action for DE toxicity is better understood. Questions have been raised as to whether toxicological findings generated from exposure to older engine exhaust can appropriately be applied to current-day engine exhaust exposures. This question is not resolvable with present information, except to note that available evidence does not point to significant shifts in DE composition relative to the total organics over the years, and that organics are believed to be in relative proportion to the mass of particles.

The primary chronic health concerns include nonmalignant respiratory effects and lung carcinogenicity. The DE particulates can be a component of ambient $PM_{2.5}$. Compared to ambient $PM_{2.5}$ with no DE component, DE is likely to have a higher proportion of fine and ultrafine particulates and is likely to have a higher or at least a varied content of toxicologically active organic compounds. Although some similarities exist between DE and ambient PM, the differences are potentially significant. A comparison of the DE RfC and the $PM_{2.5}$ standard has considerable complexity. For ambient PM we see increased mortality and morbidity in human studies from various forms of chronic respiratory disease. For DE we expect adverse respiratory effects but have not clearly observed them in human studies, possibly because few such studies have focused on respiratory effects. Animal studies conducted at higher than ambient exposure levels, the most prominent being in the rat, provide the basis for the expectation of human respiratory disease. A recommended human chronic exposure level without appreciable hazard (i.e., inhalation Reference Concentration, RfC, 5 µg/m^3) from adverse noncancer respiratory effects is provided in the assessment. From an acute exposure standpoint, DE is an irritant to the respiratory system given sufficient episodic exposure and may cause a variety of inflammation-related symptoms (e.g., headache, eye discomfort, asthma-like reactions, nausea, etc.) depending on individual susceptibility to the DE constituents. Data also suggest that DE is a factor in exacerbating or initiating allergenic hypersensitivity; this is an emerging area of concern.

The carcinogenicity of DE also has been of research and public health interest. Diesel engine exhaust is “highly likely” to be carcinogenic by the inhalation route of exposure, according to EPA’s 1996 Proposed Guidelines for Carcinogen Risk Assessment. This hazard is viewed as being applicable to ambient (i.e., environmental) exposures. Many of the organics present on the DE particles and in the gases, though in small quantities, are mutagenic and/or carcinogenic in their own right. DE shows a pattern of statistically increased lung cancer in more than 20, but not all, human occupational studies where DE exposure is prominent. Lung cancer increases are, on average, about 33-47% above background levels, though specific studies suggest some modestly higher increases. There are some uncertainties about the magnitude of the increase, because questions about exposure are almost always present in the human studies in which the increases are seen, and with lung cancer, the question of confounding by cigarette smoke is present. Nevertheless, analysis of the occupational studies shows that the pattern of increased lung cancer remains after consideration of these issues. Bladder cancer also has been elevated in some epidemiologic studies, though the totality of the evidence is too weak to form a clear conclusion. Although rat inhalation cancer bioassays were once thought to be useful for inferring a human cancer hazard or supporting human evidence, in recent years, the rat lung cancer responses seen with DE exposure are thought to be less clear for human hazard prediction and unsuitable for environmental exposure risk estimation. None of the available studies show that the lung cancer hazard is present at environmental levels of exposure, although the margin may be relatively small between some higher environmental exposures and occupational exposures where lung cancer risks are thought to be present.

The plausibility of an environmental lung cancer hazard from DE by inhalation exposure is supported by findings contained in this assessment. Overall, the evidence for a likely human lung cancer hazard by inhalation is persuasive, even though, in the absence of complete data, inferences and thus uncertainties are involved. Some of the key uncertainties include: (1) methodologic limitations inherent in epidemiologic studies, as well as a lack of reliable historical exposure data for occupationally exposed cohorts, (2) uncertainties regarding the extent of bioavailability of organic compounds present on diesel particles and their impact on the carcinogenic process, and (3) other uncertainties regarding the mode of action of DE on lung cancer in humans.

A decision has been made in this assessment that, despite the finding that DE is best characterized as highly likely to be a lung cancer hazard, the available data are currently unsuitable to make a confident quantitative statement about the magnitude of the lung cancer risk attributable to DE at ambient exposure levels. Therefore, this assessment does not adopt or recommend a specific cancer unit risk estimate for DE. However, information is provided to put DE cancer hazard in perspective and to assist decisionmakers and the public to make prudent public health judgments in the absence of a definitive estimate of the upper bound on cancer risk. Efforts to derive cancer risk estimates for environmental purposes continue, with the focus being on epidemiologic studies because the epidemiology-based estimates are always the ideal starting point, while also recognizing that the rat inhalation studies are no longer favored and other approaches identified to date have limitations.

There is no DE-specific information that provides direct insight to the question of variable susceptibility within the population. Default approaches to account for uncertainty in inter-individual susceptibility have been included in the derivation of the RfC. Individuals with preexisting lung burdens of particulates may have less of a margin of safety from DE particulate-driven hazards than might be inferred from incremental DE exposure analysis, although this cannot be quantified. DE exposure could be additive to many other daily or lifetime exposures to organics and PM. For example, adults who predispose their lungs to increased particle retention (e.g., smoking or high particulate burdens from nondiesel sources), have existing respiratory or lung inflammation or repeated respiratory infections, or have chronic bronchitis, asthma, or fibrosis could be more susceptible to adverse impacts from DE exposure. Although there is no information from studies of DE, infants and children could have a greater susceptibility to the acute/chronic toxicity of DE because they have greater ventilatory frequency, resulting in greater respiratory tract particle deposition. The issue of DE impacts on allergenicity and potential onset and exacerbation of childhood asthma is being actively investigated, but firm conclusions await peer review and publication of ongoing work.

Another aspect of differential susceptibility involves subgroups that may receive additional exposure to DE because of their proximity to DE sources. Those having outside time in their daily routine and being near a diesel emission source would likely receive more exposure than others in the population. The highest exposed are most likely the occupational subgroups whose job brings them very close to diesel emission sources (e.g., trucking industry, machinery operations, engine mechanics, some types of transit operations, railroads, etc.).

Ongoing analyses by EPA, other Federal agencies, and worldwide researchers are expected to improve the existing epidemiology and related exposure databases. These will provide new opportunities to evaluate the potential health effects of DE on the general population and susceptible subgroups.