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PROBLEM DEFINITION STUDY ON EMISSION BY-PRODUCT HAZARDS FROM DIESEL ENGINES FOR CONFINED SPACE ARMY WORKPLACES

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ORNL-118 (6-97)

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April 1984

SUPPORTED BY

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland 21701

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MARTIN MARIETTA ENERGY SYSTEMS, INC.
FOR THE UNITED STATES
DEPARTMENT OF ENERGY

Printed in the United States of America. Available from
National Technical Information Service
U.S. Department of Commerce
5285 Port Royal Road, Springfield, Virginia 22161
NTIS price codes—Printed Copy, A11; Microfiche A01

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PROBLEM DEFINITION STUDY ON
EMISSION BY-PRODUCT HAZARDS FROM DIESEL ENGINES FOR
CONFINED SPACE ARMY WORKPLACES

FINAL REPORT

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U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland, under
Interagency Agreement APO 1814, Task 3

Project Officer: Captain James W. Carroll

Date Published - April 1984

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U.S. DEPARTMENT OF ENERGY
Under Contract No. DE-AC05-84OR21400

EXECUTIVE SUMMARY

The U.S. Army is considering using diesel-powered material-handling equipment in confined space storage areas. Under such conditions, where there may be limited air volume and ventilation, one or more of the compounds released in the engine exhaust may occur at concentrations sufficiently high to be a health hazard. Consequently, there is a need for baseline documentation to identify and characterize diesel exhaust components, to identify the important factors that determine exhaust composition, to relate the potential effects of exhaust composition to air quality of confined space workplaces, and to evaluate the monitoring and modeling methodology that has been used to ensure that air quality standards are maintained under such conditions.

A review of the available data indicates that diesel exhaust consists of thousands of individual chemical compounds, of which only a relatively small fraction have been identified and quantified. These compounds are released in the exhaust as gases, vapors, liquid aerosols, or solid particulate matter. On the basis of U.S. Environmental Protection Agency (EPA) regulatory statutes governing automotive and truck emissions, the most important components of diesel exhaust are carbon monoxide, nitrogen oxides, total hydrocarbons, and total particulate matter. Although diesel exhaust emissions are regulated by EPA, standards for occupational exposures to diesel exhaust have not been promulgated by the Occupational Safety and Health Administration (OSHA); however, there are separate OSHA standards for many of the individual compounds in diesel exhaust including carbon monoxide, nitric oxide, and nitrogen dioxide. The only governmental agency that limits occupational exposures to diesel emissions is the Mine Safety and Health Administration (MSHA). MSHA has established engine emission and air quality standards for underground mines in which diesel engines are operating. Carbon monoxide, carbon dioxide, nitrogen oxides, and total aldehydes are included in these regulations. Neither MSHA or OSHA have specific standards for total hydrocarbons or diesel particulate matter.

An evaluation of the emission rates and concentration levels of the various diesel exhaust components and a review of their potential and known health effects indicate that, in terms of acute toxicity, the compounds that are of greatest concern, particularly in confined space situations, are carbon monoxide, carbon dioxide, nitrogen oxides, formaldehyde, acrolein, and sulfuric acid. Particulates may also be important, not only in catalyzing reactions leading to the formation of sulfuric acid, but also because of their adsorbed polycyclic aromatic hydrocarbons (PAHs). Because of their relatively low concentrations, the PAHs are not considered to be a major problem in terms of immediate toxicity; however, some have been identified as being carcinogenic and/or mutagenic, and it is the long-term effects of chronic exposures to diesel exhaust that are of concern in this regard.

Theoretical calculations and field data from underground mines with diesel equipment indicate that any one or more of the major diesel exhaust components can occur at concentrations exceeding the occupational

exposure limits. A review of the literature reveals that many variables - including engine design and usage, engine operating parameters and maintenance, ambient conditions, fuel composition, and the use of emission control devices, can alter exhaust composition. Changes in, or malfunctions of, any of these parameters could account for excess emissions. Studies conducted by MSHA indicate that the most important factors for minimizing emissions from diesel equipment in underground mines are: (1) properly functioning engines, (2) restrictions on maximum power output, (3) rapid and immediate dilution of exhaust with ventilation air, and (4) minimum mandatory ventilation rates for each specific engine type. In the absence of such standards, the air quality in a confined space in which a diesel engine is being operated must be determined from constant environmental monitoring and relating concentrations of specific exhaust components to short- and long-term occupational exposure limits. Attempts have been made to correlate pollutant concentrations to carbon dioxide, a stable combustion by-product, to arrive at a single key marker for monitoring. Further study is needed to determine if this technique can have wide applicability.

In the absence of monitoring data, the buildup of exhaust pollutants in a confined space can be calculated from engine emission rates, work cycle estimations, air ventilation rates, air volume of the enclosed space, and potential for mixing or stratification of the exhaust in the workplace atmosphere (as correlated with the height and area). In storage areas, the volume of material stored will affect the latter factors as well as residual air volume and possibly ventilation patterns and rates. Consequently, unless it can be shown that such models provide an ample margin of safety, workplace monitoring may still be necessary.

ACKNOWLEDGMENTS

The authors wish to thank Drs. Jack C. Dacre and Gunda Reddy, Research Toxicologists, and Dr. David H. Rosenblatt, Research Chemist, of the U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Maryland, and Drs. Eugenia E. Calle and Curtis C. Travis of the Health and Safety Research Division of Oak Ridge National Laboratory for their technical review of this report. Appreciation is also extended to Judy Crutcher, Frances Littleton, Donna Stokes, and Sherry Hawthorne of the Technical Publications Department of Oak Ridge National Laboratory for typing and editing the manuscript.

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1. INTRODUCTION

1.1 BACKGROUND

Military warehouse and field storage operations require use of forklifts and other vehicle and engine-driven material-handling equipment in confined space workplace environments. Field ammunition storage bunkers or igloos, for example, present a particularly unique confined space equipment need in the form of forklift trucks or vehicles for handling of ammunition and related munition material in remote field locations, often removed from other facilities and utility support. Although battery-powered forklifts and other battery-powered equipment are preferred for confined space use, because they pose little or no contaminant hazards to workers, the limited operating periods of this equipment before electrical recharge may present serious military combat preparedness limitations due to inadequate field electrical recharge capabilities. Petroleum-fueled diesel forklifts and other engine-driven equipment, on the other hand, may pose contaminant emission by-product hazards in these confined space environments due to limited or absent forced air ventilation systems.

Current U.S. Army need, therefore, exists to evaluate the capabilities and limitations of state-of-the-art diesel engine technology for material handling equipment to be used in confined space environments. Prominent focus is on acquisition of forklift equipment for material and munition item handling in field storage igloos or magazines. Many existing igloos are not equipped with forced air ventilation systems; rather, they depend on gravity airflow design. The limited total interior space of these igloos (an igloo may typically be 80 x 25 x 14 feet or contain 25,510 ft³ of total space, with only a 30-minute air supply when fully stocked) may also reduce the operating time for continuous use of diesel or other petroleum-fueled engine equipment because of improper fuel combustion as well as the igloo being a collecting space for diesel exhaust emissions.

Realistic management to meet the Army's needs will likely involve a combined set of constraints on (1) the types of engine-powered equipment used, including consideration of emission control devices; (2) the operating times for permissible use based on air supply and concentration limits for contaminant hazards; and (3) confined space facility design requirements for new and retrofit of existing facilities. Current efforts are underway to test best available commercial engines, such as the so-called "clean burn" diesel engines in fixed engine test cells to qualitatively and quantitatively characterize emission hazards. A second series of evaluations is planned to perform computer modeling of confined space environments to identify operating capabilities for these engines before contaminant exposure standards would be exceeded, and thus identify key operating and design parameters governing safe operation. Finally, actual measurement of contaminant levels is planned through field testing at a "typical" munition storage igloo during operation of selected diesel-powered forklifts to simulate "normal" material handling operations.

These and related studies on diesel-powered material handling equipment use capabilities in confined space workplace environments require a thorough preliminary characterization of existing information on emission by-product hazards from diesel engines. The principal focus of this report is on identifying emission by-products and on determining health hazard potentials, regulatory status, and monitoring and control potentials.

1.2 DIESEL EMISSIONS, AN OVERVIEW

The diesel engine is an internal combustion engine in which air is compressed to a high pressure and temperature causing the fuel, when injected at the proper moment, to spontaneously ignite and initiate combustion. Diesels differ from spark ignition engines in that the air is inducted into the cylinders without restriction and power output is controlled by adjusting the quantity of fuel injected. Because the amount of air entering the cylinder remains constant (except in the case of turbocharging), the ratio of fuel to air in the cylinder increases with increasing power output until a point is reached where the amount of fuel injected consumes all the oxygen available in the intake air charge. Theoretically, the stoichiometric fuel-to-air ratio for complete combustion is 0.067 (1 part fuel to 15 parts air); however, because of incomplete mixing of the air with the fuel, diesel engines generally begin to smoke even before the stoichiometric ratio is reached.

With a diesel engine operating under low power output (low fuel-to-air ratio) it might be expected that complete fuel combustion would occur and that the only combustion by-products would be carbon dioxide and water. However, because of physical and engineering constraints which prevent complete and instantaneous mixing, some of the fuel will not be completely oxidized, but will be released in the exhaust as varying amounts of hydrocarbons, elemental carbon, and carbon monoxide. The hydrocarbons in the exhaust consist not only of fuel degradation products, but also of entirely new organic compounds formed from fuel elements through interactions and chemical recombinations that occur because of the high temperatures and pressures generated during the combustion process. These conditions also enhance several other chemical reactions, including oxidation reactions leading to the formation of nitric oxide, nitrogen dioxide, sulfur dioxide, and sulfuric acid.

The various diesel exhaust components can be categorized not only by their chemical structure but also by the physical state in which they occur when the exhaust is vented to the atmosphere. Diesel exhaust is a complex mixture of gaseous compounds, vapors, liquid aerosols, and solid particulate matter. The gaseous compounds consist of low molecular weight hydrocarbons, such as alkanes and alkenes, and inorganic compounds including carbon dioxide, carbon monoxide, nitric oxide, nitrogen dioxide, and sulfur dioxide. Higher molecular weight hydrocarbons and other organic compounds (aliphatics and aromatics) are present as vapors along with water and inorganic acids. With cooling of the exhaust, some of these materials may condense into liquid aerosols. Finally, the elemental carbon released in the exhaust will occur as solid particulate

matter and will form the nucleus onto which very large molecular weight and polycyclic organic compounds will be adsorbed.

Diesel exhaust components can also be categorized according to the sensory response they evoke. Thus, diesel exhaust can be described as having an odor component, a smoke component, and a component strongly irritating to the eyes and respiratory tract. Diesel odor has been attributed primarily to volatile hydrogens; smoke and soot is caused by excess amounts of carbonaceous particulates; and the irritancy of diesel exhaust has been attributed to nitrogen oxides, sulfuric acid, and certain organic compounds such as aldehydes.

1.3 VARIABLES AFFECTING DIESEL EXHAUST COMPOSITION

As described in the previous section, the composition of diesel exhaust is the result of complete and incomplete combustion reactions, interactions between partially degraded fuel elements, and oxidation reactions involving fuel by-products and various inorganic compounds present in the combustion chamber. Because of the complexity of the reactions involved, many factors will determine the composition of the exhaust during any particular period of engine operation. Several of the key variables will be briefly described here and will be discussed in more detail in Section 4.

Engine design is one factor that can affect emissions. There are two basic types of diesel engines, one in which the fuel is injected directly into the cylinder (direct injection) and one in which the fuel is injected into a small prechamber (indirect injection, prechamber, or precombustion engine) where autoignition occurs before the fuel/air mixture is expelled into the main chamber where further vaporization, mixing, and combustion occur. Because of the enhanced mixing of the fuel/air mixture, more complete combustion of the fuel occurs and emissions are reduced. Direct injection engines can also be modified to improve fuel/air mixing, such as with a swirl chamber in the top of the cylinder. Some engines are also equipped with a turbocharger which uses exhaust gases to compress intake air before it enters the combustion chamber. In effect, a greater amount of air is inducted, which allows more fuel to be combusted, thereby increasing the power output of the engine. Under low power steady-state conditions, turbocharging can reduce exhaust emissions, but during rapid acceleration there may be an increase in emissions due to a delay in turbocharger response and fluctuations in the fuel-to-air ratio. Other engine design parameters that can affect emissions include fuel injector design and placement, nozzle opening size, and nozzle sac volume (the area at the tip of the injector where uncombusted fuel accumulates and then is expelled during the exhaust cycle).

Exhaust emissions vary with changes in power output of the engine as a direct consequence of changes in the fuel-to-air ratio. Near the maximum power output there is a rapid increase in exhaust smoke and carbon monoxide due to the lack of sufficient oxygen for complete combustion. However, for the same reason formation of nitrogen oxides is reduced at high fuel-to-air ratios. Any engine malfunction or

maintenance deficiency which alters the fuel-to-air ratio will adversely affect exhaust emissions. Incorrect timing and rate of fuel injection caused by faulty injectors or fuel pumps, and restricted intake air flow resulting from dirty air filters, are some of the factors which increase exhaust pollutants.

Because diesel fuels can vary in their chemical composition, they can also affect exhaust composition. Fuels vary in the relative amounts of aliphatic and aromatic compounds, and this alters their combustibility characteristics, causes different types and amounts of degradation products to be formed during combustion, and results in different organics and polycyclic compounds appearing in the exhaust. Fuels also vary in their sulfur content, and the sulfur dioxide emitted in the exhaust is proportional to the fuel sulfur level.

Ambient conditions that can alter exhaust emissions to some degree are temperature, humidity, barometric pressure, and ventilation rate. The latter is especially important in confined space situations where some of the exhaust may become mixed with the engine intake air.

Diesel exhaust composition can also be altered through the use of various emission control devices. These include exhaust gas recirculation and reduction catalysts to reduce NO_x emissions, catalytic oxidizers to reduce carbon monoxide emissions, water scrubbers to remove sulfur compounds, and trap oxidizer reactors to remove hydrocarbons, particulates, and smoke. No single control method will reduce the levels of all components at the same time; consequently, a combination of methods is needed for maximum efficiency.

It can be seen, then, that to accurately assess the potential health effects of workers exposed to diesel exhaust, consideration must be given to all the factors governing exhaust composition; these include engine design and usage patterns, engine operating parameters and maintenance, ambient conditions, fuel composition, and the presence or absence of emission control devices.

2. IDENTIFICATION AND CATEGORIZATION OF DIESEL EMISSIONS

2.1 REGULATED COMPONENTS OF DIESEL EXHAUST

The combustion by-products of diesel engines can be categorized as to whether or not they are regulated by federal law. These regulations take the form of emission standards which must be met by the manufacturers through modifications in engine design or emission control devices. In order to maintain ambient air quality standards, as required by the Clean Air Act, the U.S. Environmental Protection Agency (EPA) has established automotive emission standards for carbon monoxide (CO), nitrogen oxides (NO_x), total hydrocarbons (HC), and total particulate matter (PM) (see Table B-1). These standards were determined from considerations of the maximum realistic reduction in emission levels based on best available technology. They are expressed in terms of the amount of exhaust pollutant emitted per distance traveled as determined in tests designed to simulate a standard driving cycle. Separate EPA emission standards have been established for light and heavy duty gasoline-powered and diesel vehicles. The standards listed in Table B-1 are those for light duty diesel trucks.

Although the EPA regulations define specific emission limits, they are only indirectly applicable to occupational exposure situations because concentrations of exhaust pollutants can vary considerably with engine operating conditions. The regulations are also not suited for confined space workplaces where there may be a rapid buildup in one or more of the EPA-regulated exhaust components or in one or more nonregulated components. There are, however, emission standards for diesel engines operating in confined space workplaces. These are the regulations established by the Mine Safety and Health Administration (MSHA 1982) for controlling the use of diesel equipment in underground mines (Table B-1). Carbon monoxide, NO_x, and total aldehydes are the components of diesel exhaust regulated by MSHA. These standards are based on mass concentrations, and they would also be appropriate for other confined space workplaces. The MSHA diesel emission standard for CO in noncoal, nongassy mines is 3000 ppm before dilution and 100 ppm after dilution. The NO_x standard is 25 ppm (measured as NO₂) after dilution, and the total aldehyde standard is 10 ppm after dilution and measured as formaldehyde equivalents. There are no MSHA standards for total hydrocarbons, particulates, or any other component of diesel exhaust.

2.2 DIESEL EXHAUST COMPONENTS WITH WORKPLACE EXPOSURE LIMITS

Occupational exposure limits for air pollutants are promulgated by the Occupational Safety and Health Administration (OSHA 1982) of the U.S. Department of Labor. These exposure limits are generally 8-hr, time-weighted, average concentrations (TWAs) for a normal 40-hr work week. For compounds which may have an acutely toxic effect, the 8-hr TWAs may be supplemented or replaced by ceiling values, which are maximum exposure limits regardless of time period, and in some cases, by maximum peak concentrations above the ceiling value, which are maximum exposure levels limited by time (e.g., 5, 10, 30 min) and frequency

(e.g., not more than once every 2, 3, or 4 hr). Although OSHA has set exposure limits for some complex gas and particle mixtures such as coke oven emissions, it has not promulgated a standard for occupational exposure to diesel exhaust. However, OSHA regulations exist for some of the individual components of diesel exhaust, including CO (50 ppm 8-hr TWA), NO (25 ppm 8-hr TWA), and NO₂ (5 ppm ceiling value) (Table B-2). There are no OSHA exposure limits for diesel particulates or total hydrocarbons; however, a large number of the vapor phase or particulate-associated inorganics and organics are OSHA regulated (see Table B-3). Of the secondary diesel pollutants, total aldehydes and total sulfates are not regulated, but 8-hr TWAs have been established for sulfur dioxide (5 ppm), sulfuric acid (1 mg/m³), formaldehyde (3 ppm), acrolein (0.1 ppm), crotonaldehyde (2 ppm), and other aldehydes.

Another governmental agency that has established occupational exposure limits for some of the components of diesel exhaust is the Mine Safety and Health Administration (MSHA) of the U.S. Department of the Interior. To protect workers in mines in which diesel-powered equipment is used, MSHA has set 100 ppm as the maximum permissible concentration of CO, and 25 ppm as the maximum permissible concentration of NO_x in noncoal mines having no flammable gas contaminants. In addition there is a 0.5% by volume upper limit for CO₂ in mine air and a 20% by volume lower limit for oxygen. No MSHA regulations govern exposures to total hydrocarbons, aldehydes, sulfates, or diesel particulates.

The MSHA exposure limits are equivalent to ceiling values; consequently, the MSHA standard for carbon monoxide is more restrictive, but the NO_x/NO₂ standard is less restrictive than that of OSHA, particularly for short intermittent exposures. For example, considering four 30-min exposures per 8-hr workday, the OSHA 8-hr TWA would permit the CO concentration during each 30-min exposure to average 200 ppm, which is twice the level permitted by MSHA. Under the same exposure conditions, the OSHA standard would permit 30-min exposures to NO to be 100 ppm while the MSHA standard for NO_x is only 25 ppm. However, since the OSHA standard for NO₂ is a ceiling value (5 ppm), it would be more restrictive than the MSHA limit for NO_x.

Although there is no specific MSHA exposure limit for aldehydes, the 10-ppm engine emission standard in diluted exhaust gases places an upper limit on potential worker exposure to this group of compounds. This MSHA standard is for total aldehydes. It does not take into account significant differences in the minimum effects level of individual aldehydes (see Section 3.5), and thus may not offer as great a margin of safety as the OSHA standards for the individual compounds.

MSHA requires that its emission standards and exposure limits be met by adequate ventilation of work sites based on the mass emission rates of individual diesel engines. Thus, for each engine certified by MSHA a minimum ventilation standard must be met. Various site specific and epidemiological studies indicate that the MSHA standards are adequate for protecting miners from immediate adverse health effects associated with diesel exhaust.

In confined spaces with limited or no ventilation, the rate of buildup of pollutants in the workplace air will be dependent on a number of physical and environmental factors such as engine operation, air volume in the enclosed space, and rate of air exchange (see Section 4). Under such circumstances, and in the absence of any means of controlling ventilation rates, the only means for ensuring that the OSHA and MSHA exposure limits are not exceeded is by minimizing the mass output of the pollutants from the engines and restricting operating times to a duration and frequency necessitated by the onsite conditions.

Although in nonmine situations the MSHA regulations are not enforceable, both the OSHA and MSHA standards should be utilized in confined space situations to provide the maximum level of protection for the workers. Whether compliance with these standards would indirectly limit exposures to secondary diesel pollutants, such as aldehydes and sulfates, to below TWA levels will be discussed in Section 4.5.

2.3 DIESEL EXHAUST COMPONENTS WITH NIOSH AND/OR ACGIH WORKPLACE EXPOSURE GUIDELINES

The occupational exposure standards promulgated by OSHA are based on the recommendations of the National Institute of Occupational Safety and Health (NIOSH), and they usually follow fairly closely the guidelines suggested by the American Conference of Governmental Industrial Hygienists (ACGIH 1982, 1983). The NIOSH and ACGIH guidelines are periodically revised and thus may differ from the OSHA standards. Furthermore, the NIOSH recommendations for daily exposure limits are based on 10-hr time-weighted-averages (TWA), and thus differ from the 8-hr TWAs used by OSHA. In some cases NIOSH or ACGIH has recommended exposure limits for particular compounds which have not yet been set into standards by OSHA. Such differences can be seen in the case of two primary components of diesel exhaust, carbon monoxide (CO) and oxides of nitrogen (NO_x, particularly NO and NO₂) (see Table B-2). For CO, the OSHA and ACGIH exposure limits are both 50 ppm (55 mg/m³), but that of NIOSH is 35 ppm (40 mg/m³). While OSHA has not yet established a ceiling value for CO, NIOSH recommends 200 ppm and ACGIH 400 ppm.

For nitric oxide, the OSHA, NIOSH, and ACGIH TWAs are identical at 25 ppm, and only ACGIH has a recommended ceiling value (35 ppm). For nitrogen dioxide there is no OSHA 8-hr TWA but only a 5-ppm ceiling value. NIOSH has a 1-ppm 15-min ceiling value, and ACGIH a 5-ppm STEL and a 3-ppm TWA.

Many of the other compounds found in diesel exhaust have NIOSH and ACGIH recommended exposure limits. In most cases these are identical with or less stringent than the OSHA standards, as in the cases of sulfuric acid (1 mg/m³, see Table B-2) and acrolein (0.1 ppm, see Table B-12). However, for formaldehyde the standards and recommendations are substantially different (Table B-12). The OSHA standards are 3 ppm for an 8-hr TWA, 5 ppm for a ceiling value, and 10 ppm for a 30-min peak above the ceiling value. In contrast, NIOSH recommends a 30-min ceiling value of only 1.0 ppm, and ACGIH is proposing to reduce its ceiling value from 2 to 1 ppm (ACGIH 1983). These differences are especially

important in view of the potentially high concentrations of formaldehyde in diesel exhaust (see Table B-6).

There are a few compounds in diesel exhaust for which there are NIOSH or ACGIH exposure guidelines but no OSHA standards. These are listed in Table B-4 and include several alkanes and alkenes which, if present in high concentrations, could act as asphyxiants; an aliphatic aldehyde (valeraldehyde); and two suspect carcinogens, benzo(a)pyrene and chrysene. The latter two compounds occur in diesel exhaust in association with the particulate fraction.

2.4 DIESEL EXHAUST COMPONENTS WITH NO OCCUPATIONAL EXPOSURE STANDARDS OR GUIDELINES

Hundreds of compounds have been found in diesel exhaust for which there are no OSHA, NIOSH, or ACGIH occupational exposure standards or guidelines (Table B-5). In addition, it has been estimated that there may be thousands of other as yet unidentified compounds that would fall into this same category (Vostal 1980). Of the identified compounds, most are polycyclic aromatic compounds found in association with diesel particulates. Included in this group are compounds such as anthracenes, phenanthrenes, fluorenes, fluoranthenes, pyrenes, chrysenes, coronenes, carbazoles, and naphthalenes, as well as derivatives having benzo-, dibenzo-, methyl-, methoxy-, hydroxy-, nitro-, carbonyl-, or other groups attached. For only a few of these compounds is there information concerning concentrations in diesel exhaust (Table B-6). Although it is generally considered that the concentrations are too low to be a significant health hazard in terms of acute toxicity, the fact that several of these compounds, such as benz(a)anthracene and cyclopenteno(c,d)pyrene, are suspect carcinogens is a matter of special concern. This will be discussed in Section 3.4.

3. CHARACTERIZATION OF POTENTIAL DIESEL EXHAUST EMISSION HAZARDS

3.1 CARBON MONOXIDE

The following sections will briefly discuss: (1) formation and monitoring of carbon monoxide (CO) in diesel exhaust; (2) known adverse health effects resulting from exposure to CO; and (3) confined space workplace concerns. Review documents by the National Institute for Occupational Safety and Health (NIOSH 1972), National Research Council (NRC 1977a), World Health Organization (WHO 1979a), and Nightingale (1980) will be the sources of information for this section unless otherwise stated.

3.1.1 Formation and Monitoring

Carbon monoxide is formed from the incomplete combustion of diesel fuel. According to Hurn (1975), typical diesel combustion of 1 pound of fuel produces approximately 200 ft³ of exhaust gas of which about 20 ft³ is CO₂, roughly 1/3 ft³ is CO, and 1/3 ft³ is NO_x, with the remainder being free nitrogen and water vapor.

The three most commonly used methods for the routine estimation of CO in air are a continuous analysis method based upon nondispersive infrared absorption spectroscopy (NDIR), a semicontinuous analysis method using gas chromatographic techniques, and a semiquantitative method employing detector tubes. The Environmental Protection Agency has designated the NDIR method as the reference method for continuous measurement of CO. Good NDIR analyzers have a detection limit of about 1 mg/m³ (0.87 ppm). Gas chromatography is, however, particularly suitable when low concentrations of CO have to be measured with a high degree of specificity. The detector tube method is simple and can be used for estimating concentrations above 5 mg/m³. Other methods include catalytic oxidation, electrochemical analysis, mercury displacement, and the dual isotope method. Further information on each of these methods is provided in the review documents (NRC 1977a; WHO 1979a).

3.1.2 Health Effects

Carbon monoxide is an odorless, colorless, tasteless gas with a melting point of -207°C and a boiling point of -192°C. Being only slightly less dense than air (0.968), CO neither settles nor rises but diffuses to all spaces with air (Stokinger 1975). The health significance of CO as a contaminant of air is largely due to its combination with hemoglobin (Hb) to form carboxyhemoglobin (HbCO), thus reducing the availability of oxygen for cellular systems of the body and consequently reducing their functional capacity. The affinity of hemoglobin for CO is more than 200 times greater than for oxygen, which means that CO can seriously impair the transport of oxygen even when present at very low partial pressures. The principal factors affecting the change in HbCO concentration after exposure are (1) concentration of CO inspired, (2) endogenous CO production (i.e., from the breakdown of hemoglobin and other heme-containing pigments), (3) amount of exercise, (4) body size,

(5) lung health (including diffusion capacity), and (6) barometric pressure. With respect to symptomology, extrapolation from Figure 3.1 yields the following information: a HbCO level of 20% results in headache, fatigue, and drowsiness; a HbCO level of 40% results in nausea and vomiting; a HbCO level of above 50% results in coma; and a HbCO level of above 65% results in death. It should be noted that there are many variables, such as intensity of work and smoking, that will cause shifts in the curves in Figure 3.1 (Stokinger 1975). The rate of CO uptake by hemoglobin increases threefold to sixfold between rest and heavy work because of the increased rate of breathing, and the blood of tobacco smoke inhalers will contain 2 to 8% HbCO compared to an average of 1% formed from disintegrating red blood cells in nonsmoking, nonexposed adults. Variation in individual susceptibilities is also a factor since individuals with existing or potentially lowered oxygen-carrying capacity as a result of anemia, alcoholism, etc., could experience adverse effects from CO at lower concentrations than normal individuals.

The Occupational Safety and Health Administration has established an occupational exposure limit (TWA) of 50 ppm for CO (OSHA 1982). NIOSH has, however, recommended a TWA of 35 ppm (NIOSH 1972). This recommendation is based on a HbCO level of 5%, which is the amount of HbCO that an employee engaged in sedentary activity would be expected to approach in 8 hr during continuous exposure. The NIOSH criteria document, which gives the scientific basis for this recommendation, states that the employer must recognize that the level of activity will affect the uptake of CO and consequently the HbCO level. This difference is illustrated in Figure 3.2 where it can clearly be seen that for a given concentration, as physical activity increases, the time required to reach 5% HbCO decreases.

The health effects literature concerning CO exposure to humans has principally been concerned with behavioral changes, work performance, cardiovascular system effects, and effects on pregnant women and their unborn offspring. The following paragraphs will focus primarily on these areas and, unless noted, the information presented will detail effects in humans and not in experimental animals.

3.1.2.1 Behavioral Changes

One of the problems associated with evaluating the behavioral effects resulting from exposure to CO is that some workers have reported detrimental effects at HbCO levels as low as 2%, whereas others have been unable to detect significant impairment at levels from above 5% to 20%. Another complicating factor as illustrated in the NIOSH criteria document is that different investigators in attempting to repeat the work of others have failed to produce the same experimental results under supposedly identical exposure conditions.

Of particular interest to this problem definition study are investigations concerning the effects of CO exposure on driving performance and reaction time. In one study reviewed by NRC, HbCO concentrations of 7 and 12% had no effect on driving performance as judged by several indices (e.g., brake pedal applications). Similar results were found in

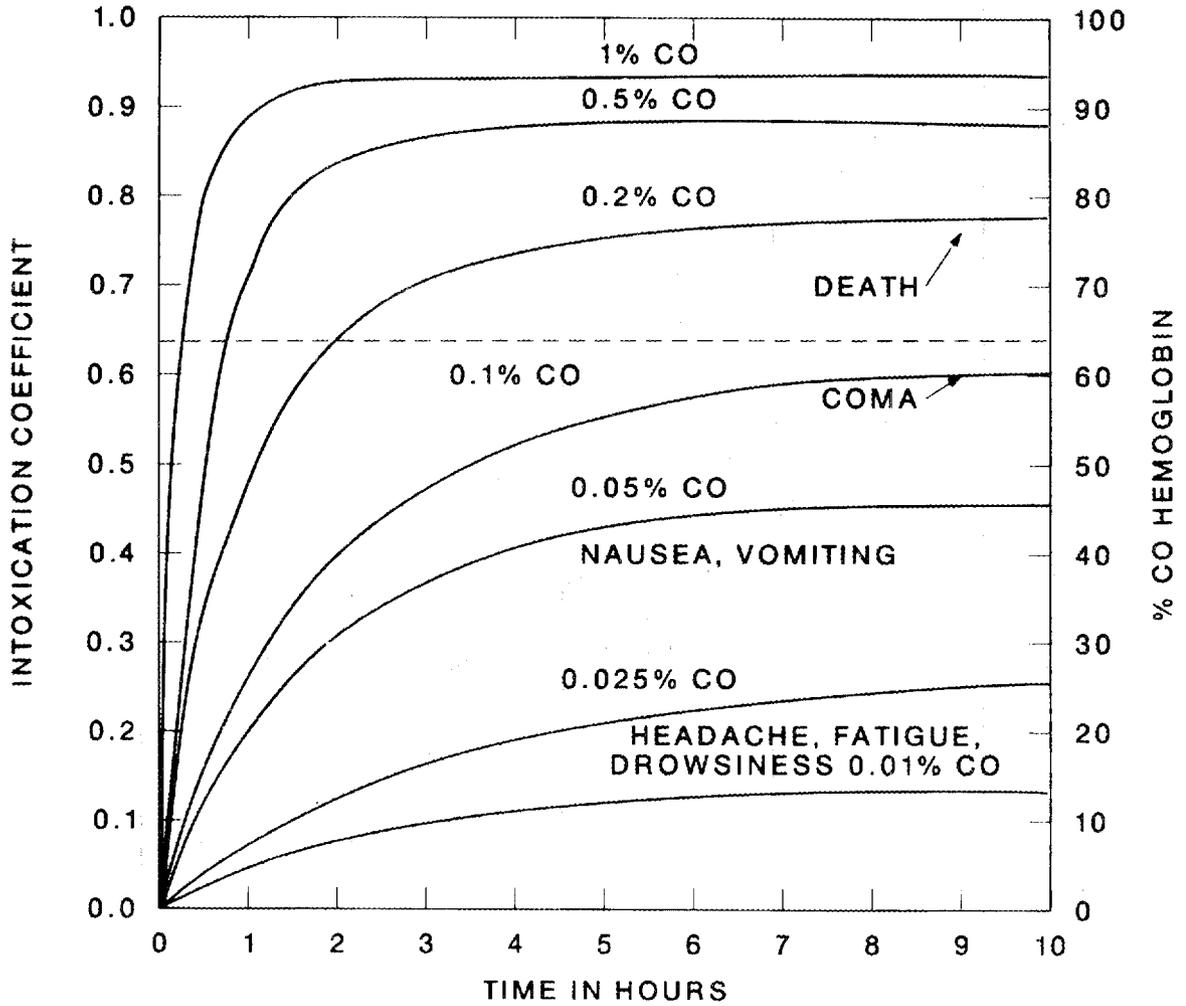


Figure 3.1. Family of curves illustrating the different rates at which hemoglobin becomes saturated with CO at a given atmospheric level of CO and the corresponding coefficient. Adapted from Stokinger 1975.

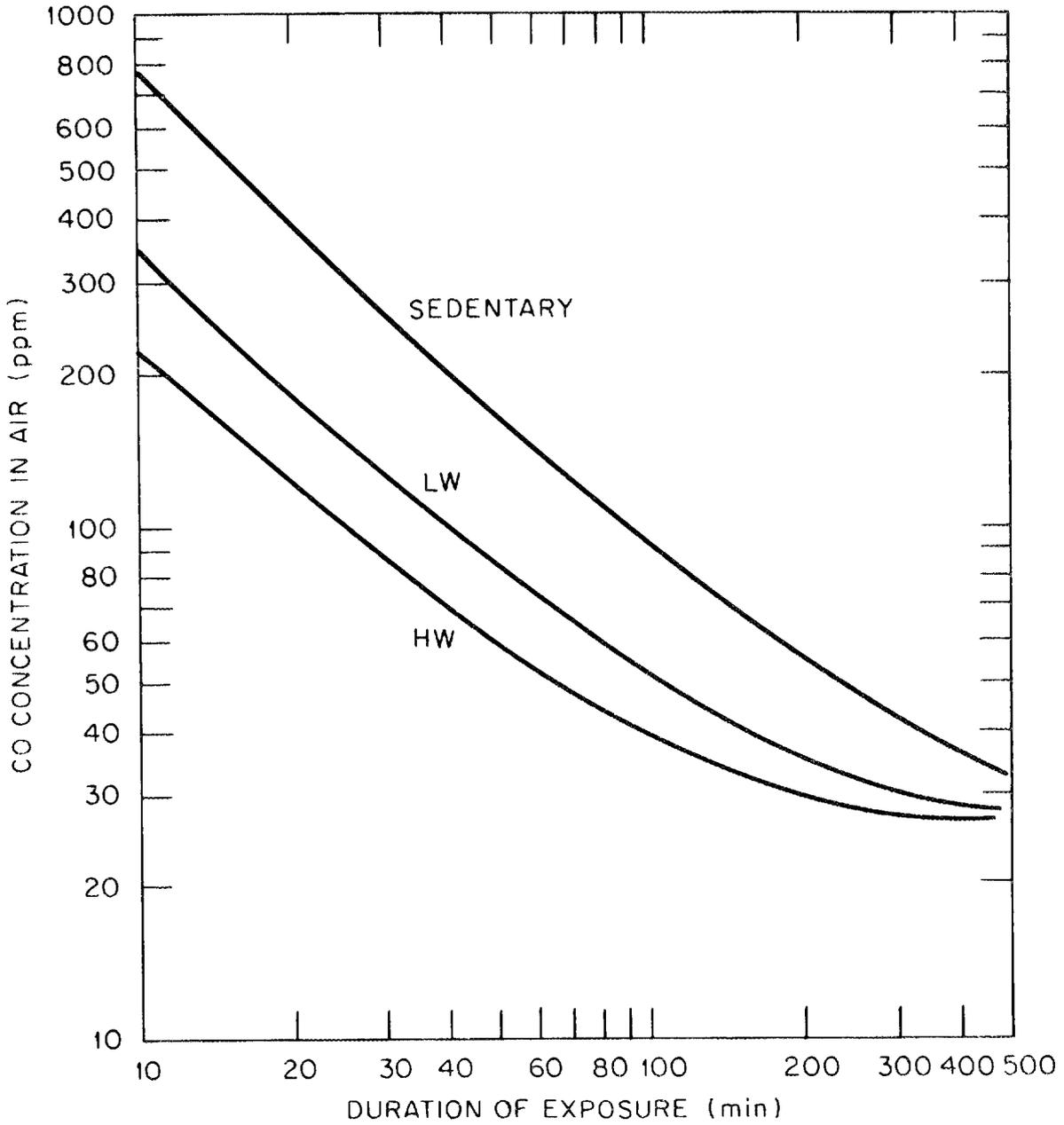


Figure 3.2. Length of time to achieve 5% carboxyhemoglobin (HbCO) at various concentrations of CO in sedentary employees, employees at light work (LW), and employees at heavy work (HW). Adapted from NIOSH 1972.

a study, also reviewed by NRC, in which subjects using a driving simulator experienced no changes in response time to any of three stimuli with HbCO levels as high as 16%. In contrast to these two studies, the NRC report also cited two studies in which reaction times to visual stimuli were decreased by HbCO concentrations ranging from 4.5 to 11.2%. For further information on behavioral changes as a result of CO exposure see Table B-7.

3.1.2.2 Work Performance

The review document by the World Health Organization states that it has long been recognized that elevated HbCO levels can affect work capacity. Studies reviewed in this report indicate that when HbCO levels reach 40-50%, performance of tasks requiring even low levels of physical exertion become impossible. However, at levels as high as 33%, agreement was found among several investigators that the performance of light to moderate work for a short period of time (5-60 min) is not significantly influenced. Further studies reviewed by the WHO concern dose-response for maximum effort and indicate that a reduction in maximum work capacity first appears at a HbCO level of approximately 4%. However, levels of 2.5 to 4%, although not reducing maximum work capacity, did reduce the length of time over which such effort could be maintained. With respect to performance at ordinary work levels (30 to 50% of maximum capacity) for prolonged periods, WHO states that it is not known what specific levels of HbCO will impair this ability. As discussed earlier in this section, OSHA guidelines are based on the HbCO level not exceeding 5%. Further information on the exposure-effect relationship between HbCO levels and work performance is provided in Table B-7.

3.1.2.3 Cardiovascular System Effects

During periods of generalized tissue hypoxia as might result from exercise or from CO exposure, an increase in myocardial activity occurs, resulting in a requirement for increased oxygen supply to the myocardium which must be met by coronary circulation. It is no surprise, therefore, that functional heart disturbances (e.g., lability of blood pressure and heart acceleration, extrasystoles, and exacerbations of angina pectoris), as well as temporary heart dilatation and cardiac asthma have been reported in cases of acute CO poisoning.

Although laboratory investigations using animals, mainly rabbits, have shown that prolonged exposure to moderate levels of CO can produce atherosclerotic changes, especially in animals with high cholesterol levels (1-2%) in the diet, this is probably not a serious concern in the context of this problem definition study since exposures of concern are intermittent and probably of short duration. However, individuals with existing heart illness may be at risk from acute exposures to low concentrations. In two carefully conducted studies, patients with angina pectoris undergoing exercise experienced detrimental effects at low HbCO levels. In one of these studies, 10 subjects were exposed to CO at 50 ppm for 2 hr, resulting in HbCO levels of 2.68% which reduced exercise time because of the onset of angina pectoris. Carboxyhemoglobin

concentrations in individuals exposed to compressed air were only 0.77%. In the other study, 10 patients were exposed to either 50 or 100 ppm CO for 4 hr. After exposure, the duration of exercise before the onset of pain was significantly shorter in both exposure groups (mean HbCO at 50 ppm was 2.8% and at 100 ppm, 4.5%) compared to nonexposed patients with angina pectoris. The time of exercise before onset of pain was, however, the same after exposure to either 50 or 100 ppm.

A similar study was conducted on patients with intermittent claudication from peripheral vascular disease. Subjects were exposed for 2 hr to either 50 ppm CO or compressed air, after which time they exercised. Time until pain was reduced after breathing CO.

3.1.2.4 Effects on Pregnant Women and Unborn Offspring

The documents reviewed indicate that insufficient knowledge exists concerning the biological effects of CO during intrauterine development and the newborn period. One important consideration in evaluating the effects of CO exposure to pregnant women is that the pregnant woman's oxygen consumption is increased 15 to 25% during pregnancy but her oxygen carrying capacity is decreased 20 to 30% or more due to the decreased concentration of hemoglobin. Also of consideration are the facts that under steady state conditions the concentrations of human fetal HbCO is 10 to 15% greater than that in maternal blood and that after a step change in inspired CO concentration, the time for maternal HbCO to reach half its steady-state value is about 3 hr compared to 7.5 hr for fetal HbCO. This means that under a wide variety of circumstances the mean fetal HbCO content is greater than that of the mother.

Most of the few available studies on the effects of CO exposure on the embryo or fetus have used high CO concentrations. An example is a study in which pregnant rats were exposed to 15,000 ppm CO for 5 to 8 min 10 times on alternate days during their 21-day gestation period. The results were maternal unconsciousness and abortion or resorption of most fetuses. In another study pregnant rabbits were exposed continuously to a moderate level of CO (90 ppm). This exposure regime resulted in HbCO levels of 9 to 10% and caused a 11% decrease in birth weights. Mortality of young rabbits during the following 21 days increased to 25% from a control value of 13%.

3.1.3 Confined Space Workplace Concerns

Typically, diesel engines produce relatively low concentrations of CO (Hurn 1975). In confined workplaces where there is inadequate ventilation, however, this is not true. As described by Hurn (1975), in a confined area without adequate ventilation the combustion fuel-to-oxygen ratio rises steadily, and production of CO begins to increase exponentially. The result is that at some point in time the concentration of CO in the exhaust increases rapidly with a parallel rapid increase in the CO content of the confined workplace air mixture. This buildup of CO is markedly accentuated when engines are working under heavy load. To support this point, Hurn provides an illustration contrasting engines

under light versus heavy load in a closed environment of 10,000 ft³ (Figure 3.3). When a diesel engine is operated under light load in such an environment, a CO concentration of 5000 ppm is reached after slightly less than 4.5 gallons of fuel have been consumed, whereas under heavy load the same engine would use only 2.5 gallons of fuel to reach the 5000-ppm level. The other concern would of course be that as the CO level is increasing the oxygen level is decreasing. After 4 gallons of fuel have been consumed, the oxygen level will be only 11%.

The scenario described above is unlikely to occur in real life situations, but it does illustrate the potential problem in confined spaces if adequate measures (i.e., ventilation and/or emission control) are not employed. See Section 4.1.3 for information concerning the use of emission control devices and their effectiveness.

3.2 NITROGEN OXIDES

Oxides of nitrogen are usually classified by their oxidation state and include nitrous oxide (N₂O), nitric oxide (NO), dinitrogen trioxide (N₂O₃), nitrogen dioxide (NO₂), dinitrogen tetroxide (N₂O₄), and dinitrogen pentoxide (N₂O₅); the term NO_x is used where individual species are not specified. With respect to diesel emissions, however, the chief oxides of nitrogen emitted are NO and NO₂; therefore, the following sections will briefly discuss the formation and monitoring of these compounds in diesel exhaust, define their principal known adverse health effects, and evaluate potential NO_x buildup in confined spaces where diesel equipment is used. Unless otherwise stated, review documents by the NATO Committee on the Challenges of Modern Society (NATO 1973), NIOSH (NIOSH 1976), National Research Council (1977b), World Health Organization (WHO 1977), and Morton (1980) will be the sources of information.

3.2.1 Nitric Oxide

3.2.1.1 Formation and Monitoring

Nitric oxide is formed during the combustion of diesel fuel according to the reaction:



The resulting concentration of the nitric oxide is dependent on (1) the load placed on the engine, (2) the ratio of the mixed air and fuel, and (3) the amount of ventilation in the area where the exhaust gases are vented. In typical diesel combustion, 1 pound of fuel produces about 200 ft³ of exhaust gas of which 20 ft³ is CO₂ and roughly 1/3 ft³ is CO, and 1/3 ft³ is NO_x, with the remainder being free nitrogen and water vapor (Hurn 1975). According to Marshall (1978), the results of testing three engines ranging from 100 to 150 bhp (4-cycle, precombustion chamber) indicated that as the load placed on the engine increases, the amount of NO as a total percentage of NO_x increases. The reason is that as the load increases more oxygen is required for fuel combustion with less available for the formation of NO₂. However, even at light loads

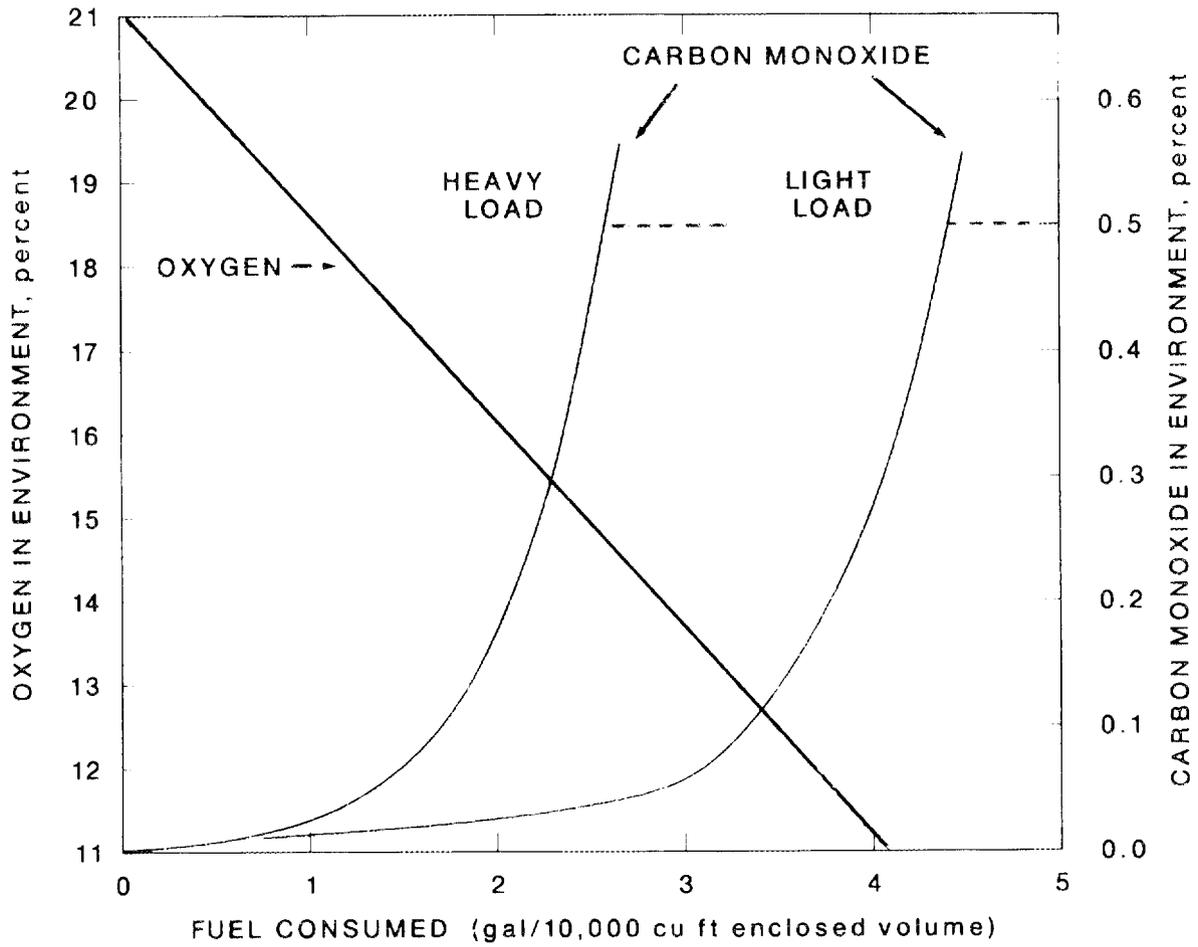
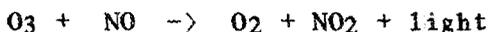


Figure 3.3. Concentrations of carbon monoxide and oxygen in an enclosed, non-ventilated environment as a function of diesel fuel consumed. Adapted from Hurn 1975.

NO is the predominant component of NO_x. With respect to the other two variables affecting NO formation (items 2 and 3 above), maximum NO formation occurs with 5 to 10% excess air and rapid dilution of exhaust gases slows the oxidation of NO to NO₂.

According to the NIOSH criteria document, most analytical procedures for NO involve oxidizing it to NO₂ and subsequently determining the NO₂ concentration. For mixtures of NO and NO₂, the method employs collection of NO₂ on a triethanolamine-impregnated molecular sieve surface, oxidation of the NO to NO₂ by a solid oxidizer, and collection of the converted NO on another section of triethanolamine-impregnated solid sorbent. The trapped NO₂ is then removed with an absorbing solution, and the concentration is determined spectrophotometrically. Alternatively, the WHO document indicates that chemiluminescence techniques based on the measurement of red light produced by the reaction:



are ideally suited to the measurement of NO and are accurate and reproducible over a wide range of concentrations with no important sources of interference. The major features are selective response to NO, sensitivity into the 1 µg/m³ range, and rapid response time (<1 second). These techniques are also well suited for the measurement of peak concentrations over averaging periods from 15 seconds to 1 hr, which, according to the WHO document, are largely inaccessible with manual methods. However, their high cost, complexity, and the necessity of some type of data logging system if long-term mean values are required for averaging periods from 1 day to 1 year are potential drawbacks.

3.2.1.2 Health Effects

No quantitative exposure-effect relationships in humans can be made for NO due to the absence of any measured environmental data. Nitric oxide has no apparent irritant properties, and its principal direct action is to convert hemoglobin (Hb) to methemoglobin (MetHb). Further, it is likely that the effects observed in NO poisoning are due to the hypoxemia which is secondary to the methemoglobinemia. Normal concentrations of MetHb in human blood range from 0 to 1.2 g/100 ml. The earliest clinical evidence of MetHb in human blood is cyanosis, which begins when the MetHb concentration reaches 10 to 15% of the total Hb. Symptoms such as labored breathing, reflecting low oxygen supply, are not likely to appear until blood levels of MetHb reach 30 to 40% of the total Hb.

In laboratory studies, animals that died following exposure to relatively high concentrations of NO (1200 ppm), were cyanotic and had high concentrations of MetHb in their blood. As measured in the arterial blood of Wistar rats, the threshold NO concentration for MetHb formation lies between 5 and 10 ppm. No evidence for MetHb formation could be found at 5 ppm after an exposure of 1 hr, whereas 10 ppm caused an increase of more than 50% over the initial MetHb value after the same exposure period. The current OSHA standard for NO is an 8-hr TWA of 25 ppm (OSHA 1982), which the NIOSH criteria document states should be

continued in the absence of data showing toxic effects for humans and animals exposed at or below this level. Tables B-8 and B-9 summarize clinical and epidemiologic studies on human exposure and the effects of exposure in experimental animals, respectively. According to the NIOSH criteria document, no studies were available to suggest that NO is either carcinogenic or mutagenic or that it causes reproductive effects. This document does, however, state that these are areas in which more research is needed.

3.2.1.3 Confined Space Workplace Concerns

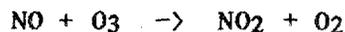
When an engine is denied adequate ventilation, as could happen when operating in a confined space such as a munition bunker, a portion of its exhaust is recycled into the intake with several changes occurring simultaneously (Hurn 1975). These changes include a decrease in oxygen levels and an increase in CO and CO₂. There is an initial increase in NO_x; but with increasing concentrations of CO₂ in the intake air and with the parallel decrease in oxygen, the amount of NO_x produced by the engine decreases. The overall result of the operation of a diesel engine in a closed environment according to Hurn is therefore a buildup of CO at an ever-accelerated rate while NO_x builds to a peak and then decays. Hurn further states that the peak level of NO_x does not represent an immediate danger for short-term exposure but that the high levels of CO do clearly present a short-term exposure hazard. With respect to the species of NO_x in the exhaust that would be favored in a confined space with inadequate ventilation, there would be an increase in the NO:NO₂ ratio because as the oxygen supply decreases as combustion continues, less oxygen would be available to form NO₂. The scenario for the vented exhaust gas would likely be an increasing rate in conversion of NO to NO₂ as the concentration of NO would be increasing, however, the amount of NO₂ formed would of course decrease over time as the oxygen concentration decreases. Thus with a decreasing oxygen supply, two simultaneously occurring events would influence NO₂ formation: (1) a decrease in the formation of NO and (2) a decrease in the rate of oxidation of NO to NO₂ both in the exhaust and in the environment into which the exhaust is vented.

3.2.2 Nitrogen Dioxide

3.2.2.1 Formation and Monitoring

Nitrogen dioxide is formed in the exhaust during the combustion of diesel fuel and to some extent in the atmosphere into which the exhaust is vented as a result of the oxidation of NO. In typical diesel combustion, 1 pound of fuel produces about 200 ft³ of exhaust gas of which roughly 1/3 ft³ is NO_x (Hurn 1975). Although most of the NO_x fraction of diesel exhaust is NO, according to Marshall (1978), as much as 30% may be in the form of NO₂ which, as indicated in the following section on health effects, is of greater concern from a human health perspective than NO. Marshall states that NO₂ formation is at a maximum when an engine is functioning under light load and, conversely, at a minimum when an engine is performing under maximum load. The reason is that at minimum load less oxygen is required for fuel combustion compared to an

engine operating at maximum load and thus more oxygen is available for oxidizing NO to NO₂. High combustion temperatures (such as would result from an engine operating under heavy load), rapid cooling and instantaneous dilution of exhaust gases promote the emission of high concentrations of NO and low concentrations of NO₂. Concerning dilution, Marshall states that if the exhaust products were diluted such that the initial level of NO was 25 ppm (the OSHA TWA), the time required for NO₂ levels to reach levels of 5 ppm (OSHA ceiling value) would be about 36 min. With further dilution of NO the time to reach 5 ppm NO₂ would of course increase. At low concentrations and in the presence of ozone (O₃), NO₂ can be formed by the reaction:



However, in confined spaces such as a munition bunker the concentrations of O₃ would probably be very low.

One monitoring method recommended in the NIOSH criteria document is collection of NO₂ on a triethanolamine-impregnated molecular sieve surface followed by removal with an absorbing solution and determination of NO₂ concentrations by reading the color of the solution with a spectrophotometer. This method is especially useful when both NO and NO₂ are to be measured (see Section 3.2.1.1). When only NO₂ is to be monitored, gas detector tubes that contain a chemically impregnated packing material which changes color to indicate the concentration of the NO₂ can be used. The use of detector tubes is inexpensive and provides rapid determination of NO₂ concentrations; however, because of interferences, difficulty in endpoint readings, and possible calibration inaccuracies, detector tubes are best used for estimating NO₂ levels rather than providing accurate determinations. Marshall (1978) indicates that chemiluminescence analyzers have demonstrated a capability to yield valid information for NO₂ and have been applied in a series of experiments conducted at the Energy Research Center in Bartlesville, Oklahoma. However, there are potential drawbacks; these are discussed in the formation and monitoring section for NO.

3.2.2.2 Health Effects

The principal effects of concern as a result of exposure to NO₂ are to the pulmonary system. Effects in humans include a decrease in effective lung compliance with a corresponding increase in expiratory and inspiratory volume after exposure to 4-5 ppm NO₂ for 10 min and marked mucosal irritation and increased pulse and respiratory rates after exposure to 25-100 ppm for 2 hr. Table B-10 summarizes several epidemiologic and experimental studies of human exposure to NO₂.

Criteria used to evaluate the toxic effects of NO₂ in animals include abnormal changes in respiration, cellular morphology of the pulmonary system, weight, reproduction, and immunoglobulin levels. In addition, reduced resistance to respiratory infection has been called the most sensitive response of animals to short-term exposures of NO₂. This reduced resistance is the result of interference by NO₂ with the lung's ability to remove inhaled deposited particles efficiently by altering

the phagocytic, enzymatic, and functional processes of the alveolar macrophages and of the ciliated epithelial cells. Table B-11 presents a summary of the results of several studies in which experimental animals were exposed to NO₂ in various exposure regimes and at different concentrations. Of particular interest to this problem definition study are exposures of fairly short duration. In one such study, rabbits were exposed to 5-50 ppm for 3 hr with a suppression of virus-induced resistance and phagocytic activity. Another is a 1-hr exposure of dogs and rabbits to 5-16 ppm which resulted in microscopic changes of the capillary endothelium and the alveolar epithelium. Although stating that more research is needed, the NIOSH criteria document could find no evidence in the available literature to indicate that NO₂ is either mutagenic or carcinogenic. One study did, however, find significant changes in estrus, litter size, and fetal weights in rats exposed to NO₂ at 1.3 ppm for 12 hr per day for 3 months. However, there are relatively few studies dealing with intermittent exposures of relatively short duration (15 min to 1 hr).

It is clear from the available literature that nitrogen dioxide (NO₂) presents a greater threat to human health than NO. For this reason OSHA has set a ceiling value of 5 ppm for NO₂ compared to an 8-hour TWA of 25 ppm for NO (OSHA 1982). Although concurring with the NO standard, the NIOSH criteria document on oxides of nitrogen recommends a ceiling value for NO₂ of 1 ppm because of a study in which healthy human subjects exposed to 4-5 ppm NO₂ had changes in arterial oxygen partial pressure, alveolo-arterial pressure gradients, and airway resistance. Although the specific concentration at which these changes begin to occur in normal human subjects is not known, NIOSH states that it is likely to be at the same or perhaps a slightly higher concentration than the 1.5-ppm level inducing pulmonary changes in humans with existing chronic bronchitis (See Table B-10).

3.2.2.3 Confined Space Workplace Concerns

The discussion for confined space concerns for NO is relevant for NO₂, and the reader is directed to Section 3.2.1.3. Even though Hurn (1975) has calculated that diesel engine operation in a closed environment would not produce NO_x concentrations high enough to cause serious health consequences, it should be noted that exposure to low concentrations of NO₂ is of concern, especially for those individuals with respiratory ailments (see the health effects section for NO₂ for further discussion).

3.3 HYDROCARBONS

The following discussion is based primarily on information provided in the following review documents: Air Quality Criteria for Hydrocarbons (PHS 1970); Vapor-Phase Organic Pollutants (NRC 1976); Patty's Industrial Hygiene and Toxicology (Sandmeyer 1981a, 1981b, 1981c); and Handbook of Toxic and Hazardous Chemicals (Sittig 1981).

3.3.1 Formation and Monitoring

Hydrocarbons are compounds consisting solely of hydrogen and carbon. They can be divided into three chemical groups: aliphatics with molecules of straight or branched carbon chains; aromatics with molecules of one or more six-carbon benzene rings; and alicyclics with molecules of one or more nonbenzene rings. Diesel fuel is a mixture of these compounds. As a result of uneven and incomplete combustion, diesel exhaust contains varying amounts of unburned hydrocarbons, original and partially degraded fuel molecules, and recombined intermediate compounds. Those compounds that are light and volatile will occur as gaseous components of the exhaust. Heavier molecular weight compounds will condense into aerosols or be adsorbed onto exhaust particulates. Particulate organics are discussed in Section 3.4.

The gaseous hydrocarbon fraction of diesel exhaust consists of light, cracked components (aliphatics) and heavier fuel-like compounds with carbon numbers up to about 24. Ethylene (7-83 ppm), acetylene (1-38 ppm), propylene (2-24 ppm), and small amounts of C₄-C₆ olefins (~1 ppm) are the predominant light hydrocarbons (Table 3.1). These constitute about 10-25% of the total. The heavy components (alicyclics and aromatics) consist primarily of indenenes, acenaphathenes, and benzothiophenes (Levins 1981); these chemical groups constitute the oily-kerosene odor component of diesel exhaust. The total concentration of this component may be 20 mg/m³ with individual compounds present at concentrations of 40 to 400 µg/m³.

TABLE 3.1 CONCENTRATIONS (ppm) OF ALKANES AND ALKENES
IN DIESEL EXHAUST^a

Chemical	0 HP 600 rpm	100 HP 1200 rpm	200 HP 2200 rpm	0 HP 600-2200 rpm
Acetylene	1.42	3.96	4.46	38.0
Ethylene	6.85	10.7	30.2	82.8
Propane	<0.05	0.07	0.07	0.77
Propylene	1.75	3.88	10.8	23.9
n-Butane	<0.05	<0.05	<0.05	0.14
1-Butene	0.37	1.08	2.91	7.00
iso-Butylene	0.26	0.51	1.33	2.85
iso-Pentane	<0.05	<0.05	<0.05	0.07
2-Butene	<0.05	0.20	0.92	1.56
n-Pentane	0.07	0.07	0.37	0.80
1,3-Butadiene	0.42	0.70	0.98	4.81
1-Pentene	0.29	0.71	1.74	5.24

^aTwo cycle, 6 cylinder, 220 hp engine and 50 cetane, No. 2 diesel fuel (Adapted from Linnell and Scott 1962b).

In addition to the oily-kerosene odor components of diesel exhaust, there are also compounds producing a smoky-burnt odor. These include alkenones; dienones; furan; furfural; methoxy-,hydroxy-, carbonyl-benzenes; benzofuranes; indanone; indenones; and naphthaldehydes (Levins 1981). The total concentration of these varies considerably but will be in the range of 5 mg/m^3 with individual chemicals present at levels of $0.1\text{--}10 \text{ }\mu\text{g/m}^3$ (Levins 1981).

The standard method for monitoring for total hydrocarbons in the exhaust gases from diesel engines is the heated flame ionization detector (SAE 1982). This procedure is required by EPA for manufacturers' compliance to federal guidelines (EPA 1980b). However, for a more detailed analysis of exhaust hydrocarbons, gas chromatography or mass spectrometry or a system interfacing these two techniques is the preferred method for identifying and quantifying hydrocarbons in the nanogram concentration range.

3.3.2 Health Effects

The short chain saturated aliphatic hydrocarbons in diesel exhaust are relatively nontoxic. No effect-levels for methane and ethane are 100,000 and 50,000 ppm, respectively. At higher concentrations, however, the gases may act as asphyxiants by reducing oxygen levels. The C₃ to C₈ alkanes show increasing narcotic properties. At 100,000 ppm, pentane produces narcosis in 5-60 min, and, at 10,000 ppm, octane causes narcosis in 30-90 min. The TLVs for these higher saturated alkanes are 50-1000 ppm (OSHA 1982). C₅-C₇ alkanes can cause chemical dermatitis after prolonged and repeated exposure, and C₆-C₁₆ compounds can also cause pulmonary damage when directly aspirated into the lungs. Studies have shown that the smaller alkanes are not teratogenic, mutagenic, or carcinogenic; however, some C₁₀-C₁₂ compounds may act as carcinogen promoters (Sandmeyer 1981a).

Although they are chemically more reactive than the alkanes, the alkenes have nearly as low a level of toxicity. They are not neurotoxic and are only weak anesthetics. Although repeated exposures to high concentrations have produced hepatic damage and hyperplasia of bone marrow in animals, similar effects have not been seen in humans. The recommended maximum permissible occupational exposure levels are generally set at about 1000 ppm, which is the level above which oxygen levels would be critically low.

The alicyclic hydrocarbons are similar to the aliphatics in that they have a low level of acute toxicity and do not bioaccumulate. At high concentrations they may act as anesthetics and central nervous system depressants. Tetralin is an irritant to the eyes, skin, and mucous membranes. In one case study, an oral dose of 1.5 mL/kg was not lethal, and resulted only in nausea, vomiting, intragastric discomfort, and transient liver and kidney damage. For decalin the lowest inhalation dose to cause an effect in man was 100 ppm, and for cyclohexane the no-effect level for a nonhuman primate species was 1243 ppm.

Indans and indenenes are other alicyclics found in diesel exhaust. Indan itself is reported to be relatively nontoxic, but exposure to high vapor concentrations of indene (800-900 ppm) may produce liver damage and splenic and renal injury (Sittig 1981). The recommended maximum permissible air concentration is 10 ppm and the short-term exposure limit 15 ppm (ACGIH 1982).

Aromatic hydrocarbons are much more toxic than aliphatics and alicyclics. The vapors are more irritating to the mucous membranes. The simple aromatics, such as benzene, toluene, styrene, and xylene produce such effects at concentrations of 100 to 200 ppm. Other observed symptoms are fatigue, weakness, confusion, and skin paresthesia. Chronic exposure to benzene can cause hematological changes including leukopenia and anemia. Similar effects result from exposure to xylene, but not toluene. The 8-hr TWA for benzene is 10 ppm, the ceiling value 25 ppm, and the maximum peak 50 ppm for 10 min. For other alkyl benzenes, the TLVs range from 25 ppm (for trimethylbenzene) to 100 ppm (for styrene) (ACGIH 1982; OSHA 1982).

Naphthalenes make up another group of aromatics found in diesel exhaust. Naphthalene may cause eye and respiratory tract irritation, headache, nausea, and profuse perspiration when inhaled. Optic neuritis may also occur. Acute exposures can result in hemolytic anemia. The OSHA (1982) occupational exposure limit is 10 ppm, and the ACGIH (1982) recommended short-term exposure limit is 15 ppm. The alkyl naphthalenes appear to be slightly less toxic than naphthalene. Unlike naphthalene, methyl naphthalene is not a skin irritant or photosensitizer. However, more complex naphthalene derivatives, such as acenaphthene (1,8 ethylenenaphthalene) may cause substantial irritation to the skin and mucous membranes. This compound causes genotoxic effects in lower organisms and might therefore be mutagenic to humans. There are no OSHA or ACGIH standards for acenaphthene.

Other odor components consist of oxidized or hydroxylated derivatives of the above-mentioned compounds, as well as heterocyclics such as furan. In general the carbonyl derivatives tend to be chemically more reactive than the parent compounds and, consequently, toxic effects may be greater; however, specific data on many of these compounds are not available. The aliphatic ketones are relatively nontoxic, but some are neurotoxic at high concentrations. TLVs range from 5 to 100 ppm. The aliphatic aldehydes are strong irritants, and these will be discussed separately (see Section 3.5). Of the higher molecular weight compounds, the benzofurans, methoxybenzenes, and indanones are probably the least toxic, but some hydroxybenzenes, furfurals, and naphthaldehydes are strong irritants with TLVs of about 5 ppm.

3.3.3 Confined Space Workplace Concerns

The light gaseous hydrocarbons in diesel exhaust appear to pose no major health hazards for confined space workplaces. This is due to both the low concentrations of these compounds as well as the high toxicity thresholds. Total concentrations of alkanes and alkenes may be about 20 ppm; minimum safe exposure limits are generally several orders of

magnitude higher. Furthermore, there is no evidence that chronic exposure to low levels of these gases causes any adverse health effects.

The slightly higher molecular weight hydrocarbons, which are associated with diesel odor, such as simple alicyclic and aromatic compounds, have a greater potential for toxicological effects in confined workplaces. Toxicological effects vary considerably from compound to compound. Some are primarily skin or eye irritants, others can cause systemic effects following high acute exposures. The TLVs for these compounds may be as low as 10 ppm. The limited data available suggest that diesel exhaust levels of these compounds may be within safe limits (e.g., for benzene and toluene exhaust concentrations are 0.1-1 ppm; see Table B-6). There is also very little quantitative information available concerning the levels of alicyclic, heterocyclic, and aromatic oxygenates in diesel exhaust, and consequently it is difficult to evaluate potential health effects in confined space workplaces. The TLV for a number of these compounds is 5 ppm, but further study is needed to determine whether this exposure limit will be exceeded in confined space workplaces before other factors, such as carbon monoxide and NO_x , reach critical levels.

Additional investigations are also needed to determine to what degree these exhaust gas hydrocarbons undergo further chemical reactions after leaving the exhaust system. In confined spaces with limited ventilation these hydrocarbons will most likely be recirculated through the combustion system in the intake air. Consequently, they will undergo further oxidation and other chemical reactions, and this would have the net effect of limiting the air concentrations within the workplace, while at the same time resulting in an increase in the air levels of the more oxidized species, such as the aliphatic aldehydes.

3.4 PARTICULATES

Suspended particulates are very finely divided solids or liquids that are dispersed in air. To remain suspended for a period of hours or more, the particles must have a settling velocity that is small in comparison with the effects of natural air movements. This minimum settling velocity is equivalent to, or less than, that of spherical particles of unit density (1 gm/cm^3) having a diameter of $10 \mu\text{m}$ or less. Because suspended matter can vary in size, shape, and density, it is useful, for comparison purposes, to indicate its settling velocity in terms of the aerodynamic diameter, or the diameter of spherical particles of unit density with equivalent settling rates. The aerodynamic diameter is not only indicative of how long the particles would remain suspended, but also to what extent they would be inhaled and where they would be deposited in the upper and lower respiratory tract (Figure 3.4).

The exhaust of a diesel engine contains a large amount of particulate matter (see Table B-6), most of which is less than $1 \mu\text{m}$ and therefore within the size range of suspended, respirable particles. These particles consist of elemental carbon; unburned hydrocarbons; water-soluble inorganics such as sulfuric acid and sulfates (which may also be

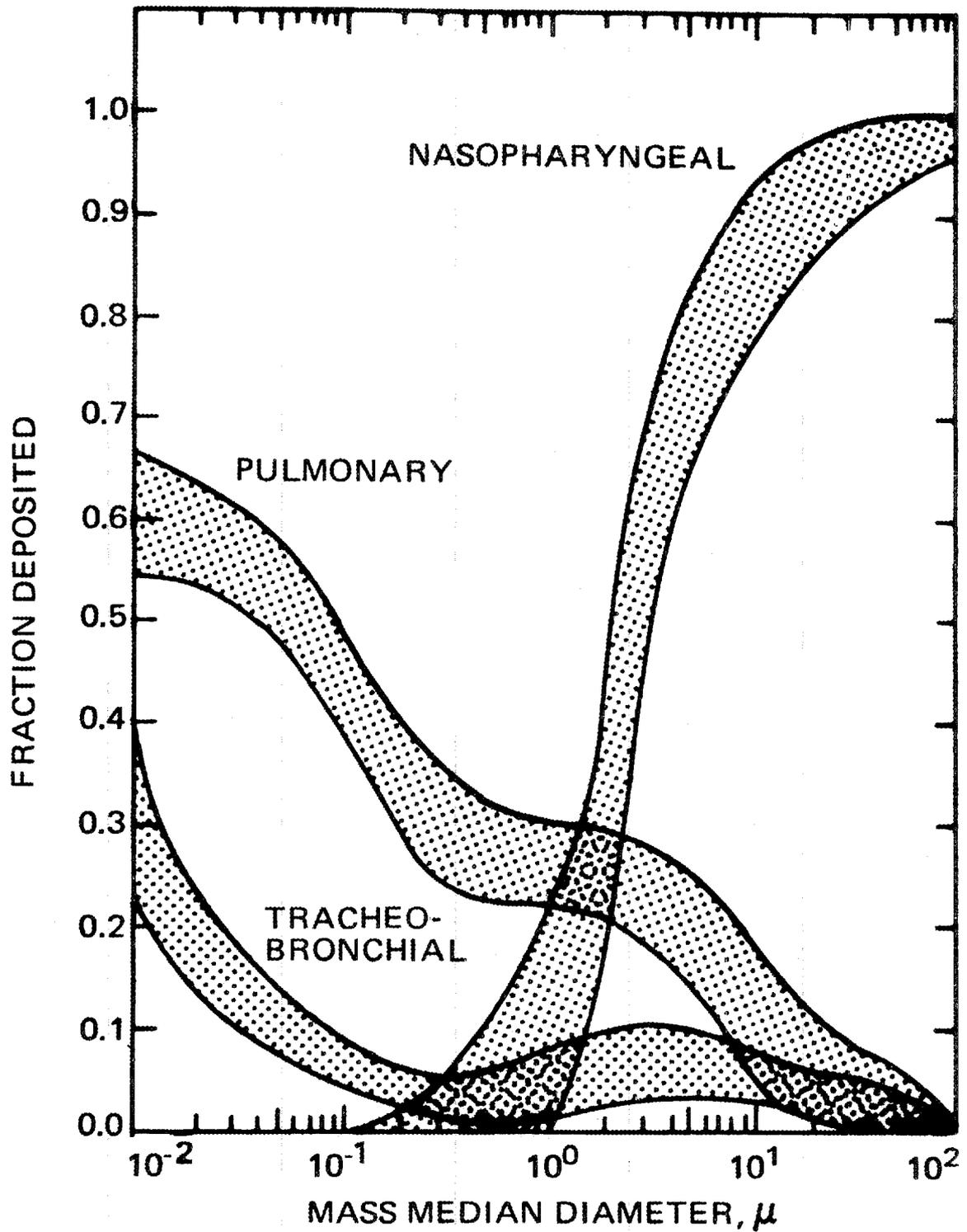


Figure 3.4. Fraction of particles deposited in respiratory tract compartments as a function of particle diameter. Adapted from Task Group on Lung Dynamics 1966.

present in the form of liquid aerosols); and small amounts of silicon, iron, zinc, lead, copper, manganese, and other minerals. A detailed discussion of sulfuric acid and other sulfates is given in a separate section (see Section 3.6). Of the other particulate components, little will be said about the trace metals present, since they occur in only minute amounts and, in themselves, are unlikely to be major health hazards. The bulk of the particulate matter consists of elemental carbon (30-75%) and unburned hydrocarbons (10-40%). These may have a direct physical effect as a result of deposition in the lungs, and/or they may have a chemical effect depending on rates of solubilization and bioavailability.

The following review of the potential health effects of diesel particulates and their associated hydrocarbons is based primarily on information provided in the following review documents: Sulfur Oxides and Suspended Particulate Matter (WHO 1979b), Health Effects Associated with Diesel Exhaust Emission (Santodonato et al. 1978), Health Effects of Exposure to Diesel Exhaust (NRC 1981a), Health Effects of Diesel Exhaust Emissions (EHA 1978), Air Quality Criteria for Particulate Matter (PHS 1969), and An Initial Assessment of the Literature on the Measurement, Control, Transport, Transformation and Health Effects of Unregulated Diesel Engine Emissions (Andon et al. 1979).

3.4.1 Formation and Monitoring

Diesel particulate matter forms in the engine combustion chamber in areas where the temperature is sufficiently high for decomposition of the original fuel hydrocarbons, but where there is insufficient oxygen for complete oxidation to CO₂ and water (Johnson 1975). The elemental carbon formed in this way is insoluble, nonvolatile, crystalline particles in the submicron size range. Various large molecular weight, incompletely burned hydrocarbons of low volatility (as well as new compounds formed by chemical reactions between these hydrocarbons) condense or are adsorbed onto the carbon nuclei to form the large (up to 10 microns) irregularly shaped or chainlike particles typical of diesel exhaust. Although the actual maximum diameter of diesel particles may be as great as 10 μm, the mass median aerodynamic diameter is usually only a few tenths of a micrometer, and the number median diameter is even smaller, with less than 1% of the particles larger than 0.05 μm (Williams 1982).

There are two major methods for analyzing for suspended particles (Perez 1981, WHO 1979b). In the gravimetric method the particles are removed from a sample of air (usually a high volume 24-hr sample) by filtration onto a glass fiber material or through a synthetic membrane, and the collected material is weighed under controlled temperature and humidity conditions. By miniaturizing the components and using a battery-operated air pump, this method can be adopted as a personal air sampler (Sherwood and Greenhalgh 1960). The second sampling method involves the use of an electrostatic precipitator in which the particles are deposited electrostatically on the inside surface of a metal tube. The amount of deposited material is then determined by direct weighing.

Separation of the elemental carbon and organic carbon components of diesel particulates can be accomplished by solvent extraction (Soxhlet) or thermogravimetrically. Fractionation of the organic carbon extracts has been accomplished with high-performance liquid chromatography. This technique has also been used for the identification and quantification of specific compounds such as benz(a)pyrene. For a more complete chemical analysis of the organics, including the polynuclear aromatic hydrocarbons, mass spectrometric methods have been used.

3.4.2 Health Effects

Although suspended particles may have transitory irritant effects on the eyes and skin, they are potentially most damaging to the respiratory tract. Inhaled particles can cause direct toxicological effects to epithelial tissues, alter pulmonary functions, or modify the toxicological effects of other inhaled compounds. Furthermore, compounds adsorbed onto particulates may cause damage to the respiratory tissues, or produce toxic effects at other sites in the body if the particles with which they are associated are phagocytized and enter the circulatory or lymphatic systems. Although much of the inhaled particulate matter is transported out of the respiratory tract by the mucociliary apparatus, it can then be swallowed, and enter the digestive tract where the particles or the adsorbed compounds might have a direct effect on the intestinal mucosa or be taken up and transported through the blood.

In evaluating the potential toxic effects of diesel exhaust particulates, it is very difficult to isolate the elemental carbon and adsorbed hydrocarbons from other exhaust components, gaseous and nongaseous, without modifying various physical and chemical factors that might substantially alter any physiological effects. Thus, organic solvent extracts of the adsorbed hydrocarbons, when tested in an in vitro system may have very different effects than the particulates have in in vivo systems. Furthermore, the particulates may act as catalysts to enhance the conversion of copollutants NO and SO₂ to their corresponding acids, and thus increase their irritant potency (see Section 3.6). Such factors must be kept in mind when the health effects data for diesel engine particulates are evaluated.

3.4.2.1 Carbon Component

Elemental carbon comprises 30-75% of the diesel exhaust particulate matter. Although the general consensus is that elemental carbon is chemically unreactive and therefore of little toxicological importance (Sittig 1981), as a physical irritant it may contribute to alterations in pulmonary structure and/or function, particularly if exposure levels are sufficiently high to overload the normal pulmonary clearance mechanisms of phagocytosis and mucociliary transport. Actual analysis of these effects under experimental conditions is complicated by the fact that the carbon component of diesel exhaust particulates, in the absence of the adsorbed organic component, would have different physical characteristics (size, shape, density) and, thus, have different lung deposition characteristics and possibly different effects. Although no studies have attempted to evaluate the physiological effects of the carbon

component of diesel particulates alone, some useful comparative data can be obtained by reference to studies on carbon black (PHS 1969, NIOSH 1978, ILO 1983). This material consists of fine carbon particles of 0.01-0.5 μm and is therefore similar in size to diesel particulates. It is obtained by combusting oil or natural gas. Laboratory and epidemiological studies indicate that exposure to carbon black at concentrations of 8.2 mg/m^3 or above for more than 10 years can result in pneumoconiosis and pulmonary fibrosis, similar to that caused by nonspecific respiratory irritants. Other possible effects associated with carbon black include skin irritation, keratosis and leukoplakia, and myocardial dystrophy. The current OSHA 8-hr TWA for carbon black is 3.5 mg/m^3 . Thus, there is a theoretical basis for adverse pulmonary effects resulting from chronic exposure to diesel particulate carbon. However, like diesel exhaust particulates, carbon black normally has associated with it a varying amount (0.01-0.5%) of adsorbed organic compounds including polynuclear aromatic hydrocarbons (PAH), and these may contribute to some of the observed effects. The presence of PAHs has generated considerable concern about the potential carcinogenicity of carbon black. This has also been true for diesel exhaust particulates, as will be discussed in the next section.

3.4.2.2 Organic Compounds

As a result of the incomplete combustion of diesel fuel, the exhaust from diesel engines contains numerous organic compounds (see Table B-5), including low molecular weight volatile organics (discussed in Section 3.3) and high molecular weight nonvolatile organics associated with the particulate matter of the exhaust. The nonvolatile organics constitute 10-40% of the particulate matter and include a wide variety of polycyclic aromatic compounds such as anthracenes, phenanthrenes, fluorenes, benzfluorenes, dibenzfluorenes, fluoranthenes, pyrenes, chrysenes, perylenes, coronenes, carbazoles, quinolines, and cholanthrenes. The benzo- and dibenzo- derivatives of some of these compounds have also been found, as well as derivatives having methyl-, methoxy-, hydroxy-, nitro-, and carbonyl substituents. It has been estimated that as many as 20,000 different organic compounds may be associated with diesel exhaust (Vostal 1980). Only a small percentage of these have been identified, and for only a few is there quantitative data (see Table B-6).

Questions have been raised concerning the potential toxic effects of these particulate-associated organics on individuals exposed to diesel exhaust. Generally, such high molecular weight compounds tend to have a relatively low level of chemical reactivity, and compounds which are adsorbed onto an inert material such as elemental carbon tend to have a reduced bioavailability. Furthermore, most of the PAHs are either insoluble or have a very low aqueous solubility; consequently, the rate at which they are solubilized in the pulmonary tissues is expected to be slow. Finally, because many organic compounds are associated with particulates, the concentration of individual compounds is expected to be quite low, which would minimize potential toxic effects unless synergistic interactions occur.

There is no evidence that diesel particulate organics contribute significantly to the immediate pulmonary irritation that can result from exposure to diesel exhaust. Although some of the partially oxidized hydrocarbons, such as those with carbonyl substituents may be potentially irritating, any such effect would be small in comparison to the effects produced by the primary irritants in diesel exhaust, sulfuric acid (see Section 3.6) and aliphatic aldehydes (see Section 3.5). However, as part of the particulate matter, the adsorbed organics would contribute to any long-term direct pulmonary changes, such as fibrosis, which the particles might cause. Although chronic exposure to diesel exhaust has been shown to result in pathological changes (pneumonitis) in the lungs of some experimental animals, this has not been true in all studies, and in some cases no abnormal pulmonary changes occurred even though particulate concentrations were as high as 7 mg/m^3 . Unfortunately, in very few studies have attempts been made to differentiate between effects caused by diesel particulates and those caused by the various gaseous copollutants. Thus, the variable results may be due to differences in exhaust compositions, but other factors to be considered are differences in species susceptibility and exposure conditions, such as frequency and length of exposures. The variable results also make it difficult to extrapolate to human exposure situations. Epidemiological studies of occupationally exposed subjects, such as garage workers, miners, and truck drivers, do not reveal any consistent association of chronic obstructive lung disease with exposure to diesel exhaust. Thus, the role of particulates and their adsorbed organics is difficult to assess without further experimental study.

Another question yet to be answered unequivocally concerns the possible mutagenicity and carcinogenicity of diesel particulate matter. Several of the adsorbed PAHs, such as benz(a)pyrene, dibenzpyrenes, benz(a)anthracene, dimethylbenzanthracene, dibenz(a,h)anthracene, chrysene, and cyclopenteno(c,d)pyrene are known carcinogens. Many of these compounds are also mutagenic in microbial systems as well as in cell cultures of higher organisms. Likewise, organic solvent extracts of diesel particulate matter which contain these PAHs have been shown to be mutagenic and carcinogenic (Claxton 1980; Nesnow and Huisingh 1980; Pitts et al. 1982). Although no epidemiological studies have demonstrated a significant increase in lung cancer in workers exposed to diesel exhausts (NRC 1981), Harris (1981) has calculated that the upper confidence limits of potential risk for such workers represents about a 0.05% proportional increase in incidence per unit of exposure ($1 \mu\text{g}$ of particulates/ m^3 for one year). Harris notes, however, that the estimated lower confidence limits must include the possibility of no effect or even a reduction in lung cancer. If either of the latter possibilities proves to be true, it may be due to the fact that the PAHs occur in concentrations too low to be effective and/or that they have only a limited bioavailability.

Limited quantitative data concerning levels of PAHs in diesel exhaust indicate that for most compounds concentrations are usually below 0.1 mg/m^3 (see Table B-6). For benz(a)pyrene, concentrations of 0.00005 mg/m^3 to less than 0.6 mg/m^3 have been reported. Benzanthracene has been measured at 0 to 0.017 mg/m^3 . These concentrations, as well as

that of the total particulates, will be reduced by dilution with air. Furthermore, lung retention of diesel particulates has been estimated to be only 15% of the inhaled amount, and this would reduce exposure levels accordingly. Finally, the most important factor is the rate at which the adsorbed carcinogens would leach out of particulates deposited in the lungs. Although much conflicting evidence exists, some in vitro studies suggest that the adsorbed PAHs are slowly released from diesel particulates upon incubation with some physiological fluids such as serum and lung cytosol (McClellan et al. 1982); however, only a small amount of the mutagenicity appears to be transferable to the physiological media (Brooks et al. 1980). Other studies have demonstrated that diesel particulate suspensions have a similar level of cytotoxicity to human fibroblast cultures as do organic solvent extracts of these particulates, indicating that the adsorbed compounds were biologically active (McCormick et al. 1980). The particulates without the adsorbed organics were inactive in this system.

Under in vivo conditions, however, much of diesel particulate matter deposited in the alveoli is engulfed by macrophages. Under low exposure conditions much of this material would be transported up the respiratory tract by way of the mucociliary apparatus. Under heavier exposure conditions some of the macrophages may also remove the particulate material by way of the lymphatic system, and in chronic assays the lymph nodes may become black as a result of the accumulation of particulates. Although in some studies the particulates have been found in lung epithelial cells, generally the macrophages carry the heaviest burden. Within the macrophages the particulates are confined within lysosomal membranes. Thus, the lack of observed biological activity of the particulate-associated PAHs may be due to their nonbioavailability as a result of their being sequestered in intracellular lysosomal vesicles. They may also, to some degree, be inactivated or degraded by lysosomal enzyme systems (Siak and Strom 1981).

A final question concerns the possibility that the adsorbed PAHs might be leached out of the particulate matter in the digestive or lymphatic systems. The chemicals could then be taken up and transported through the body to cause adverse physiological effects at sites far removed from the lungs. There is no laboratory evidence for such effects, and one study which showed that aryl hydrocarbon hydrazase activity in liver and lung tissue was not induced in animals inhaling diesel exhaust, suggests that any PAHs released from the particulates after phagocytosis, must have been metabolically degraded to some degree (Chen and Vostal 1981).

3.4.3 Confined Space Workplace Concerns

The available data indicate that any immediate toxic effects caused by exposure to diesel exhaust, such as eye, skin, or respiratory irritation, would most likely be caused by components other than the particulates or their adsorbed organics. The particulate matter might however contribute to the irritant effects by acting as a catalytic substrate for the oxidation of SO₂ to sulfuric acid. In confined spaces a buildup

of diesel particulates might enhance this conversion process and thereby increase the irritant potency of the exhaust.

Long-term effects of diesel particulates in confined spaces would be mitigated primarily by the necessarily intermittent nature of the exposures. Although there is the possibility that chronic, long-term exposures to diesel particulates might eventually induce fibrotic changes in pulmonary tissues, no epidemiological evidence indicates that this occurs in occupational exposure situations, and it would be even less likely to occur with short-term intermittent exposures.

To the degree that inhalation of diesel particulates places a stress on pulmonary clearance mechanisms, it might be anticipated that even intermittent exposures would exacerbate preexisting congestive lung conditions and possibly increase a worker's susceptibility to pulmonary infections. Both of these possibilities require further laboratory and epidemiological investigation.

Concerning the carcinogenicity of compounds associated with diesel particulates, no evidence exists to suggest that under confined space conditions the carcinogenic risk would be increased significantly; however it should be noted that there have been few laboratory studies which have used multiple short-term exposure regimes. Theoretically, short-term exposures to large concentrations of diesel particulates might cause transitory overloading of the pulmonary clearance mechanisms and result in more of the particles being deposited in pulmonary epithelial cells, where they may have a greater toxicological effect than those engulfed by the alveolar macrophages. Clearance of the particles from the lungs would not necessarily be enhanced by the intermittent nature of the exposures because clearance times for particles in the alveoli are on the order of days to weeks.

3.5 ALDEHYDES

Aldehydes are hydrocarbon oxidation products characterized by the CHO chemical group. In the atmosphere, aldehyde formation is dependent on photooxidation reactions; however, in diesel combustion systems, the high air to fuel ratios and incomplete combustion reactions account for the high levels of aldehydes produced. Although both aliphatic and aromatic aldehydes can be found in diesel emissions, the low molecular weight aliphatics and, particularly, formaldehyde and acrolein, are the predominant species. Aldehydes in general are chemically more reactive than their hydrocarbon parent compounds and yield acyl and alkyl radicals and carbon monoxide. Thus, on a theoretical basis they have the potential for causing greater adverse health effects.

The following discussion is based on the data given in the references in Tables B-12 and B-13, and in those in the review documents Formaldehyde and Other Aldehydes (NRC 1981b); Formaldehyde (IARC 1982); Formaldehyde Toxicity (Gibson 1983); Handbook of Toxic and Hazardous Chemicals (Sittig 1981).

3.5.1 Formation and Monitoring

In compression-ignition engines such as the diesel, a number of chemical reactions take place before the actual ignition of the fuel mixture (see Henein 1973, for review). During this preignition (or ignition delay) period, the concentration of aldehydes in the cylinders increases (as well as that of several liquid hydrocarbons and peroxides) until it reaches a peak level just before the start of the pressure rise resulting from the combustion process. Autoignition occurs when the concentrations of these intermediate compounds reaches a critical level. Because the combustion process does not occur uniformly throughout the cylinder, the aldehydes in some areas will not undergo further oxidation, and consequently they are emitted in the engine exhaust, along with the other combustion by-products, such as hydrocarbons, particulates, NO_x, SO₂, and CO.

The aldehyde content of diesel engine exhaust is not regulated by the EPA, and consequently, there is no EPA-specified analytical method. Although numerous methods have been developed, none of these are completely specific for aldehydes alone, and, in addition, different aldehyde species often give different levels of response.

Because the low molecular weight aliphatic aldehydes are very reactive and volatile, most analytical techniques for their determination require, as an initial step, the formation of relatively stable and nonvolatile chemical complexes (see NRC 1981b for review). Chemicals used to form these complexes include: bisulfite, hydroxylamine, semicarbazone, phenylhydrazines, chromotropic acid, 3-methyl-2-benzothiazolone (MBTH), Girard-T reagent, 4-hexylresorcinol, pararosaniline, and 4-amino-3-hydrazino-5-mercapto-1,2,4-triazole (Purpald). Many of these aldehyde complexes are chromophores that can then be analyzed spectrophotometrically.

In analyzing for total aliphatic aldehydes in diesel exhaust, MBTH, Purpald, and 2,4-dinitrophenylhydrazine (DNPH) are the most commonly used reagents. The MBTH method can detect aldehyde concentrations as low as 0.03 ppm, but formaldehyde produces a disproportionately strong response compared to the other aldehydes, and nitrogen dioxide and other nitrogen-containing compounds can cause interferences. The Purpald method has less interference from nonaldehyde compounds, but gives different responses for different species of aldehydes. The detection limit is 0.04 ppm. In the DNPH method, ketones cause interferences, and the detection limit is only 0.2 ppm.

When coupled to a gas chromatograph, the DNPH method has been used to identify and measure individual species of aldehydes in diesel exhaust (Springer and Baines 1978); however, in this method some derivatives, such as those of propionaldehyde and acrolein, overlap and are difficult to separate from acetone, which interferes. Thin layer chromatography and HPLC, coupled to a UV detector, can also be used for separation and identification.

Two of the most important aldehydes occurring in a dieselized workplace environment are formaldehyde and acrolein. The chromotropic acid and pararosaniline methods have been used extensively for formaldehyde determinations. Both are spectrophotometric techniques with a minimal detection limit of about 0.04 ppm. Nitrogen dioxide, alkenes, phenol, and other aliphatic aldehydes interfere in the chromotropic method, and sulfur dioxide and cyanide in the pararosaniline method. The Girard-T reagent method with polarographic detection has been validated for formaldehyde analysis by NIOSH (1979), but other volatile aldehydes cause significant interference.

The 4-hexylresorcinol method with spectrophotometric detection is specific for acrolein and is the NIOSH-recommended method for workplace monitoring. The detection limit is about 0.01 ppm, and there is little interference from other aldehydes.

There are several direct analysis methods for aldehydes. These include microwave, infrared, and laser-fluorescence spectroscopy, gas chromatography, HPLC, and ion chromatography. The spectroscopic methods are expensive laboratory techniques not routinely used. Gas chromatographic analysis for formaldehyde has been attempted with various types of detectors (e.g., flame ionization, thermal conductivity, electron capture, photoionization), but these methods have one or more drawbacks such as thermal degradation of the aldehydes, lack of sufficient sensitivity, nonlinear response, or short lifetime. Ion chromatography is reported to be very sensitive to formaldehyde, but other organic compounds may also be oxidized to the formate ion and thus give inaccurate results. The use of HPLC for aldehyde analysis avoids the problems of thermal degradation associated with gas chromatography; however, an adequate detector system has not been found for direct analysis. Indirect HPLC analysis of the DNPH-aldehyde derivatives (also ketones) has been achieved with the use of a ultraviolet detector (Perez 1981; Creech et al. 1982). Although the precision of this method is very good in the range of 0.05 to 10 ppm, and most of the C₁-C₅ aldehydes are clearly separated and identified, acrolein is not completely separated from the ketone acetone (Creech et al. 1982).

For workplace monitoring of organic vapors, active and passive (diffusion-controlled) badge monitors have been developed (Lautenberger et al. 1980). In these monitors the organics are adsorbed onto activated charcoal or activated alumina, then desorbed with an organic solvent and later analyzed by chromatographic or spectroscopic methods. These methods have been used to monitor aldehyde exposure levels of coal miners working on or near operating diesel engines (Holland 1978).

3.5.2 Health Effects

This section will focus mainly on the health effects of formaldehyde and acrolein, as these are the two main aldehyde components of diesel exhaust. However, because the toxicological effects of most aldehydes are similar, differing only in degree, it is the total aldehyde concentration which must be considered in evaluating potential health hazards.

3.5.2.1 Irritancy

Aldehydes are primary irritants to the skin, eyes, and mucous membranes. Olefinic aldehydes, such as acrolein, are generally stronger irritants than the saturated aldehydes, such as formaldehyde. The latter, in turn, are stronger irritants than alicyclic and aromatic aldehydes, such as benzaldehyde. The irritant potency decreases with increasing molecular weight or increasing chain length. The lower molecular weight species generally have a greater effect on the eyes and upper respiratory tract, while the heavier species have a more direct pulmonary effect.

The irritant threshold for formaldehyde can be as low as 0.1 ppm for some people, but with prolonged exposures a tolerance level can develop such that the threshold rises to 2-3 ppm. Short exposures (10-30 min) to 4-5 ppm are usually severely irritating. At concentrations of 10-20 ppm, breathing becomes difficult, and at 50 ppm and above, severe pulmonary damage may occur. Laryngitis, bronchitis, and bronchopneumonia have been observed following acute exposures. The OSHA (1982) 8-hr TWA for formaldehyde is 3 ppm, the ceiling value 5 ppm, and the 30-min ceiling value 10 ppm (Table B-12). NIOSH (1982) recommends a 30-min ceiling value of only 1 ppm.

Acrolein is a much stronger eye and respiratory tract irritant than formaldehyde. A concentration as low as 0.3 ppm can cause acute irritation in 5-10 min. Lacrimation occurs in seconds after exposure to 0.67 ppm, and eye irritation becomes intolerable after a few minutes exposure to 1 ppm. The 8-hr TWA for acrolein is 0.1 ppm (OSHA 1982; ACGIH 1982), and the short-term exposure limit 0.3 ppm (ACGIH 1982).

Crotonaldehyde is similar to formaldehyde in its irritant potency. A 10-min exposure to 4 ppm results in irritation to the eyes, nose, and respiratory tract. The 8-hr TWA for crotonaldehyde is 2 ppm (OSHA 1982), and the recommended STEL is 6 ppm (ACGIH 1982). The irritant potencies of the other aldehydes in diesel exhaust (e.g., acetaldehyde, propionaldehyde, benzaldehyde) are only 1/10 to 1/100 that of crotonaldehyde, and, because of their relatively low concentrations in diesel exhaust (Table B-12), they would contribute only a small amount to the overall irritant effect.

Some individuals may become sensitized to aldehydes, particularly formaldehyde, such that even exposures below normal threshold values will induce skin reactions. There is also the possibility, though unconfirmed, that sensitization might result in asthma-like symptoms.

3.5.2.2 Pulmonary Effects

Both animal and human studies indicate that the more reactive aldehydes can cause minor changes in pulmonary function even at very low exposure levels (Table B-13). Most saturated aldehydes tend to increase respiratory rates, but formaldehyde and unsaturated aldehydes, such as acrolein, decrease respiration (Ikeda et al. 1980). An increase in

pulmonary resistance can also occur, indicative of a bronchoconstrictive effect, which may exacerbate a preexisting congestive lung problem.

It has also been reported that, in mice, exposure to acrolein can enhance susceptibility to bacterial infection in the lungs, in a manner similar to that caused by dilute diesel exhaust (Campbell et al. 1980).

Acute exposures to aldehydes can result in degeneration of epithelial tissues of the lungs and upper respiratory tract. Hyperemia, hemorrhages, edema, and cellular necrosis have been observed.

3.5.2.3 Carcinogenicity

In several animal species tested, chronic exposure to acrolein, acetaldehyde, and formaldehyde resulted in metaplastic and hyperplastic changes in the epithelial cells along the entire length of the respiratory tract. These histopathological changes were followed by a significant increase in the occurrence of nasal and laryngeal carcinomas. Although there is no laboratory or epidemiological data to suggest an increased carcinogenic risk in humans occupationally exposed to aldehydes, the animal studies indicate that these compounds should be treated as potential human carcinogens (Griesemer et al. 1982).

3.5.2.4 Mutagenicity

Under certain experimental conditions, some of the aldehydes, particularly the more reactive ones such as formaldehyde, acrolein, and crotonaldehyde, are mutagenic in microbial assay systems. In tests on cell cultures of higher organisms, including human lymphocytes, aldehydes induced an increase in sister chromatid exchanges. Other in vitro studies have shown that aldehydes can cause increases in micronuclei and chromosomal aberrations. Further studies are needed to determine the genotoxic effects of aldehydes under in vivo conditions.

3.5.2.5 Teratogenicity

The teratogenicity of formaldehyde has been tested in dogs, mice, and rats, and in all cases it was found that formaldehyde caused no morphological abnormalities. Similar results have been found for acrolein tested in vitro on rat embryos. Acetaldehyde, however, has been found to be teratogenic in rats and mice; the most severe abnormalities being those affecting the central nervous system. For the other aldehydes present in diesel exhaust, there is insufficient data to determine whether they are teratogenic or not.

3.5.2.6 Other Effects

Animal studies have demonstrated that acute exposures to a variety of aldehydes can produce histopathological changes and physiological effects which do not occur following chronic exposures. The most severe of these effects is cellular damage to the kidney and liver following exposure to low molecular weight aldehydes such as formaldehyde,

crotonaldehyde, and acrolein. This damage results in hyperemia, hemorrhages, and edema of the affected organ.

Other effects seen in animal studies include increased blood pressure (acetaldehyde and propionaldehyde), increased heart rate (saturated aldehydes), decreased heart rate (formaldehyde and unsaturated aldehydes), changes in blood cell counts (acetaldehyde), depression of motor activity (acetaldehyde), and narcosis (most aliphatic aldehydes). In most cases the concentrations required to produce such effects are considerably above the irritant threshold for these compounds, and thus it is unlikely that human exposures would approach such levels. However, for certain of these effects, such as those in the central nervous system, more information is needed to determine whether the threshold levels for humans might approach potential occupational exposure conditions.

3.5.3 Confined Space Workplace Concerns

Because they are such strong irritants to the eyes, skin, and mucous membranes, aldehydes can be a major problem in confined space workplaces in which diesel engines are operating. With inadequate ventilation, air concentrations of specific aldehydes could gradually increase until they exceed the permitted occupational exposure limits. Although the total aldehyde concentration provides a general measure of exposure level, there are wide differences in irritant potency of the individual compounds. Formaldehyde, acrolein, and crotonaldehyde are the strongest irritants, the 8 hr TWAs being 1 ppm, 0.1 ppm, and 2 ppm, respectively (Table B-12). For formaldehyde the OSHA 30-min ceiling value is 10 ppm, but both NIOSH and ACGIH propose lowering this to 1 ppm. The short-term exposure limit for acrolein is 0.3 ppm, and that for crotonaldehyde is 6 ppm (ACGIH 1982). In comparison, the concentration ranges in undiluted diesel engine exhaust are 1.8-43 ppm for formaldehyde, 0.2-11.4 ppm for acrolein, and 0.02-0.8 ppm for crotonaldehyde (Table B-12). Thus, unless the air to exhaust gas dilution ratio is at least 50 to 1, the levels of acrolein and formaldehyde would approach the short-term exposure limits, and with inadequate ventilation and limited air volume these limits could be exceeded. However, under such conditions, partial recirculation of the exhaust gases into the engine with the intake air (engine rebreathing) might lead to further oxidation of the aldehydes and reduce the concentrations in the air. Conversely, reductions in oxygen-to-fuel ratios due to engine rebreathing might cause changes in the rates of the delayed ignition reactions such that greater amounts of aldehydes may be released in the exhaust.

If total aldehyde levels in undiluted exhaust are maintained below 10 ppm and ventilation rates are kept at a level to ensure a continuous 50-to-1 dilution of the exhaust gases, the threshold level would not be exceeded for any aldehyde, and monitoring of specific compounds would not be needed. If the total aldehyde level exceeds 10 ppm and/or there is a reduction in ventilation, it would then become increasingly important to monitor levels of formaldehyde and acrolein.

3.6 SULFATES AND SULFURIC ACID

3.6.1 Sources and Formation Chemistry

Sulfates appear in the atmosphere as a result of several natural processes and man-made activities (Kellogg et al. 1972). In the environmental sulfur cycle, biogenically produced hydrogen sulfide (H_2S) is one natural source of atmospheric sulfur. This compound is released by anaerobic microorganisms found in soil, marshes, and tidal flats. The H_2S is oxidized in the atmosphere to sulfur dioxide (SO_2) which, in turn, is oxidized in several steps to sulfuric acid (H_2SO_4). The hygroscopic H_2SO_4 forms into aqueous droplets within which other sulfates may form. In atmospheric samples, the predominant forms of sulfate are H_2SO_4 , NH_4HSO_4 and $(NH_4)_2SO_4$ (Charlson et al. 1974). The sulfuric acid and sulfates are removed from the atmosphere by dry deposition or precipitation. They may then be reduced biologically to reinitiate the sulfur cycle. It has been estimated that atmospheric residence times for sulfur compounds range from 1 to 18 days (Kellogg et al. 1972).

Other processes that contribute to and alter the natural sulfur cycle include releases of H_2S , SO_2 and sulfates from volcanoes, dispersion of magnesium sulfate in wind-generated aerosols from the oceans, dry deposition of SO_2 on solid surfaces and soils, direct uptake of SO_2 by plants, and direct diffusion of SO_2 into and out of water bodies (Kellogg et al. 1972).

To the natural sulfur cycle, man's activities contribute a significant amount of sulfur. Inputs of sulfur are in the form of $CaSO_4$ in gypsum dust and Na_2SO_4 from paper mills, but particularly in the form of SO_2 released during the combustion of fossil fuels. About 95% of the total sulfur entering the atmosphere as a result of man's activities occurs as SO_2 (EPA 1975). Fuels such as No. 2 diesel may contain as much as 1% sulfur (by weight) and, with complete oxidation during combustion, this would yield, under stoichiometric conditions, 600 ppm SO_2 in raw engine exhaust (Vogh 1969). SO_2 output, however, is a function of air-to-fuel ratio (A/F) (Figure 3.5). For a 20-to-1 ratio each 0.1% sulfur in the fuel will yield about 43 ppm SO_2 (Johnson 1975). For various fuel sulfur levels and air-to-fuel ratios the exhaust SO_2 level can be calculated with the following equation (Johnson 1975):

$$(SO_2)_{\text{conc}} = \left(\frac{2}{1+A/F} \right) \left(\frac{\text{Percent sulfur}}{100} \right) \frac{M_{\text{exh}} \times 10^6}{M_{SO_2}}$$

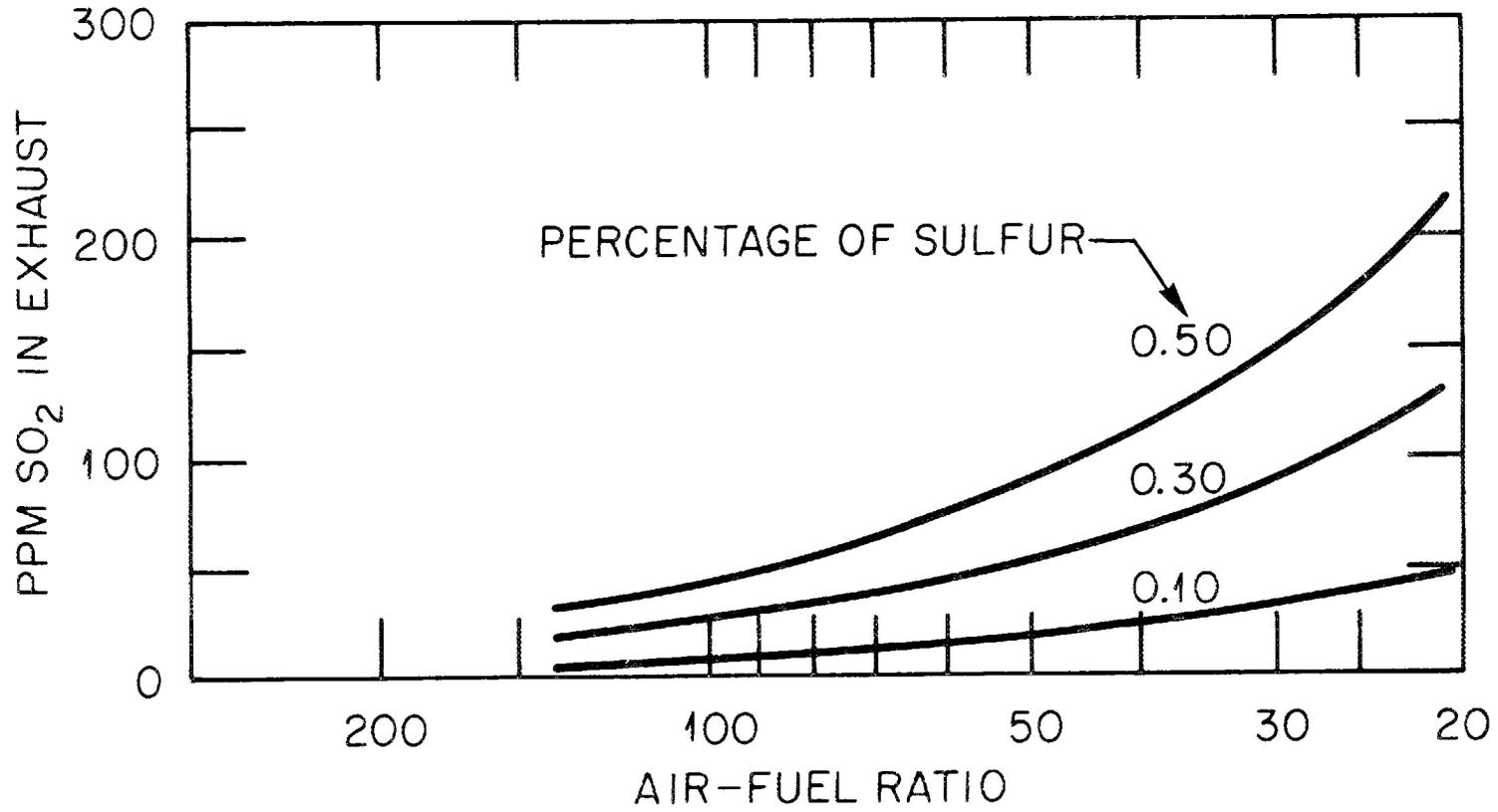


Figure 3.5. Sulfur dioxide concentration in diesel exhaust for fuels with different sulfur content as a function of air-fuel ratio. Adapted from Johnson 1975.

where

$(SO_2)_{conc} = SO_2$ concentration, ppm,

Percent sulfur \equiv percent sulfur by mass in the fuel, percent,

$M_{exh} =$ molecular weight of exhaust products
lbm/lb-mole $\cong 29$,

and

$M_{SO_2} =$ molecular weight of SO_2 , lbm/lb-mole = 64.

Generally, the average sulfur content of No. 2 diesel fuel is about 0.2% (Bureau of Mines, as cited by Springer and Stahman 1977), and, due to varying air-to-fuel ratios over a normal driving cycle, the average concentration of SO_2 in diesel exhaust is about 40 ppm (Levins 1981).

A small portion of the exhaust SO_2 may be further oxidized to SO_3 (see Khatri et al. 1978 for discussion of equilibrium and reaction kinetics). For lean fuel mixtures the maximum SO_3 levels are predicted to be from 1 to 2.5% of the SO_2 levels (Wark and Warner 1976). In the presence of water the SO_3 hydrates to H_2SO_4 . Hydrocarbons and other pollutants associated with motor vehicle exhaust enhance the oxidation processes and allow for a more rapid formation of sulfuric acid (WHO 1979b). Under certain conditions the conversion process may occur at a rate as high as 18% per hour (Rall 1974).

SO_2 in diesel exhaust can be absorbed directly in water droplets or be adsorbed onto suspended particulate matter. In aqueous aerosols, conversion to sulfate proceeds through a sulfite or bisulfite oxidation step (Wood et al. 1975). In the presence of ammonia the reaction proceeds very rapidly due to the formation of ammonium sulfate (McKay 1971).

Adsorbed onto particulate matter, SO_2 is also rapidly oxidized to sulfuric acid, particularly if metal catalysts are present and the relative humidity is high. Various sulfate species may form, depending on the chemical composition of the particulates. Soot, such as that released from diesel engines, is a very effective catalytic agent for the conversion of SO_2 to sulfates (Chang et al. 1979). Sulfuric acid generally accounts for more than 90% of the water-soluble sulfates in diesel exhaust (Truex et al. 1980). The concentration of H_2SO_4 may be as high as 44 mg/m³ but generally averages 0.2-5 mg/m³ (see Table B-6). The chemical characterization of soot particles in terms of sulfate species present has not been thoroughly investigated; however, in diesel exhaust, elements such as Si, Fe, Zn, Al, Ni, Mn, Ca, Ba, Cr, Cu, Ti, and Pb have been found (Frey and Corn 1967; Braddock and Bradow 1975), and one or more of these may be complexed with sulfates in addition to ammonium ions.

Because sulfates can have very different levels of toxicity and chemical reactivity, the evaluation of atmospheric sulfates on the basis

of total water-soluble sulfates may not provide an accurate estimate of a potentially hazardous condition. Identification and quantification of the molecular species present during particular exposure conditions is needed to determine possible adverse health effects.

3.6.2 Current Regulatory Status

Although national air quality standards exist for sulfur dioxide, there are none for sulfuric acid or total atmospheric sulfates. The current U.S. standard for SO₂ is 0.03 ppm (80 µg/m³) annual arithmetic mean and 0.14 ppm (365 µg/m³) 24-hr average not to be exceeded more than once per year (EPA 1971).

Of the fifty states, only four have air quality standards for sulfates (Table B-7). In California the standard is 25 µg/m³ for a 24-hr average. Similarly, in Pennsylvania the 24-hr standard is 30 µg/m³. In addition, Pennsylvania has a 30-day standard of 10 µg/m³. North Dakota has a 12 µg/m³ 24-hr standard which can not be exceeded 1% of the time, and the maximum annual arithmetic mean is set at 4 µg/m³. In Missouri the 24-hr standard for sulfuric acid is 10 µg/m³, which is not to be exceeded more than once in any 90 consecutive days.

For total atmospheric sulfates associated with aerosols or particulates in a workplace environment, neither the Occupational Safety and Health Administration (OSHA) nor the Mine Safety and Health Administration (MSHA) have established minimum standards; however for sulfuric acid OSHA has set 1 mg/m³ as a maximum exposure limit (8-hr TWA) (OSHA 1982).

Several difficulties are involved in setting air quality standards for sulfates. Levels of atmospheric sulfates are not static but are affected by environmental variables such as light, temperature, and humidity, which determine oxidation rates of precursors such as SO₂. The levels of the precursor sulfur oxides, and the presence of catalysts, atmospheric particulates, and ammonia will also influence reaction rates. Thus, sulfate levels at a point source of emissions may be quite low, but may increase significantly with time. Another factor to be considered is that atmospheric sulfates may occur as sulfuric acid, ammonium sulfate, ammonium bisulfate, or any one of a large number of sulfate salts. It is difficult to differentiate these various chemical species analytically, and, because they have varying levels of toxicity and chemical reactivity, a single standard may not necessarily reflect the actual exposure conditions.

Although the OSHA occupational exposure standard for sulfuric acid (1 mg/m³) is applicable to confined space situations, it may not provide an adequate level of protection if ammonia levels are high and most of the sulfate is present as ammonium sulfate and ammonium bisulfate. In the absence of specific standards, attention must be given not only to initial levels of sulfates, but also to SO₂ and ammonia levels and other factors, such as temperature, humidity and particulates, which would enhance sulfate formation.

3.6.3 Monitoring Technology

Two analytical techniques are routinely used for determining total aqueous sulfate levels in atmospheric samples; these are the turbidimetric method and the methylthymol blue method. Of the 28 states whose air quality control agencies analyze atmospheric particulate samples for sulfates, 20 use either one or both of these methods (see Table B-14). In both methods, suspended particulates are collected on filters (Hi Vol samples usually collected over a 24-hr period) and then extracted with water. Barium chloride is mixed into the solution to form a barium sulfate precipitate. In the turbidimetric method, the extent of turbidity of the resulting suspension, as measured spectrophotometrically, is a measure of the total water-soluble sulfates and sulfuric acid. In the methylthymol blue method, the barium remaining in solution is complexed with methylthymol blue, and the remaining uncomplexed methylthymol blue is measured colorimetrically. By knowing the initial amount of barium chloride and methylthymol blue added to the solution, back calculations can be made to determine the original concentration of total water-soluble sulfate. Both methods have been automated and provide for analyses down to a detection limit of about 3 $\mu\text{g}/\text{mL}$. However, both methods, as well as others that rely on filters for the collection of particulate samples, are subject to several potential sources of error including incomplete sampling at certain particle sizes, oxidation of gaseous SO_2 to sulfates on the filter surface, conversion of soluble sulfates to insoluble ones, and the possible neutralization of sulfuric acid by ammonia or other contaminants (Tanner and Newman 1976). The last-mentioned factor would not affect the quantitative results in either the turbidimetric or methylthymol blue method because total sulfates are measured in both methods.

Numerous other methods exist for analyzing for sulfates (see Tanner and Newman 1976 for review). These include gravimetric, titrimetric with Thorin indicator, colorimetric with barium chloranilate, spectrophotometric with barium dye complexes, automatic absorption spectrophotometry, specific-ion electrode analysis, ion chromatography with conductimetric detection, and flash volatilization with flame photometric detection. The latter method reportedly has a 10% precision at concentrations of 0.5 $\mu\text{g}/\text{mL}$ sulfur, can be done in a very short time (5-10 min), and requires only 5 μL or less of sample.

The barium chloranilate method is often used to analyze for total soluble sulfates in automobile and truck exhaust (Tejada et al. 1979). In this method exhaust particulates are collected on a filter. The sulfates are leached out of this sample with a solution of isopropyl alcohol and water. The solution is then passed through ion exchange resins to remove interfering ions and treated with barium chloranilate. The sulfates precipitate out as barium sulfate, and chloranilate ions are released, the concentration of which is determined by ultraviolet spectrophotometry. The method can measure trace quantities of sulfates (0.01-0.1 μg) in sample volume of 1 mL. However, nitrate ions and organics will interfere with the accuracy of the result.

Various methods have been proposed and tested for separating and identifying sulfuric acid and the various sulfate compounds in aerosol samples. Included among these are: selective thermal volatilization; solvent extraction with isopropanol, benzaldehyde, or other organic solvents, and gas phase ammonia titration (see Tanner and Newman 1976 for review). Infrared spectroscopy, colorimetry of derived bis-(diethyldithiocarbamate)-copper (II) complex and electron microscopy are other methods that have been tested, but are reported to be less selective (Tanner and Newman 1976). X-ray photoelectron spectroscopy has been used to identify the various sulfur oxidation states in ambient aerosols, but this technique does not identify the associated cations (Craig et al. 1974). However, when coupled with solvent extraction procedures that have the potential for separating sulfuric acid, ammonium sulfate and bisulfate, and metal sulfates and bisulfates, a thorough characterization of aerosol sulfate species might be attainable.

3.6.4 Health Effects

Because this report is concerned with the potential health effects of diesel exhaust in confined space conditions, major emphasis will be placed on experimental and epidemiological data that deal primarily with inhalation exposures. Of the inorganic sulfate compounds that are present in diesel exhaust, sulfuric acid, ammonium sulfate, and ammonium bisulfate are the three major components, and, consequently, the toxicology of these three compounds is of greatest significance. In evaluating toxicological effects, it should be kept in mind that these compounds are associated with engine exhaust particulate matter, and the size, shape, density, and chemical composition of the particulates may have a substantial effect on the rate of uptake, respirability, and effects of the sulfur compounds. In contrast, most laboratory studies have tested relatively pure liquid aerosols, whose toxicological effects may not be identical to those of the particulates. A final qualification that must be made in evaluating the available health effects data concerns the difficulties in monitoring sulfuric acid and sulfate levels during inhalation exposures. Ammonia released in expired air will neutralize part or all of the sulfuric acid. Even during analytical procedures to determine sulfuric acid levels, some neutralization will occur, particularly if the aerosol samples are collected on filters. Any interpretation of the results of a particular study must take these factors into consideration. The health effects discussed in the following sections are summarized in Table B-15.

3.6.4.1 Lethality

Mists of sulfuric acid have been found to be moderately to highly toxic to laboratory animals. Treon et al. (1950) reported that the lowest dose of inhaled sulfuric acid (particle size $< 2 \mu\text{m}$) that was lethal to some test animals was 178 ppm to rats exposed for 7 hr, 140 ppm to mice exposed for 3.5 hr, and 22 ppm to guinea pigs exposed for 2.75 hr. Similarly, Amdur et al. (1952a) found that the lethal inhalation dose to guinea pigs was 18-60 mg/m³ for an 8-hr exposure. Particles of 2.7 μm were more toxic than particles of 0.8 μm . Wolff et al. (1979) also exposed guinea pigs to two different aerosol sizes (0.4 and

0.8 μm). For the larger size particles the LC50 was calculated to be 30 mg/m^3 . With the smaller sized particles the LC50 level was not reached even at a concentration as high as 109 mg/m^3 .

3.6.4.2 Mutagenicity

The only information that was found concerning the potential mutagenicity of inorganic sulfates was a single microbial study involving ammonium sulfate (Litton Bionetics, Inc. 1975). In this study the D4 strain of the yeast Saccharomyces cerevisiae, and the TA-1535, TA-1537, and TA-1538 strains of the bacterium Salmonella typhimurium were exposed to ammonium sulfate concentrations of 2.5 to 10%. The assays were conducted with and without mammalian metabolic activation preparations. Both positive and solvent controls were included in the tests. Under all tested conditions the ammonium sulfate exhibited no mutagenic activity.

3.6.4.3 Carcinogenicity

The National Cancer Institute has issued a series of reports in which literature-derived carcinogenicity data for various chemicals has been tabulated (NCI 1951-1978). Included in the listing is information on sulfuric acid and ammonium, barium, beryllium, cadmium, cobalt, copper, iron, nickel, sodium, zinc, and magnesium sulfates. The available evidence indicates that sulfuric acid and ammonium sulfate are not carcinogenic (Alarie et al. 1973). Several of the metallic sulfates when injected subcutaneously produced local sarcomas at the injection site; but only one, beryllium sulfate, was reported to cause pulmonary cancers as a result of inhalation exposures (Schepers et al. 1957; Reeves et al. 1967). However, beryllium is not among the metals reported to occur in diesel exhaust particulates (see Frey and Corn 1967), and therefore, beryllium sulfate is not likely to be a carcinogenic hazard in confined space situations.

3.6.4.4 Teratogenicity and Embryotoxicity

The teratogenicity and embryotoxicity of sulfuric acid and inorganic sulfate compounds has been evaluated in only a few laboratory studies. Murray et al. (1979) exposed CF-1 mice and New Zealand white rabbits to 0, 5.7 ± 1.2 , or 19.3 ± 4.9 mg/m^3 of sulfuric acid (median particle diameter 1.6 μm at 5.7 mg/m^3 and 2.4 μm at 19.3 mg/m^3) for 7 hr/day during the period of major organogenesis. The exposures had no adverse effect on litter size, live fetuses per litter, resorptions per litter, fetal sex ratio, or fetal body size. The offspring of the exposed mice did not have a higher incidence of malformations than controls; however, at the highest exposure, two fetuses were conjoined in the upper body and both had cranial malformations. In rabbits no significant increase in fetal malformations was observed, although there was an increased incidence of small nonossified areas of the skull.

No studies were found in which the teratogenicity of ammonium sulfate and ammonium bisulfate was evaluated. However, some metallic sulfates may be teratogenic, and this is most likely due to the action of

the cation rather than to the sulfate moiety. Cadmium and lead salts are known to cause malformations in hamsters (Fern and Carpenter 1967a,b). No information was found to suggest that levels of metallic sulfates were sufficiently high in diesel exhaust to be a teratogenic health hazard.

3.6.4.5 Respiratory and Pulmonary Effects

Sulfuric acid and the inorganic sulfates that are present in diesel exhaust are respiratory and pulmonary irritants. Irritant potency is dependent not only on the chemical species present and their concentrations, but also on the size of the aerosol or particulate with which they are associated and on the susceptibility of the exposed species or individuals.

In tests in guinea pigs, Amdur and coworkers found that the degree of irritation, as measured by the percentage increase in airway resistance, was greatest for sulfuric acid, followed in decreasing order by zinc ammonium sulfate, ferric sulfate, zinc sulfate, ammonium sulfate, ammonium bisulfate, cupric sulfate, ferrous sulfate, sodium sulfate, and manganese sulfate (Amdur and Corn 1963; Amdur and Underhill 1968; Amdur et al. 1978b). In these studies sulfuric acid was ten times greater an irritant than ammonium sulfate and ammonium bisulfate.

Several other studies have compared the effects of various sulfates on pulmonary function. In tests on healthy human volunteers, Morrow et al. (1979) found that sulfuric acid (1 mg/m^3 , $0.5\text{--}1 \text{ }\mu\text{m}$) produced a greater decrease in specific airway conductance than did equivalent concentrations of ammonium bisulfate (NH_4HSO_4), sodium bisulfate (NaHSO_4), and ammonium sulfate ($\text{NH}_4)_2\text{SO}_4$). Furthermore, the bronchoconstrictor action of carbachol was potentiated to a greater degree by H_2SO_4 than by the other compounds.

Avol et al. (1979) exposed both healthy and asthmatic men for 2 hr/day for 1, 2, or 3 days to aerosols of ammonium sulfate ($104\text{--}337 \text{ }\mu\text{g/m}^3$) ammonium bisulfate ($4\text{--}47 \text{ }\mu\text{g/m}^3$) or sulfuric acid ($93\text{--}111 \text{ }\mu\text{g/m}^3$) of mass median aerodynamic diameter of $0.28\text{--}0.62 \text{ }\mu\text{m}$. Although the exposure levels were not sufficiently high to produce adverse pulmonary effects, symptoms of respiratory distress were more frequent in exposures to sulfuric acid than to the other two compounds. This was particularly true for asthmatics.

The effects of sulfuric acid and ammonium sulfate on pulmonary function in donkeys were evaluated by Schlesinger et al. (1978). Although neither chemical had any adverse effect on pulmonary resistance, compliance, or regional deposition, 1-hr exposures to $194\text{--}1364 \text{ }\mu\text{g/m}^3$ of H_2SO_4 ($0.3\text{--}0.6 \text{ }\mu\text{m}$) reduced tracheobronchial mucociliary clearance. Ammonium sulfate, at concentrations up to $2000 \text{ }\mu\text{g/m}^3$, had no such effect. Tracheal mucus velocity was not altered in sheep exposed for 4 hr to $1\text{--}1.7 \text{ mg/m}^3$ of submicron aerosols of eleven different sulfate compounds (ammonium sulfate and bisulfate, zinc ammonium sulfate, and zinc, ferrous, ferric, copper, nickel, manganese, sodium and aluminum sulfates) (Sackner et al. 1981). These same compounds when tested in dogs

at aerosol concentrations up to 17.3 mg/m³ for 7.5 min or 4.1-8.8 mg/m³ for 4 hr had little effect on pulmonary mechanics, nor did 1 mg intravenous injections; however a 100-mg intravenous dose of copper sulfate did cause a decrease in specific total respiratory conductance as well as adverse cardiac and hematological effects (Sackner et al. 1981).

The effects of sulfuric acid and aluminum sulfate on the pulmonary function of rats have been compared by Lewkowski et al. (1979). Exposures were continuous for 7 and 8 weeks. Sulfuric acid levels were 4.05 mg/m³ (0.5 μm) and 2.49 mg/m³ (<0.24 μm), and aluminum sulfate levels were 2.04 mg/m³ (2.0 μm) and 2.59 mg/m³ (1.4 μm). Both compounds increased pulmonary resistance and respiratory frequency of adult rats. In juvenile rats aluminum sulfate increased respiratory frequency, functional residual capacity, and static deflation volume. Overall, aluminum sulfate was more detrimental to pulmonary function than was sulfuric acid. Considering that aluminum is known to occur in diesel exhaust particulates, the possibility exists that aluminum sulfate might also be present. Whether it occurs in sufficient quantities to be a potential health hazard requires further investigation.

Except for aluminum sulfate, the other inorganic sulfates tested have been shown to be weaker pulmonary irritants than sulfuric acid. Consequently, any exposure standards for total sulfates, if based on sulfuric acid equivalents, should provide the greatest margin of safety.

Because of the known toxicity of sulfuric acid, many laboratory studies have been carried out in order to better define safe exposure limits. Animal studies have shown that species can vary considerably in their pulmonary susceptibility to aerosols of this compound. Treon et al. (1950) exposed four species of rodents to sulfuric acid aerosols (<2 μm) for up to 7 hr and found that the lowest lethal dose was 22.1 ppm for guinea pigs, 140 ppm for mice, 178 ppm for rats, and 374 ppm for rabbits. Schwartz et al. (1977) also found that guinea pigs were very sensitive to H₂SO₄ exposures. Damage to the respiratory system (bronchial and alveolar lesions) occurred in this species during short-term exposure (4 day) to >7 mg/m³, however, respiratory lesions (in the trachea and larynx) occurred in mice only at 125 mg/m³. Concentrations as high as 172 mg/m³ in rats and 500 mg/m³ in monkeys did not cause any pulmonary damage.

Pulmonary effects of H₂SO₄ also vary with the mass concentration of the test aerosol, but the effects have not always been identical from one study to another. In studies on guinea pigs Amdur et al. (1978a) found that pulmonary flow resistance increased and pulmonary compliance decreased at concentrations as low as 100 μg/m³ (1 hr exposure). Silbaugh et al. (1981a) reported similar changes in guinea pigs at H₂SO₄ levels of 14.6 mg/m³ and higher, but none at 1.2 mg/m³. Brownstein (1980) observed that after 4 hr exposure to 32.6 mg/m³ the lungs of guinea pigs exhibited bronchiolar epithelial desquamation.

Most studies on humans have shown that sulfuric acid aerosol mass concentrations as high as 1 mg/m³ for 2 hr will not result in any adverse pulmonary effects (Sackner et al. 1978; Horvath et al. 1982;

Horstman et al. 1982; Kerr et al. 1981); however, in one study a 1 mg/m³ aerosol of 0.5-1- μ m particles was reported to cause small but significant changes in maximum expiratory flow volume at 60% total lung capacity and significant changes in partial expiratory flow volume at 40% and 60% total lung capacity (Morrow et al. 1979). In addition, one study reported transitory alterations in mucociliary clearance at 100 μ g/m³ (Leikauf et al. 1981), another reported throat and respiratory irritation at 233 μ g/m³ (Horvath et al. 1982), and in a third a 5- to 15-min exposure to only 350 μ g/m³ resulted in shallow rapid breathing (Amdur et al. 1952b).

Concentrations above 1 mg/m³ may produce respiratory distress. A deep breath of a 5 mg/m³ aerosol produced coughing in healthy human volunteers (Amdur et al. 1952b) and 10- to 60-min exposures to 2.9-39 mg/m³ (mean values not given) produced coughing, bronchoconstriction, rales, lacrimation, and rhinorrhea (Sim and Pattle 1957).

The pulmonary effects of sulfuric acid are also known to vary with the size of the particles with which they are associated. In tests on guinea pigs, Amdur (1969) found that pulmonary flow resistance induced by H₂SO₄ decreased with increasing particle size. Particles of 0.1 μ m had an effect about 5.5 times greater than those of 2.5 μ m. A 6-mg/m³ level of 2.5- μ m particles was required to produce a 50% increase in pulmonary flow resistance, but a concentration of only 1.9 mg/m³ of 0.8- μ m particles was needed to cause the same effect. In a later study (Amdur et al. 1978a) 0.3- μ m particles were found to be more irritant than 1- μ m particles for mass concentrations of 0.1-1 mg/m³. Thomas et al. (1958) reported that 0.9- μ m particles were more active toxicologically to guinea pigs than those of either 0.6 μ m or 4.0 μ m when the exposure concentration was in the range of 1-4 mg/m³. The 8-hr LC₅₀ value to guinea pigs was found by Wolff et al. (1979) to decrease from 109 mg/m³ for 0.4- μ m particles to 30 mg/m³ for 0.8- μ m particles. In addition, Pattle et al. (1956) found that 2.7- μ m particles were more toxic to guinea pigs (LC₅₀ 6.8 ppm) than those of 0.8 μ m (LC₅₀ 14.9 ppm). In tests on humans, Sim and Pattle (1957) found that a dry aerosol (RH 62%) containing 0.99- μ m particles at a concentration of 39.4 mg/m³ caused less severe respiratory and pulmonary distress than a wet aerosol (RH 91%) containing 1.54- μ m particles at a concentration of 20.8 mg/m³.

The differing results given in these studies might be accounted for by the fact that pulmonary (alveolar) deposition of particles is not linearly correlated to particle size. Maximum deposition occurs at particle sizes below 0.1 μ m. Deposition decreases with increasing particle size up to about 0.5 μ m, slowly increases to a secondary maximum at 2.0 μ m, and then rapidly decreases at larger sizes (Task Group on Lung Dynamics 1966) (see Figure 3.3). Furthermore, the particle size of the sulfuric acid aerosol is affected by relative humidity (Cavender et al. 1977). Because sulfuric acid is hygroscopic it will rapidly equilibrate with the relative humidity of the environment and then will reequilibrate after entering the respiratory tract, where the relative humidity is 98% (Cavender et al. 1977). Under relatively dry external environmental conditions (5% relative humidity) sulfuric acid particles may increase as much as threefold in size after entering the respiratory

tract. Changes in particle size will also result in changes in the molarity of the sulfuric acid in the particles. At a constant temperature of 20°C, the molarity will decrease with an increase in relative humidity (about 9 at 20%, 5 at 60%, and 1 at 98%) (Cavender et al. 1977). Thus, conditions which favor the formation of aerosols of particle size of about 2.0 μm in the lungs will result in greater alveolar deposition and may have a very different physiological effect than slightly smaller or larger particles which would be deposited in the upper airways. Inhalation of very small H_2SO_4 particles ($\leq 0.1 \mu\text{m}$) under conditions of high humidity would result in the highest alveolar deposition rate because there would be little change in particle size in the lungs.

In contrast to the numerous studies conducted on the pulmonary effects of short-term H_2SO_4 exposures, little work has been done on the effects of chronic exposures. Alarie et al. (1973) have reported that monkeys exposed for 78 weeks to 2.43 mg/m^3 (3.60 μm particle size) or 4.79 mg/m^3 (0.73 μm) suffered lung damage and reduced pulmonary function. There were no significant deleterious effects at lower concentrations. Guinea pigs exposed to 0.08–0.1 mg/m^3 (0.84 and 2.78 μm) for 52 weeks showed no changes in pulmonary structure or function. Higher concentrations were not tested.

Lewis et al. (1973) exposed beagle dogs to 0.89 mg/m^3 of H_2SO_4 aerosol (90% of particles less than 0.5 μm) for 21 hr/day for 620 days (RH 43–50%, temp. 73–76°F), and found a significant reduction in total lung weight; in average pulmonary single breath carbon monoxide diffusion capacity; lung residual volume; net lung volume when inflated; and in the difference between inflated and deflated net lung volumes. The chronic exposures also resulted in a significant increase in total expiratory resistance. The authors concluded that these changes were indicative of the development of obstructive pulmonary defects.

For a H_2SO_4 mass concentration of 1 mg/m^3 similar to that used in the studies of Lewis et al., Bushtueva (1957) found that a 65-day exposure to rats resulted in histopathological changes in the lungs. Ninety percent of the aerosol particles were less than 2.0 μm .

These few studies indicate that chronic exposure to H_2SO_4 at levels near the recognized TLV (1 mg/m^3) can result in alterations in pulmonary structure and function. However, these effects were produced under conditions of continuous or near continuous exposure. For intermittent occupational exposures it would be expected that the minimum effective mass concentrations would be much higher.

3.6.4.6 Susceptibility to Infection

As respiratory irritants, sulfuric acid and other inorganic sulfates have the potential for altering the defense mechanisms of the respiratory tract (mucociliary clearance and alveolar macrophage function) to such a degree that the organism becomes more susceptible to microbial infections. In one study on humans, transitory alterations in mucociliary clearance were seen following exposure to H_2SO_4 of only 100

$\mu\text{g}/\text{m}^3$ (Leikauf et al. 1981). In mice, interferon production (an indicator of cell function and resistance to infection) of alveolar macrophage cultures was reduced, but that of tracheal organ cultures was unaffected following 10- to 14-day exposures to 125-154 mg/m^3 (Schwartz et al. 1979).

In a study on mice Gardner et al. (1977) found that 900 $\mu\text{g H}_2\text{SO}_4/\text{m}^3$ for 3 hr did not increase the susceptibility of the animals to infection by Streptococcus pyogenes (evaluated in terms of changes in mortality rates); however, when the exposures were preceded by exposure to ozone, there was a significant increase in infections. Rates of infection in mice can also be increased by concurrent exposure to H_2SO_4 and particulates (Fenters et al. 1979). Thus, it would seem that at sufficiently high concentrations, sulfuric acid alone might reduce respiratory resistance to infections, but further study is needed to verify this assumption. Various inorganic sulfates are also known to cause an increased susceptibility to infections in laboratory animals (Ehrlich 1980). In these cases, it appears that metallic cations (Cd, Cu, and Zn) are the primary causative agents. The presence of ammonium (as in zinc ammonium sulfate) somewhat neutralizes the effect.

3.6.5 Confined Space Workplace Concerns

As noted in Section 3.6.4, from a toxicological standpoint sulfuric acid is the most hazardous sulfur compound occurring in diesel exhaust, and it represents more than 90% of the total water-soluble sulfates present (Truex et al. 1980). It is a primary irritant to the respiratory and pulmonary systems, and because of this maximum occupational exposure limits have been set at 1 mg/m^3 . Whether H_2SO_4 levels in diesel exhaust pose a health hazard to workers in confined spaces is dependent on several factors: (1) the sulfur content in the diesel fuel; (2) the rate of oxidation of the fuel sulfur to SO_2 ; (3) the rate of conversion of the SO_2 to sulfuric acid; (4) the amount of mixing of the exhaust with fresh air (ventilation rate); (5) the air volume of the confined space in which the exhaust is vented; and (6) the length of time the engine is operated in the confined space. As noted in Section 3.6.1, the higher the fuel sulfur content and the greater the load on the engine (as reflected in a lower air-to-fuel ratio) the higher the concentration of exhaust SO_2 (Figure 3.4). Although sulfate formation, expressed in terms of percent conversion of fuel sulfur, may actually decrease with increasing fuel sulfur level (Truex et al. 1980), mass emission rates of SO_4 generally increase with increases in fuel sulfur. Frisch et al. (1979) reported sulfate levels of 0.9-2.6 mg/m^3 in the undiluted exhaust of diesel engines using fuel with 0.11% sulfur, 0.9-3.7 mg/m^3 for fuel with 0.27% sulfur, and 4.0-8.1 mg/m^3 for fuel with 0.44% sulfur. These same authors found that for the 0.27% sulfur fuel, sulfate emissions were 0.94-2.54 mg/m^3 when the engine was operating at 25% load and 2.44-3.72 mg/m^3 when the engine was operating at 75% load. In the latter case the sulfate level increased when the exhaust was diluted with air at a ratio of 8 to 1 and 25 to 1 but decreased at a 50 to 1 dilution. With a 25% load, there was an overall decrease in sulfates with increasing dilution ratio, such that there was a 50% decrease in sulfate levels at a 50 to 1 dilution ratio. This general trend was

attributed to the reduction in particulate levels and subsequent reduction in catalytic interaction between SO₂ and the particulates to form sulfates.

Because sulfuric acid concentrations in undiluted diesel exhaust may be at or above the current OSHA exposure limit of 1 mg/m³, worker exposure in confined space situations would depend considerably on ventilation and dilution factors. For a H₂SO₄ exhaust level of 5 mg/m³ a minimum fivefold dilution of the exhaust would be needed to reduce the concentration to the TLV. The ventilation rate corresponding to this level of dilution can be calculated from the following equation:

$$Q = V \cdot \frac{C}{Y}$$

where Q = the required amount of air (m³/min),
V = volume of the exhaust gas at full load and rated speed (m³/min),
C = concentration of sulfuric acid (ppm), and
Y = exposure limit (ppm).

Thus for a H₂SO₄ concentration of 5 mg/m³ (1.2 ppm), a TLV of 1 mg/m³ (0.24 ppm), and, a V of 0.07 m³/min per rated horsepower [which is an approximate value for a moderate size engine at full load (ILO 1983)], then Q will equal 0.35 m³/min (12.4 cfm) per rated horsepower. In the absence of a ventilation system, the buildup of sulfuric acid in a confined space will vary with the mass emission rate from the engine and the total enclosed air volume. For an emission rate of 9-33 mg/bhp·hr (Frisch et al. 1979) and an enclosed volume of 10,000 ft³ (280 m³), and assuming complete mixing, the H₂SO₄ level would be 0.008 mg/m³ to 0.03 mg/m³ per rated horsepower after 15 minutes of operation. The mass emission rate given here was calculated for a heavy duty diesel (rated brake power, 210 hp) operated at 25% and 75% full power (Frisch et al. 1979), but it illustrates the fact that in the absence of an adequate ventilation system, sulfuric acid levels in an enclosed space in which a diesel engine is operating could rapidly reach the occupational exposure limit.

3.7 ODOR, NOISE, AND SMOKE EMISSIONS

3.7.1 Diesel Exhaust Odor

Diesel exhaust odor is highly noticeable and can be very annoying, but it is not yet classified as an air pollutant. In confined space workplaces the intensity of the odor may result in less than satisfactory working conditions.

Aldehydes have traditionally been considered to be the main species responsible for diesel exhaust odor (Ricardo and Glyde 1941). Doubt has been cast on this by more recent studies. Linnell and Scott (1962a) determined the odor thresholds for diluted diesel exhaust and its major components and found that formaldehyde and acrolein did not contribute

significantly to exhaust odor, but that NO₂ might, especially under full load conditions. Hydrocarbons were considered to be the most likely contributors to exhaust odor. To reduce odors to threshold levels, a dilution factor of 140 to 475 was needed.

Reckner et al. (1965) extracted the odor components of diesel exhaust by bubbling the gas through a 5% aqueous solution of sodium bicarbonate. Subsequent extractions led to a tentative identification of the odorous compounds as alcohols, hydroxy ketones, and weakly polar acids.

Using a panel of persons performing odor assessments of raw and modified diesel exhaust and synthetic blends representing portions of diesel exhaust, Vogh (1969) also found that low molecular weight aldehydes contributed little to the diesel exhaust odor. Sulfur and nitrogen oxides were also examined as odorants, but of these only NO₂ appeared to be a potential odor contributor. The potential for a high number of odorous compounds in diesel exhaust was nonetheless recognized, and it was suggested that hydrocarbons and oxygenates other than carbonyls might be the major contributors.

The odor profile of diesel exhaust has been described by workers at Arthur D. Little (see Spindt et al. 1971, Levins 1981 for review) as having an oily-kerosene component and a smoky-burnt component. Because of the subjective nature of human odor panel estimations, A. D. Little has developed a chromatographic Diesel Odor Analysis System (DOAS) for predicting and describing diesel odor. The system is qualitative and semiquantitative and provides two main results, one being an indication of the oxygenate fraction and the other of aromatics, as these two fractions include the major odorants.

The DOAS readings are correlated with an odor intensity scale called Total Intensity of Aroma (TIA). The TIA is a 7-step scale (0-3) with 0 indicating no odor, 1½ a slight to moderate odor and 3 a strong odor. Levins (1981) emphasizes that as is the case for most sensory responses, perceived odor intensity is an exponential function of odorant concentration. Thus a factor of 10 reduction in odorant concentration would only give rise to a 1 TIA (total intensity of aroma) unit reduction.

Prior to the establishment of the DOAS, diesel odor research was based on subjective comparative analyses using a series of reference odors and a 12-step odor intensity scale (D units). Studies cited by Levins (1981) indicate a fairly good correlation between the two scales:

$$TIA = 0.20 + 32 D \quad (r^2 = 0.90)$$

The odor profile of a typical diesel exhaust, as determined by the DOAS, would have a TIA of 2, with the oily-kerosene component having a TIA of 1½ and the smoky-burnt component a TIA of 2. Thus, the smoky burnt component, which is the liquid chromatographic oxygenate (LCO) fraction, accounts for most of the diesel exhaust odor. This can be expressed mathematically (Levins 1981) as:

$$\text{TIA} = 1.0 + 1.0 \log \text{LCO} \quad (r^2 = 0.996)$$

with LCO being expressed in mg/m^3 . According to Levins (1981), the total LCO concentration in diesel exhaust averages $5 \text{ mg}/\text{m}^3$ with individual compounds at 0.1 to $10 \text{ } \mu\text{g}/\text{m}^3$. Concentrations will vary with engine and fuel conditions and can vary by a factor of 1 TIA unit between engine models.

Henein (1973) cites work showing that mono- and polyoxygenated partial oxidation products and certain fuel fractions are largely responsible for odor. Alkyl benzenes, alkyl naphthalenes, indenenes, tetralins, and indanes are responsible for the oily-kerosene part of the odor. A number of oxygenated aromatic structures are related to the smoky-burnt odor. Sulfur species may contribute their own type of odor. It was found that odor intensity does not vary appreciably with engine speed, increases with load, and decreases with improvements to the injection system. Catalytic reactors were highly effective in reducing odor. A consideration in enclosed spaces is that engine rebreathing of exhaust and impediments to good aspiration cause increased odor. It may be noted that odor components may be present at very low concentrations (parts per trillion) and still give noticeable odor (Arthur D. Little, Inc. 1969).

In a study done by Lawter and Kendall (1977), personal-vicinity and exhaust samples of aldehydes and other oxygenates suggest that mine personnel are, on average, exposed to diesel exhaust diluted about 100-fold. From the chromatographic oxygenate concentrations (1.5 to $1.9 \text{ mg}/\text{m}^3$), an average diesel odor intensity of 3 (strong - on a scale of 0-7) would be predicted but was not observed. There was evidence that some odor components were associated with particles and therefore did not contribute to the perceived odor. While frequent exposure to odor intensity levels of 2-3 could constitute a significant annoyance to the miners, the reactions observed were more those of recognition than annoyance, suggesting that the levels were in fact fairly low.

Tables 3.2 and 3.3, taken from Spindt et al. (1971), and based on the results of the Arthur D. Little studies, show some compounds of diesel exhaust and the odor they are responsible for. Knowing the composition of odor components will provide a basis for reduction of odor in the exhaust.

TABLE 3.2. CHEMICAL STRUCTURE VERSUS ODOR
FOR THE OILY-KEROSENE ODOR COMPONENTS^a

Structure	Odor Type
Alkyl benzenes	Oily
Alkyl indenenes	
Alkyl indans	Kerosene
Alkyl tetralins	
Methyl naphthalenes	Sensation (feel or irritation)

^aAdapted from Spindt et al. 1971.

TABLE 3.3. TENTATIVE SMOKY-BURNT ODOR COMPONENTS^a

Structure Class ^b	C Range	Odor Description
Alkenone	C5-C11	Oxidized oily
Furan	C6-C10	Irritation, pungency
Dieneone	C9-C12	Sour, oxidized oily
Furfural	C6-C7	Burnt, oily
Methoxy benzene ^c	C8-C9	Smoky, pungency
Phenol ^c	C7-C12	Burnt, smoky, particle size, pungency
Benzaldehyde ^c	C7-C10	Burnt, smoky, metallic, pungency
Benzofuran	C8-C9	Particle size, smoky
Indanone ^c	C9-C13	Metallic, smoky, sour
Indenone	C9-C10	Linseed-oily, sour
Naphthol ^c	C10-C14	Smoky, burnt
Naphthaldehyde	C11	Smoky

^aAdapted from Spindt et al. 1971.

^bIncludes hydroxy and methoxy derivatives.

^cMost abundant classes.

Actual health effects from odor components are ill defined, due to the fact that the odor components are present in only low concentrations. Potential health effects of diesel exhaust hydrocarbons are discussed in Section 3.3.

Diesel exhaust odor can be minimized by reducing hydrocarbon emissions. This can be accomplished through engine design (reduction in fuel nozzle diameter and nozzle sac volume or the use of a heat-insulated element as part of the precombustion chamber) or through engine operating parameters (change in the rate and timing of fuel injection). In addition the use of fuels with lower aromatic content may also reduce odor components in the exhaust.

3.7.2 Diesel Engine Noise

In enclosed areas where diesel equipment is being used, engine noise can be a potential health problem leading to ear damage and possible hearing loss. Diesel engine noise levels in underground mines have in some instances exceeded the federal standard of 90 dBA (8-hr TWA) (Sutton 1975). NIOSH (1982) recommends a TWA of 85 dBA and a ceiling value of 115 dBA. As noted by Daugherty et al. (1983), the Army maximum noise limit is 85 dB. Daugherty et al. (1983) also report that diesel engines of the size range being considered for material hauling equipment (35-80bhp) produce noise levels of 95 to 105 dB. Therefore, unless noise levels are reduced by insulating the engines, hearing protection would have to be provided to personnel operating the equipment in a confined space.

Stokinger (1975) points out that high noise levels enhance the effects of ototoxic chemicals. However, there is no evidence that such a synergistic effect occurs with diesel noise and exhaust components.

3.7.3 Diesel Exhaust Smoke

Smoke emissions from a typical diesel engine range from 1 to 10 mg/ft³ (0.05-0.3 g/bhp·hr) (Johnson 1975). Exhaust smoke becomes visible when the concentration reaches about 5 mg/ft³. During cold start-ups a bluish-white haze, consisting of unburned and partially burned fuel molecules, lubricating oil, and condensed water vapor, is emitted in the exhaust. With increasing load this is replaced by a whitish-grey smoke having a larger fraction of carbon particles. At or near maximum load, or during rapid acceleration, the exhaust becomes black and opaque to light. This soot, which consists of a large amount of elemental carbon as well as adsorbed organic compounds, results from the incomplete combustion of the fuel due to the high fuel-to-air ratios and lengthening of the fuel injection period (see Henein 1973 for review). In areas of the combustion chamber where there is insufficient oxygen, pyrolysis of the fuel molecules takes place, resulting in the formation of acetylene and hydrogen. The simultaneous condensation and dehydrogenation of acetylene yields particles of elemental carbon, and partial oxidation of the carbon causes an increased amount of carbon monoxide to occur in the exhaust.

Smoke measurements for mobile diesel vehicles are usually made according to the EPA Federal Test Procedures (see Johnson 1975 for review). The test cycle, originally designed for heavy duty diesels, consists of two acceleration periods (one of 5 sec and one of 10 sec) and a 35-sec lugdown period. During the test, smoke opacity is measured continuously with a light-extinction meter. The smoke meter is calibrated such that 0% opacity is indicated for no-smoke levels and 100% opacity when no light penetrates the smoke plume (at a 90° angle to the direction of flow).

Johnson (1975) presents federal smoke test cycle data for a typical turbocharged and naturally aspirated (NA) diesel engine. For the acceleration modes, the turbocharged engine gave higher peak smoke opacity readings (40-70%) compared to the NA engine (10-25%); however, for the lugdown mode, smoke emissions were lower for the turbocharged engine (about 5% opacity compared with 10-15% opacity). For comparative purposes the 15 highest half-second values in the acceleration mode and the 5 highest in the lugging mode are averaged to give an "acceleration smoke value" and a "lugging smoke value". In an example given by Johnson (1975), these averages ranged from 3 to 25% for acceleration and 2.2 to 18.5% for lugdown.

In confined spaces of limited air volume and ventilation, smoke emissions from diesel engines may be an important factor in affecting working conditions. Reduced visibility and worker discomfort may increase the risk of accidents and injuries. Although long-term health effects resulting from occupational exposure to diesel smoke have not been clearly established, there is the potential for adverse pulmonary effects (see Section 3.4).

In view of these considerations, efforts should be made to reduce smoke emissions from diesel engines used in confined space to the lowest practical level. Since soot formation occurs primarily at maximum power output, derating an engine and limiting its maximum fuel-to-air ratio would contribute significantly to lowering excess smoke emissions (Hurn 1975). Smoke emissions can also be adjusted by altering the rate and timing of fuel injection (Henein 1973). In addition, using a fuel of lower cetane number, lower density, and reduced aromatic content will also result in lower smoke emissions.

There is at present insufficient data concerning smoke emissions of the diesel engines being considered for use in Army material handling equipment to determine what, if any, control measures must be taken to reduce smoke level under expected operating conditions. Field tests under actual working conditions would provide valuable information in this regard.

3.8 INTERACTIVE HEALTH EFFECTS OF DIESEL EXHAUST COMPONENTS

The physiological and toxicological effects of diesel emissions can be enhanced or altered as a result of the combined actions or interactions of the various exhaust components. Thus, NO₂, SO₂, sulfuric acid, inorganic sulfates, and aliphatic aldehydes such as formaldehyde and

acrolein are all primary irritants to the eyes, mucous membranes, and respiratory tract (EHA 1978). Exposure to any one of the compounds, at sufficiently high concentrations, would result in burning and itching sensations in the eyes, nose, and throat and may produce wheezing and coughing. When these irritants are present together, as in a diesel exhaust, their combined effect could produce a threshold response at concentrations below the TLVs for the individual compounds. Several of these exhaust components, including SO₂, sulfuric acid, and formaldehyde are bronchoconstrictors and cause a decrease in pulmonary flow resistance. There is evidence that nitrogen dioxide can produce a similar effect.

At high concentrations both NO₂ and formaldehyde can cause pulmonary edema and other pathological changes in the lung tissues, and acute exposures to NO₂ or SO₂ can lead to bronchiolitis obliterans.

Because of potential damage to lung epithelial tissues, many diesel exhaust gases can have similar adverse effects on pulmonary mucociliary clearance rates. Diesel particulates and sulfuric acid aerosols, as well as SO₂ and NO₂, decrease mucociliary clearance, and the long-term result may be a reduced resistance to bacterial infection (WHO 1979b). Campbell et al. (1980) report that the infectivity-enhancing effect of diesel exhaust could be attributed to both NO₂ and acrolein.

Carbon monoxide and nitric oxide have similar modes of toxicological activity in that both reduce the oxygen-carrying capacity of hemoglobin. Consequently, these two gases would have additive effects in producing hypoxemia in exposed individuals.

Carbon dioxide acts as a respiratory stimulate, and, therefore, it may indirectly increase the toxic effects of other exhaust components by increasing lung concentrations.

Sulfur dioxide and diesel particulate matter have an indirect interactive health effect in that the particulates can function as a catalytic surface which enhances the rate of conversion of SO₂ into sulfuric acid. The sulfuric acid has toxicological effects similar to but much more potent than SO₂. Conversely, the adsorption of much of the diesel exhaust hydrocarbon fraction, and especially the polycyclic aromatic compounds, onto the diesel particulates may limit the toxic effects of these compounds by reducing their bioavailability (see Section 3.4).

Kane and Alarie (1978) tested mixtures of formaldehyde and acrolein, both respiratory rate inhibitors, and found that they act at the same receptor site and exhibited competitive agonism. In later studies, these same authors analyzed the effects of mixtures of SO₂ and acrolein on respiratory rate and found that, depending on concentration ratio, either compound could alter or block the effect of the other (Kane and Alarie 1979).

These examples illustrate the difficulties encountered in attempting to evaluate the potential health effects of such a complex

gas-particle mixture as diesel exhaust. Of major concern for confined space situations is the possible additive effects of the major exhaust components. Consequently, it may not be adequate to ensure only that the occupational exposure limits for the individual exhaust components are not exceeded; but it must also be ascertained that the combined effect does not exceed a threshold value. For mixtures of compounds that produce similar toxic effects, OSHA (1982) requires that the sum of the concentrations of the compounds (when expressed as the fractional part of their individual TWAs or ceiling limits) not exceed 1.0.

$$E_m = \frac{C_1}{L_1} + \frac{C_2}{L_2} + \dots + \frac{C_n}{L_n}$$

where: E_m is the equivalent exposure of the mixture,
C is the concentration of a particular compound,
L is the exposure limit for that compound.

4. CONFINED SPACE WORKPLACE CONCERNS WITH THE USE OF DIESEL ENGINES

4.1 CONTROL OF DIESEL EMISSIONS

The operation of diesel engines in confined spaces can result in the buildup of exhaust gases and particulates to levels potentially hazardous to human health. As stated by Hurn (1975) with respect to the safe operation of diesel engines in mines, two approaches are possible: (1) accepting emissions from the engine at any level and providing adequate ventilation to reduce the toxicant concentrations to acceptable levels or (2) minimizing the amount of toxic material produced by the engine and thereby minimizing the amount of ventilation required. It is the second approach which will be the subject of this section.

Smaby and Johnson (1979) have categorized diesel engine control technology into the areas of fuel and fuel/air modification, engine and fuel injection design modifications, and exhaust after-treatment modifications. These areas will provide the framework for the following discussion.

4.1.1 Fuel and Fuel/Air Modification

This section will briefly report on fuel composition, fuel additive effects, exhaust gas recirculation (EGR), turbocharging, fuel and alcohol fumigation, and water addition.

4.1.1.1 Fuel Composition

Diesel engines can run on a variety of fuels, and the makeup of the fuel has been shown to have effects on the exhaust emissions (see Ryan et al. 1981, for review). Those fuel properties having the greatest effect on the amount of exhaust smoke produced are boiling range, viscosity, specific gravity, aromatic content, hydrogen content, and cetane number (Ryan et al. 1981). A number of studies have indicated that smoke and particulate emissions can be reduced through use of fuels with a lower cetane number; lower density and/or lower aromatic content (Henein and Bolt 1969; Shamah and Wagner 1973; Frisch et al. 1979). Hare (1975) tested three fuels of increasing density, aromaticity, and sulfur content and found that particulate emissions were lowest (28-59 mg/m³) for the lightest fuel (No. 1 kerosene or DF-1) and highest (48-65 mg/m³) for the No. 2 diesel fuel.

Bykowski (1981), studying 11 fuels, found higher aromatic levels to be associated with increased emissions, whereas increased olefin levels were generally associated with decreased emissions. In testing five fuels in two different engines, Baines et al. (1982) found emissions of hydrocarbons and particulates to be the most sensitive to differences in the fuels, whereas NO_x emissions were relatively constant (Table 4.1). Hydrocarbons and CO increased with the minimum quality fuels.

TABLE 4.1. EMISSIONS SENSITIVITY TO DIESEL FUEL VARIATIONS^a

	Volkswagen		Mercedes-240D	
	5-fuel avg.	Range ^b avg.	5-fuel avg.	Range ^b avg.
HC, g/km	0.29	186%	0.44	79%
CO, g/km	0.58	55%	0.63	22%
NO _x , g/km	0.60	13%	0.81	19%
Fuel economy, mi/gal	40.5	10%	26.6	9%
Particulate, g/km	0.24	83%	0.31	47%

^aAdapted from Baines et al. 1982

^b $\frac{\text{Range}}{\text{Avg.}}$ = (Maximum minus minimum)/mean of 5 values.

The fuels used were (1) Diesel Fuel No. 2 (DF-2) Emission Test Fuel (used by EPA in 13-mode testing of heavy-duty diesels), (2) a DF-2 having the properties of a "national average" diesel fuel, (3) DF-1, (4) a DF-1 representing a minimum-quality DF-2 typical of the direction towards which diesel fuels are moving, and (5) a premium-quality DF-2.

Ryan et al. (1981) have noted that jet fuels, and particularly JP-7 jet fuel, have many properties - including high gravity, low boiling range end point, low aromatic content and low viscosity - which would result in considerably reduced diesel particulate emissions, even though the cetane number (53) is above that of No. 2 diesel fuel (cetane number 47.8).

Holtz (1960) reported on tests conducted by the Bureau of Mines which showed that a low cetane fuel (cetane number 30) and low fuel-to-air ratios (data not given) resulted in slight increases in carbon monoxide and aldehydes when compared with higher cetane fuels (up to 72) and higher fuel-to-air ratios. Similarly, Iwai et al. (1976) reported a more than threefold increase in acetaldehyde, acrolein, formaldehyde, and crotonaldehyde in the exhaust of a diesel engine run on a cetane No. 45 fuel in comparison to the same engine operated on a cetane No. 55 fuel (see Table B-12).

Taigel (1951-52) tested several fuels of low sulfur content and which were nitrogen-base free and found a reduction in exhaust NO_x.

The sulfur content of diesel fuel has a direct effect on the amount of sulfur oxides released in the engine exhaust. The limits of sulfur content for military fuels (GSA 1980) are 0.25 mass % for grade DF-A (arctic grade), 0.50 for DF-1 (winter grade), 0.50 for DF-2 CONUS (Continental U.S.) regular grade, and 0.70 for DF-2 OCONUS (outside Continental U.S.) regular grade. These limits seem somewhat higher than the sulfur content found in a variety of commercially available diesel fuels (Clark et al. 1982, Huisingh et al. 1981, Hare 1975). Hare and Bradow (1979) reported that the sulfur content of a "national average"

DF-2 was 0.25 mass % (Bureau of Mines 1976 survey). Truex et al. (1980) note that diesel fuels average about 0.2 mass % sulfur content.

Truex et al. (1980) found that diesel sulfate (SO_4^{2-}) emissions are more or less independent of engine type or operating mode, and that the SO_4^{2-} accounted for several percent by mass of the total airborne exhaust particulate matter. Diesel sulfate emissions were found not to be proportional to the fuel sulfur level, the percent sulfur-to-sulfate conversion increasing as fuel sulfur decreased (0.96% conversion for 0.190% fuel sulfur, 1.8% for 0.110% fuel sulfur, and 8.6% for 0.009% fuel sulfur). The mechanism of conversion was postulated to be oxidation of SO_2 on the surface of carbonaceous diesel particulates.

In contrast to the situation described above for emission of SO_4^{2-} , diesel SO_2 emission levels are a direct function of the percent sulfur in the fuel (Johnson 1975). A hyperbolic relation exists, however, between air-to-fuel ratios and SO_2 emissions at different sulfur contents, as shown in Figure 3.5. In this figure, an air-to-fuel ratio of 20 to 1 is taken to represent 100% load and an air-to-fuel ratio of 125 to 1 represents 0% load. The author concluded that SO_2 in diesel exhaust would not be a major problem considering the average sulfur content of U.S. diesel fuel oils. At an average of 0.25 mass % sulfur content, it would appear that even for low-emission engines, SO_2 would not be the limiting emission for dilution of diesel exhaust so that all pollutants would be below the TLVs. The Mine Safety and Health Administration requires that all diesel engines approved for use in underground noncoal mines use diesel fuel with not more than a 0.5% sulfur content (Holtz 1960).

Changes in diesel exhaust composition with fuel type have also been correlated with the results of biological assays of diesel particulate extracts. Baines et al. (1982) found an increase in Ames test response (mutagenicity) when higher-aromatic-content fuels were used and also when a commercial cetane improver was used. Clark et al. (1982) also found that high aromatic content of diesel fuel could enhance the production of mutagenic combustion products. The effects were also dependent on engine type, with an engine having a precombustion chamber showing less production of mutagenic components than a larger engine with a swirl-type combustion chamber.

4.1.1.2 Fuel Additives

There is a considerable literature on the use of additives in diesel fuel. The GSA specifications (GSA 1980) give permissible levels for antioxidants (gum preventers), cetane improvers (reduction of ignition lag), corrosion inhibitors, and fuel-system icing inhibitors. Smoke-reducing additives, which are not specified, give conflicting results. Thus, as reported by Truex et al. (1980), while addition of a barium additive resulted in 15-55% reduction in particulate emissions at higher and lower but not at intermediate speeds, use of a barium-calcium additive gave an increase in total particulate mass emission rates, even though smoke-meter readings were reduced, through decrease in size of the particulates. It is generally considered (Smaby and Johnson 1979)

that the effect of this class of additives is usually more or less cosmetic (i.e., reducing the visibility of the smoke, rather than reducing the amounts of particulates).

While further discussion of the effects of additives on emissions is beyond the scope of this report, one "additive" which must be considered as a usually necessary part of the fuel is the cetane number improver. The cetane number of a fuel is its behavior with respect to that of a standard fuel made of a certain volume percent of cetane (cetane number 100) blended with α -methylnaphthalene (cetane number 0), a high cetane number indicating high ignitability. Because of this high ignitability, high cetane number fuels improve the starting characteristics of diesel engines and reduce the white smoke that is usually associated with cold starts. The effect on black smoke, however, is exactly opposite (Smaby and Johnson 1979) - the longer ignition delay of a low cetane number fuel allows better fuel/air premixing and thereby reduces the particulates formed during diffusion burning. Thus there is a trade-off. Studies reported by Smaby and Johnson (1979) indicate that optimum emissions are generally achieved at cetane numbers between 50 and 60; however, these authors note that the strong interaction between cetane number and combustion chamber/fuel injection design makes it difficult to separate and quantify the effects of cetane number relative to emission levels.

Examples of cetane number improvers which are used include nitrates, nitroalkanes, nitrocarbonates, and peroxides (Lapedes 1978). The GSA (GSA 1980) allows as cetane number improvers amyl nitrate, isopropyl nitrate, hexyl nitrate, cyclohexyl nitrate, 2-ethylhexyl nitrate, and octyl nitrate, to a level of 0.25 percent by weight for DF-A and 0.50 percent by weight for DA-1 and DF-2. The cetane number for DF-A must be at least 40, and for DF-1 and DF-2 CONUS and OCONUS at least 45.

4.1.1.3 Exhaust Gas Recirculation

Exhaust gas recirculation (EGR) is the controlled recirculation of a portion of the exhaust gas into the intake air for the principal purpose of reducing NO_x emissions (Smaby and Johnson 1979; Hurn 1975). Smaby and Johnson, however, state that EGR has few benefits when engines are operating at high loads (50-75% of maximum) due to the shortage of excess air accompanying these conditions. They present figures which indicate that under 50 and 75% load, as the percentage of EGR increases, NO_x emissions continue to decrease, but hydrocarbons, smoke, and CO emissions increase. At lower loads, Smaby and Johnson report that EGR results in higher hydrocarbons being emitted and poor driveability. Thus, an extensively controlled EGR system [such as that reported by Stumpp and Banshof (1978, as cited in Smaby and Johnson 1979) in which NO_x emissions were controlled by 37-60% without increasing hydrocarbon emissions] is a necessity. Other potential problems with the use of EGR include deposits in the intake manifold and valves and increased engine

wear. Smaby and Johnson conclude that there is a need for considerable development work if EGR is to be used with diesel engines.

4.1.1.4 Turbocharging

With respect to emission control, the chief advantage of a turbocharger is a reduction in steady-state smoke levels (Smaby and Johnson 1979; Hurn 1975). The principal reason for using a turbocharger is, however, to improve power output. As described by Smaby and Johnson, the conventional turbocharger utilizes a small turbine driven by the exhaust gas, with the output from the turbine being used to drive a compressor that compresses the inlet air. The result of this compression of inlet air is higher charge rates in the combustion chamber, thereby increasing power. The improvement effected by the turbocharger stems from the fact (Ricardo and Glyde 1941) that in even a moderate-speed diesel no possible design of a mechanical injector can bring the diesel fuel to the air in the short cycle time available. Therefore, the air must be brought to the fuel, either by swirl or by the use of a precombustion chamber or similar means, or the quantity of air handled must be increased, to increase the likelihood of oxygen molecules meeting fuel droplets. This is achieved by packing in air with a turbocharger, which does not preclude use of most of the other means of reducing the emissions or of improving the diesel cycle efficiency.

An example of results achieved by the use of a turbocharger is given by Smaby and Johnson (1979). With a turbocharger correctly matched to an engine, fuel economy improved 6%, specific output increased from 0.29 to 0.39 bhp/in³, steady-state smoke levels were improved, and CO emissions were reduced by 20%. This was a direct-injection engine. In a swirl-chamber engine which had NO_x emissions of 3.2 g/bhp-hr when naturally aspirated and 3.5 g/bhp-hr when turbocharged, use of an after-cooler to decrease the temperature of the charge reduced the NO_x emission to 2.9 g/bhp-hr.

Smaby and Johnson do, however, note that while steady-state smoke levels are reduced, excessive smoke during acceleration as well as unsatisfactory acceleration is a shortcoming. This results from the inertia of the turbine and compressor, causing a delay in response during acceleration (Dorfler 1975, as cited in Smaby and Johnson 1979).

Turbochargers are being fitted to very small diesels, smaller than the engines considered in this problem definition study; however, the extra initial cost, higher demands for maintenance, and possible costs for replacement are potential drawbacks.

4.1.1.5 Fuel and Alcohol Fumigation

As described by Smaby and Johnson (1979), fuel fumigation involves adding a portion of the fuel charge to the intake air before the air enters the combustion chamber. This aspirated fuel is thoroughly mixed with the intake air and is slowly oxidized in the combustion chamber, causing an increase in compression pressure and a smoothing out of the pressure diagram, thus reducing combustion noise. A study by Russell

(1977, as cited in Smaby and Johnson 1979) found that with 20% fuel fumigation at peak torque conditions, both smoke and noise were reduced, NO_x was basically unchanged, and brake specific fuel consumption (BSFC) was slightly increased. However, at full load with 20% fumigation, although a small improvement in smoke was observed, fuel economy decreased substantially. In addition, unburned hydrocarbons increased unacceptably, especially at light load conditions. Smaby and Johnson concluded that fuel fumigation is not a very feasible approach to fuel economy/emissions problems.

Heisey and Lestz (1981) have prepared an extensive evaluation of the performance and emissions characteristics of alcohol fumigation in a direct injected diesel engine, both with and without the addition of water. Increased CO and unburned hydrocarbon formation resulted during alcohol fumigation; water content had no significant effect. However, relative NO_x emissions decreased with higher alcohol/water content for all load emissions. In addition, particulate emissions were reduced by ethanol fumigation, but limited data suggest that the biological activity of this particulate may be increased as measured by the Ames test (Heisey and Lestz 1981).

4.1.1.6 Water Addition

The primary purpose of water addition is to reduce the combustion temperature which in a diesel engine will result in reduction of particulates and NO_x (see Ryan et al. 1981, for review). A number of practical problems in using water addition which must be considered include (Bascom et al. 1971, Daimler-Benz 1976, Marshall and Fleming 1971b, all cited in Smaby and Johnson 1979):

1. Control of the water-to-fuel ratio under varying load and speed conditions.
2. Necessity of a tank to hold large amounts of water.
3. Durability of the engine and the water injection equipment.
4. Corrosion of the water system.

The following sections will briefly describe emulsion, fumigation, and injection as water-addition methods.

Emulsion

Research by Dainty et al. (1981) and Lawson (1981) on using water/fuel emulsions was conducted using both the Deutz F6L714, four-cycle, air-cooled, V-configuration, indirect-injection engine and the Detroit Diesel Allison Division 8V71N, two-cycle, water-cooled, 8-cylinder, V-configuration, direct-injection engine. Their results can be summarized as follows:

1. The application of emulsified fuel treatment to the two-stroke engine was not practicable.
2. Fifteen percent water content in the fuel of the Deutz engine resulted in a 40 to 50% reduction in both NO_x and particulates.
3. Fifteen percent was found to be an optimum value to achieve significant NO_x and particulate reductions without increasing CO; however, increases were observed in emissions of total hydrocarbons.

Callahan et al. (1983) tested two water-in-fuel microemulsions (5% and 20.7%) and found that, depending on engine parameters such as compression ratio, injection rate, and injection timing, substantial reductions in particulate emissions could be achieved. The 5% emulsion was more effective than the 20.7% emulsion, and injection timing was the most important engine-operating parameter. In a continuation of these studies O'Neal et al. (1983) found that although the microemulsion fuel yielded lower particulates (-28.4%, average) and oxides of nitrogen (-10.4%) but higher emissions of HC (+32.2%) and CO (+65.3%) in steady-state tests, the cycle-averaged emissions in all categories were increased in transient tests.

Fumigation

Fumigation of water into the inlet air is another way of introducing water into the combustion chamber. Smaby and Johnson (1979) state, however, that water-fuel emulsions can do a better job with fewer practical problems.

Injection

This method consists of adding water to the combustion chamber by direct injection. Smaby and Johnson (1979) consider that the practical problems (e.g., a large amount of storage water) do not justify the use of water injection.

4.1.2 Engine and Fuel Injection Design Modifications

This section will briefly discuss combustion systems, lubricating oil consumption control, adiabatic engines, and the use of ceramic materials in diesel engines.

4.1.2.1 Combustion Systems

Although diesel engines are currently built with a variety of combustion chamber configurations, the major distinctions are direct injection versus indirect injection (Smaby and Johnson 1979). With respect to emissions control, indirect injection engines are stated by Smaby and Johnson to be more effective than direct injection engines because the higher turbulence and high rate of pressure rise in the swirl chamber engine promote more complete combustion.

Smaby and Johnson (1979) cited two studies which compared emissions characteristics of direct versus indirect engines. The first (Torpey et al. 1971) found that gaseous emissions were approximately twice as high with direct injection engines. In the second study, Stewart et al. (1975) observed that, for engines operating at full load, indirect injection engines have lower particulate emissions; however, this situation was reversed at lower engine loads.

As stated by Smaby and Johnson, fuel injection characteristics such as injection timing, injection pressure, and the shape of the injection rate versus time are very important in obtaining low emissions, especially with respect to direct injection engines. The most important of these with respect to emissions is injection timing, with the most universal trend being an improvement in NO_x as injection is retarded. However, as injection is retarded most other engine characteristics are degraded. This is illustrated in a study by Torpey et al. (1971, as cited in Smaby and Johnson 1979). They found that injection retard gave more NO_x improvement with direct injection engines than with indirect injection engines; however, the retarded condition of the direct injection engine produced high smoke and hydrocarbon emissions, making it impractical to operate with the injection retarded very far. Thus, one of the advantages of the indirect injection engine is the ability to retard the injection further than with the direct injection engine, thereby improving NO_x emissions. New methodologies are underway to more effectively control the combustion process in the direct injection engine. Some of these are discussed in Smaby and Johnson (1979).

4.1.2.2 Lubricating Oil Consumption Control

Lubricating oil represents one source of gaseous phase hydrocarbons and particulates from diesel engines (Smaby and Johnson 1979). The three major sources of lubricating oil are through the intake valve stem guide, through the piston clearance and rings, and through the exhaust valve stem guide. Oil leakage from the exhaust valve represents the greatest source of emissions since oil from the other two sources would probably be partially oxidized during combustion. Therefore, attention to the composition of lubricating oils as well as leak stoppage are important with respect to emissions control.

4.1.2.3 Adiabatic Engines

The adiabatic diesel engine, which according to Smaby and Johnson (1979) is only in the development stage, provides an insulated combustion chamber using high temperature materials that allow "hot" operation. Reduced emissions of white smoke, odor and particulates, hydrocarbons, and carbon monoxide are expected (Stang 1978, as cited in Smaby and Johnson 1979) but with an increase of NO_x (Schultz 1976, as cited in Smaby and Johnson 1979).

4.1.2.4 Ceramic Materials

The development of ceramic engine components has been pursued in connection with the development of adiabatic engines. As stated by

Smaby and Johnson (1979), these materials can withstand high temperatures, thereby increasing the efficiency of catalytic converters, improving the performance of thermal reactors, and helping to promote hydrocarbon and particulate oxidation.

4.1.3 Exhaust After-treatment Modifications

After the components of diesel emissions have been formed, there are several methods to reduce them before they are emitted to the atmosphere. This section will discuss four of the principal methods: (1) catalysts, (2) scrubbers, (3) particulate traps, and (4) reactors.

4.1.3.1 Catalysts

There are basically two types of catalyst systems - oxidation catalysts that oxidize hydrocarbons and CO to water and CO₂ and reduction catalysts that reduce nitrogen oxides to nitrogen (Smaby and Johnson 1979). Reduction catalysts are, however, not practical for diesel engines because of the large amount of excess air and low CO concentrations (Daimler-Benz 1976, as cited in Smaby and Johnson 1979).

Oxidation catalysts require excess oxygen and high temperatures for optimum operation (Smaby and Johnson 1979). In diesel exhaust, excess oxygen is always available, but, because of this excess, the exhaust temperatures are often not high enough for efficient conversion. It should be noted that when a diesel engine is operating in a confined space with inadequate ventilation the potential exists for decreasing efficiency of a catalyst due to declining oxygen levels; efficiency would then become a function of oxygen levels. After reviewing a number of reports, Smaby and Johnson (1979) state that in general, 200°C is the minimum exhaust gas temperature that will produce satisfactory catalyst performance. Exhaust gas temperatures for diesel engines typically range from 150°C to 550°C. Exhaust temperatures increase slightly with speed and directly with load. Thus, with adequate ventilation, an oxidation catalyst's efficiency will be maximized when the engine is under full load.

In addition to hydrocarbons and CO, oxidation catalysts will provide effective odor control since most of the odor from diesels is related to partially oxygenated hydrocarbons. Effective results have been reported even at low temperatures (Sercombe 1975, as cited in Smaby and Johnson 1979). Tests have shown no significant effect on NO_x, but, on the positive side, only minor differences in the NO₂ to NO_x ratio were observed (Marshall 1978).

Smaby and Johnson state that most investigations concerning diesel catalysts have shown that particulate emissions are not affected by catalysts. They report that while this may be true if observations are based on total particulate emissions, it is expected that much of the adsorbed material on the particulates would be oxidized, thus reducing toxicity. An abstract of a Japanese paper (Fukazawa et al. 1982) indicates that when the catalyst temperature is greater than 200°C, soot particles become smaller. Changes in particle size will affect lung

depositional characteristics and thus possibly alter potential health effects (see Section 3.4).

Experiments conducted by Marshall (1975) at the Bartlesville Energy Research Center indicated that noble-metal-based catalysts (platinum) were more efficient than nickel-based catalysts in oxidizing CO and hydrocarbons.

One negative consideration is that catalysts promote the formation of SO₃ from the less toxic SO₂ (Marshall 1975), a serious concern because diesel fuels may contain 0.1 to 0.5% sulfur by mass. Mogan et al. (1977, as cited in Smaby and Johnson 1979) found that, without catalysts, the conversion of SO₂ to SO₃ is about 2-5%, whereas, with catalysts, the conversion can be as high as 90%.

4.1.3.2 Scrubbers

Water scrubbers have been used extensively to control particulates from diesel-powered mine vehicles (Mogan et al. 1977, as cited in Smaby and Johnson 1979). As discussed by Smaby and Johnson (1979), representative water scrubbers have been shown to capture 20-30% of the exhaust particulates and hydrocarbons and 40-60% of the SO₂. The addition of a catalyst converter upstream (towards the engine) of the scrubber improved the particulate capture to 40-50%, presumably due to a decrease in the hydrophobicity of the particulates by an increase in oxygenation of the particle components. Further research (Mogan et al. 1977, as cited in Smaby and Johnson 1979) has shown that advanced-design scrubbers are capable of trapping 80-99% of submicrometer-size particles. Some problems with scrubbers are cost of equipment, freezing and corrosion, need for frequent maintenance, disposal of water/sludge mixture, and fogging.

More recent research by Mogan et al. (1981) using a Volvo BM 861U underground truck has shown a 75% reduction in soot (38 mg/m³ before scrubbing to 9.5 mg/m³ after scrubbing) and a 90% reduction in SO₂ (200 ppm before to 20 ppm after).

4.1.3.3 Particulate Traps

A particulate trap consists of some type of filter arrangement for the purpose of removing particulates from the exhaust. In the simplest traps used filters are periodically replaced with new ones, and this system may be the most practical for use in military forklifts. Lawson (1981) studied the efficiency of particulate traps in a Deutz F6L 714 diesel engine with the exhaust cooled upstream of the filter by water injection. He found that exhaust filters were excellent for removing both particulates and H₂SO₄.

For prolonged use it is desirable to regenerate particulate traps. The term trap-oxidizer is used to denote a trap with a mechanism by which the collected particulate is oxidized to regenerate the trap. This is an approach strongly favored by the U.S. EPA (EPA 1980b) to meet the proposed 1985 light-duty diesel truck particulate standard of 0.26

g/mile. The problem with this arrangement is in maintaining the high-temperature conditions that ensure continual oxidation. An alternative is to oxidize the particulate only occasionally, when enough organic material has been collected by the trap to aid the process and when the exhaust temperature is high enough to initiate oxidation.

As noted by several authors (e.g., Williams 1982, Smaby and Johnson 1979), catalysts may be added to the particulate trap in order to lower the ignition temperature of the trapped material. The addition of catalysts does, however, increase the formation of sulfates from SO₂ via SO₃.

4.1.3.4 Reactors

The continuation of the combustion of exhaust gases after they leave the combustion chamber can reduce diesel exhaust emissions and is accomplished by a thermal reactor (Smaby and Johnson 1979). According to Smaby and Johnson, the reactor actually performs the same function as the oxidation catalyst, and, in fact, since catalytic materials are used to coat the reactor surface, the distinction between the two becomes obscure. Because reactors can also oxidize soot at higher temperatures, especially if a catalyst is added, the mechanism represents a combination of catalysts, traps, and reactors.

4.1.4 Summary

As summarized by Hurn (1975), the following is indicative of the status of diesel emission control:

To control smoke	{ Turbocharging Power limitation
To control CO, hydrocarbons, and odor	{ Catalytic conversion Fuel injection refinements
To control NO _x	{ Exhaust recirculation Water injection Modification of fuel injection, timing, and rate

A similar summary of emission hardware options is provided by Lawson (1981) in Table 4.2 in which both advantages and disadvantages of each method are noted.

TABLE 4.2. EMISSION CONTROL HARDWARE OPTIONS^a

Component Description	Performance	Advantages	Disadvantages
Catalysts	High-efficiency CO, THC reduction	Simple, practical application	SO ₂ to H ₂ SO ₄ conversion and possibly some NO ₂ formation
Conventional water scrubbers	Unsatisfactory for any emissions	Good flameproof device	Water consumption and servicing
Venturi water scrubbers	High efficiency possible for particulate removal	Flameproof application possible	High engine back pressure. Large clean water consumption requiring auxiliary operating equipment
Exhaust filters	Excellent for particulate and H ₂ SO ₄	Simple application with no auxiliary operating equipment required	Development of servicing techniques required
EGR	Good for NO _x reduction	Simple application	Causes increase in particulate emissions, CO and THC

^aAdapted from Lawson 1981.

Lawson (1981) states that combined systems offer the greatest potential for underground diesel emission control. The principal benefit is to cancel out shortcomings of specific components. He offers two possible combinations as the most promising. The first is the EGR/catalyst/filter combination. In this design, EGR decreases NO_x, but increases CO, total hydrocarbons, and particulates, which are controlled by the catalyst (CO and hydrocarbons) and the filter (particulates). A significant finding was that the filter was effective in reducing the H₂SO₄ emissions related to the catalyst (formed from oxidation of SO₂ to SO₃ and subsequent conversion to H₂SO₄ by reaction with water). When the exhaust gas was cooled by water injection upstream of the filter,

over 90% of the H₂SO₄ was removed by the filter. The second combination is H₂O/fuel emulsion/catalyst/filter. The water emulsion reduces both NO_x and particulates while the catalyst prevents an increase in total hydrocarbons caused by the water emulsion, and the filter further reduces particulate matter so that over 95% of particulates are removed with this combination system. Although indicating that integration of the two systems might be beneficial, Lawson states that for underground application, the EGR/catalyst/uncooled filter approach may prove to be the most practical.

4.2 EFFECT OF AMBIENT CONDITIONS ON DIESEL ENGINE EMISSIONS AND POTENTIAL HEALTH EFFECTS

4.2.1 Temperature

The effects of temperature, humidity, and barometric pressure on diesel exhaust emissions are interrelated in that all three factors affect the density of the intake air and thus can alter the fuel-to-air ratio in the combustion chamber. Holtz (1960) calculated that, for diesel engines with high ratings, there would be an increase of about 0.005 in the fuel-to-air ratio with an increase in intake air from 60°F to 120°F under low humidity conditions at sea level. For a fuel-to-air ratio of 0.060, a rise in inlet air temperature from 60°F to about 100°F would result in carbon monoxide levels in the exhaust above the current MSHA exposure limit of 100 ppm.

Reckner et al. (1965) tested two large (220 and 300 hp) direct injection diesels placed in a semienclosed area and found that when the air temperature was quite high (data not given) the engines discharged more soot and carbon monoxide than when the temperature was low. CO levels were 3700 ppm at high temperatures and 1400 ppm at low temperatures. There was a 60% difference in soot levels.

Different results were reported by Braddock (1982) who, in testing two small diesel engines (48 and 105 hp), found that for conditions corresponding to ambient temperatures of 43-67°F, and 70-82°F, there was an increase in particulate emissions at the lower temperatures. This increase was attributed to uncombusted diesel fuel. No significant temperature-related changes were seen in the emission rates of HC, CO, or NO_x.

High temperatures in an enclosed space might increase the toxicity of the gaseous components of diesel exhaust. Stokinger (1975) cites studies which indicate that the effects of a large number of central nervous system depressants would increase from 2 to 17 times with an increase in temperature from 26° to 36°C. Kerosene and paraffin showed this effect to a great degree.

4.2.2 Humidity

Holtz (1960) calculated, on theoretical grounds, the effects of increased relative humidity and intake air temperature on fuel-to-air ratio and concluded that humidity increases the ratio for any given

temperature and that there is a relatively greater increase at higher temperatures. At 60°F the ratio may increase only 0.01 with a change from 0 to 100% relative humidity (RH) but at 100°F it may increase by 0.05 with the same change in humidity. This is due to the higher vapor pressure of water at the higher temperature. Under low humidity conditions and for a fuel-to-air ratio of 0.060, the carbon monoxide level in the exhaust would reach a critical level (100 ppm) when the intake temperature increased about 40°F (from 60° to 100°F); however, at a relative humidity of 100%, the same CO level would be reached with only a 20°F increase.

Hare and Bradow (1977) tested four diesel engines (62-122 hp) under varying conditions of humidity (about 10 to 158 grains H₂O per pound-mass dry air) and found that no significant changes in HC, CO, or CO₂ emission rates; however, emission rates of NO_x decreased markedly (about 10-30%) at high humidities. The authors proposed several types of correction factors to calculate NO_x emissions at a standard humidity which would then be comparable for different engine tests.

4.2.3 Altitude

Holtz (1960) calculated that an increase of 3000 ft in altitude would result in about a 0.005 increase in the fuel-to-air ratio for diesel engines with high ratings. At 6000 ft the increase in the ratio may be as high as 0.05. For engines operating near maximum load, this may result in incomplete fuel combustion and dangerous levels of carbon monoxide in the exhaust. This situation can be avoided by reducing the maximum fuel injection rate, and adjustments to the engine should be made on the basis of the elevation at which the engine will be used (Holtz 1960).

The reduced oxygen levels at high altitudes can have an adverse effect on diesel engines if the fuel-to-air ratio is significantly increased. Less complete combustion of the fuel would result in an increase in concentration of unburned hydrocarbons, particulates, and CO in the exhaust.

The toxicity of the gaseous components can be considerably enhanced at high altitudes. This is particularly true for carbon monoxide which, because of the reduced partial pressure of oxygen, will combine to a greater extent with hemoglobin (see Section 3.1.2). Stokinger (1975) notes that a 3% HbCO level at 15,000 feet would produce toxicological effects (impaired performance) equivalent to 20% HbCO at sea level.

4.2.4 Ventilation

In an enclosed area with no air ventilation, the operation of a diesel engine will result in rapid changes in the concentration of oxygen, carbon dioxide, nitrogen dioxide, and carbon monoxide. The rates of change will be a function of engine size and operating conditions. For several different diesel engines (2 cycle direct injection, 128 bhp, and 4 cycle indirect injection, 90 bhp), Marshall and Hurn (1973, see also Hurn 1975) found that the oxygen level in an enclosed nonventilated

space decreased by 3 to 4.5% per pound of fuel consumed per 1000 ft³ of air volume (cumulative decreases were 4.5% after 1 lb, 8% after 2 lb, and 11.5% after 3 lb). At the same time, the CO₂ level increased at a rate of about 3% per pound of fuel consumed per 1000 ft³ of air volume. The NO_x level increased to a maximum of about 200 ppm at 1.75 lb fuel consumed per 1000 ft³, then slowly declined. Depending on the power output of the engines, the increase in carbon monoxide in the enclosed space was generally linear for about the first 1 to 1.5 pounds of fuel consumed per 1000 ft³, but the rate then became exponential such that a CO level of 5000 ppm was reached after only 1.5 to 3 lb of fuel was consumed per 1000 ft³. This would be equivalent to about 2.5 gal of fuel consumed per 10,000 ft³ under heavy load and about 4.5 gal of fuel consumed per 10,000 ft³ under light load (Figure 3.3). Marshall and Hurn (1973) calculated that for a 100 hp engine operating in a 5000 ft³ space a CO level of 5000 ppm would be reached in 10 min. Under such conditions the MSHA exposure limit of 100 ppm would be reached in only a few minutes. These calculations demonstrate the importance of ventilation in confined spaces in which diesels are used. Marshall and Hurn (1973) further determined that if the ventilation rate is such that the CO₂ level in the intake air is kept to a maximum of 0.5% (TLV for CO₂), equivalent to about 5 to 14% exhaust in intake air, depending on power output, then the carbon monoxide level in the intake air would remain below 100 ppm; however, they also found that under these conditions the NO_x level would be 28-86 ppm and therefore greater than the MSHA exposure limit of 25 ppm. To keep the NO_x level below the exposure limit, the amount of exhaust gases recirculated had to be kept to about 4% of the intake air. Marshall (1978) reported that for six high-speed diesel engines (3150-5000 rpm and 48-127 bhp) the NO_x (as NO) emission rates were 1.6 to 2.6 g/hr/rated hp or about equal to the acceptable maximum emission rate (AMER) for NO if the minimum ventilation is based on maintaining the CO₂ level at its threshold limit value of 0.5%. However, for these same engines, the CO emission rates were 3.3-12.7 g/hr/rated hp, or 2 to 3 times the AMER for CO. It was noted by Marshall that the CO levels could be reduced by using higher ventilation rates, by using a catalytic converter, or by derating the engines and thus limiting the maximum fuel flow. Although the latter solution will increase NO_x emission rates slightly, ventilation rates would be reduced by as much as 50%. Marshall (1978) notes that, for a very efficient engine, the minimum ventilation rate (based on dilution of CO₂) would be 40 cfm/rated hp.

To determine the minimum ventilation requirements for diesel engines operated in underground mines, the U.S. Bureau of Mines has proposed the following formula (Holtz 1960; ILO 1983):

$$Q = V \cdot \frac{C}{Y}$$

where Q = the ventilation rate (m³/min),
 V = exhaust flow at full load and rated speed (m³/min),
 C = concentration of exhaust pollutant (ppm), and
 Y = exposure limit for the exhaust pollutant (ppm).

Ventilation rates are calculated for each of the major groups of exhaust pollutants and the highest value determines the minimum requirement. This value is then doubled to provide an extra margin of safety in case there are any additive or cumulative effects of the exhaust components (Holtz 1960).

4.3 EFFECTS OF ENGINE OPERATING PARAMETERS ON AIR QUALITY

The effects of engine design on emissions (i.e., mainly whether the engine is direct injection or indirect injection) have been discussed in Section 4.1.2. In this section, the effects of how the engine is operated and of some engine settings and operating conditions on emissions are considered.

"White Smoke" is a manifestation of the emission of unburned fuel droplets when the engine is cold (starting up) or when it has cooled off by idling and is suddenly subjected to load without adequate warm-up. The obvious solution to the emission of white smoke is to run the engine outside the bunker to get it to proper operating temperature before it is put on load in the bunker, and to avoid long periods of idle in the bunker. In this connection Bosecker and Webster (1971) have shown an advantage of the indirect injection engine in that, at low loads when fuel quantities are small, combustion can take place almost totally in the prechamber, retaining heat and aiding burning and thereby reducing the amount of white smoke emitted on starting or while idling. Also, indirect injection engines generally show less sensitivity to injection pressures and orifice sizes of the injectors than do direct injection engines (Ricardo and Glyde 1941, Smaby and Johnson 1979), making the engine more tolerant to operating conditions with respect to smoke emission. However, high pressures and small injector orifices for direct injection engines provide good atomization of the fuel and low smoke when properly timed and injected at properly controlled rates (Smaby and Johnson 1979).

While turbochargers at steady state reduce emissions all around (except possibly for NO_x ; see Section 4.1.1.4), a turbocharged engine may nonetheless smoke under transient conditions due to lag of the turbocharger (Smaby and Johnson 1979).

With respect to the fuel injection rate profile, experimental work (for instance, Grigg 1976, as cited in Smaby and Johnson 1979) indicates that the termination of injection should be as fast as possible. As well as for economy, a purpose of a fast cutoff of injection is to control smoke and unburned hydrocarbons by reducing the amount of fuel injected in large drops. To further control "dribble," injection lines are made as nonelastic as possible, dampers may be fitted, and what is called nozzle sac volume (dead-end volume in the injectors) is made as small as possible (Smaby and Johnson 1979).

Overfast acceleration (i.e., injecting the fuel faster than the engine can use it) is a prime cause of emissions of particulates and CO and hydrocarbons (Smaby and Johnson 1979, Murayama et al. 1982). Training of operators to pick up loads slowly in the bunkers would minimize

such emission. Also, rate profile controlled injection pumps can be fitted (discussed by Smaby and Johnson 1979) to respond to the confined-space need.

As described by Smaby and Johnson (1979), the particulate formation process is initiated as soon as the fuel is injected into the combustion chamber and then continues in the exhaust and in the atmosphere during and after dilution of the exhaust. Of the events in the cylinder, the ignition delay period, the time between first injection and ignition, is significant in the particulate formation process, with a variety of chemical and physical processes taking place during this time. The physical processes include those connected with the decomposition, heating and vaporization, and mixing of fuel and air in the jet spray area. As the fuel is heated and exposed to air, it undergoes significant preignition reactions, such as pyrolysis. This chemical phase accelerates rapidly, and as it becomes increasingly exothermic, ignition occurs. Even during burning, while most of the fuel is quickly vaporized and burned, a small but significant portion of it is pyrolyzed, cracked, polymerized, and partially oxidized into a vast array of compounds. In the presence of free carbon from incomplete combustion, it is this fraction which gives rise to the material included in the particles. In the exhaust, growth of the particles occurs, along with condensation of hydrocarbons, sulfuric acid and water, and further conversions, and this process may continue in the atmosphere.

As discussed by Smaby and Johnson (1979), it appears that control of HC would have a favorable effect on both particulate emissions and odor by reduction of the fraction of the fuel involved in the above-described events. The authors suggest as a feasible technical approach to reduce emissions the use of four cycle engines having precisely controlled injection systems with open or closed-loop control using a microprocessor and engine/vehicle/ambient signals that would surpass the already existing hydraulic or mechanical controls of the injection rate.

An important operating parameter is engine temperature. Advantages of a fairly high temperature, as discussed by Smaby and Johnson (1979), are wider fuel tolerance, reduced white smoke, reduced odor and particulates, and reduced HC and CO. On the negative side, however, is increased emission of NO_x .

In the review and assessment of reduced emissions engines prepared for the Army by Science Applications, Inc. (Daugherty et al. 1983), an 8-hr shift duty cycle was used, selected to represent operating conditions in a closed environment (Table 4.3).

It is expected that this duty cycle would be comprised of 90/150 inside (i.e., 90 seconds in the shelter and 60/150 or 60 seconds outside) or, on a longer time basis, 60% of the duty cycle inside and 40% outside.

TABLE 4.3. MATERIALS HANDLING EQUIPMENT
Duty Cycle (8-hr shift)^a

Load	Bhp	Hours	Load x Hours
Off		1.7	0.00
Idle	0	2.0	0.00
25%	12.5	1.0	0.25
50%	25.0	3.0	1.50
75%	37.5	0.2	0.15
100%	50.0	0.1	0.1

$$2.00/8 = 0.25 \text{ average load}$$

^aAdapted from Daugherty et al. 1983

Daugherty et al. (1983) give a listing of operational parameters for diesel engines (Table 4.4). Some of these are seen to be more parameters of engine characteristics, thermodynamics, and performance than they are "operating parameters." The authors take account of this in their selection of engines; the questionnaire sent to prospective engine suppliers asked for data on the following parameters: engine speed, torque, brake horsepower (bhp), brake mean effective pressure (BMEP) (calculated from test data), fuel consumption, brake horsepower specific fuel consumption (BSFC) (calculated from test data), air intake volume, ambient temperature and barometric pressure, exhaust gas volume and temperature, engine noise output, exhaust emissions in parts per million (ppm) of carbon monoxide (CO) nitrogen oxides (NO_x) and hydrocarbons (HC), and exhaust particulates and Bosch Smoke Number.

To facilitate comparisons, the engine manufacturers were asked to provide data in accordance with the thirteen-mode diesel engine test cycle as described in paragraph 86.336-79 of the Federal Register, Vol. 42, No. 174, September 8, 1977.

Initial review of the test data disclosed that it would not be practical to compare the exhaust emission characteristics of different engines by comparing the values in ppm of CO, NO_x, and HC in the engine exhaust, or in the envisioned work cycle. The ppm of an exhaust gas component is simply the fraction of the total exhaust gas volume represented by that component. Due to the wide variation in exhaust gas volumes produced by different engines, it was necessary to compare the exhaust emissions of different engines in absolute terms. Grams per hour of each measured exhaust gas component produced by an engine operating at a particular power output were used for this comparison.

Fuel consumption and exhaust emission data for three engines, a direct injection Perkins engine and two indirect injection Deutz engines (3-cylinder and 4-cylinder) are given in Table 4.5.

TABLE 4.4. OPERATIONAL PARAMETERS FOR DIESEL ENGINES²

Feature	Typical Units
Maximum Continuous Work Output	kW, bhp at specific rpm with stated limits on max. ambient air temperature (°C, °F) and elevation above sea level (m, ft)
(Note: unstated ("assumed") conditions for other parameters are usually maximum continuous output)	
Maximum Transient or Intermittent Work Output	kW, bhp at specific rpm with stated conditions and time (may indicate temperature rise in °C, °F)
Mechanical Efficiency	% (bhp over "indicated" horsepower)
Maximum Continuous Work Output at a Range of Engine Speeds	kW, bhp at selected rpm (or graphical depiction) with stated conditions
Maximum Torque (not independent of rpm and output)	N-m, lb-ft at specific rpm
Fuel Consumption	Liters/hr, liters/kWh, kg/hr, kg/kWh, gal/hr, or lb/bhp-hr (of specific grade fuel, usually No. 2 diesel), at standard conditions of temperature and altitude, with engine at specific speeds (rpm) and work output levels (may be presented graphically)
Brake Thermal Efficiency	% (work output over energy in fuel)
Brake Specific Fuel Consumption (BSFC)	lb/bhp-hr
Oil Consumption	Liters or kg per unit of time (lb or gal/qt per time unit) at assumed rpm and output
Brake Mean Effective Pressure (BMEP)	bar, kilopascals, psi at assumed rpm and work output
Exhaust Temperature Leaving Cylinder	°C, °F at assumed conditions of rpm and output

TABLE 4.4 (continued)

Feature	Typical Units
Exhaust Temperature Leaving Exhaust System	°C, °F at assumed conditions of rpm and output
Volume of Exhaust Gas	Liters, ft ³ per unit of time under assumed conditions
Exhaust Backpressure (at cylinder exhaust)	kilopascals, psi under assumed conditions
Intake Air Requirement	Liters, ft ³ per unit of time under assumed conditions
Volumetric Efficiency	% (of volume admitted at std. temp., pressure, engine operation over piston displacement)
Vibration Spectrum	Magnitude of acceleration (G) over spectrum of frequencies (Hz) at assumed or specified conditions
Liquid Cooling System Flow Rate	Liters/min, gal/min at assumed rpm
Cooling System Heat Discharge Rate	Calories/min, Btu/min at assumed rpm and work output
Aftercooler or Intercooler Heat Exchange Rate	Calories/min, Btu/min at assumed rpm and work output
Governor Performance	Recovery time and droop (%) based on work output and % load variations (may be in rpm or specific output parameters, e.g., voltage for electric generation applications)
Mean Time Between Failure (MTBF)	Hours (between defined "failures" for operation at assumed/specified work cycles)
Mean Time Between Overhauls (MTBO)	Hours (between defined levels of required repair and maintenance operations)
Mean Time to Repair (MTTR)	Hours (to accomplish average repair causing a "failure")

^aAdapted from Daugherty et al. 1983.

TABLE 4.5. FUEL CONSUMPTION AND EXHAUST EMISSION LEVELS FOR THREE DIESEL ENGINES^f

	Perkins 4.2032 ^a Rated hp = 61.6 Engine Size = 3328 cc	Deutz F3L 912W ^b Rated hp = 46.6 Engine Size = 2827 cc	Deutz F4L 912W ^c Rated hp = 62.2 Engine Size = 3770 cc
<u>FUEL FLOW (lb/hr)</u>			
Fuel flow at 12.5 hp	10.00	8.58	10.26
Fuel flow at 25 hp	13.91	12.36	13.73
Fuel flow at 37.5 hp	18.16	16.76	17.69
Fuel flow at 50 hp	22.56	20.28 ^d	21.94
Total fuel (lb) per 8-hr shift ^e	27	24	27
<u>CO EMISSIONS (g/hr)</u>			
CO emissions at 12.5 hp	120.77	19.62	36.34
CO emissions at 25 hp	85.35	17.40	27.5
CO emissions at 37.5 hp	66.29	21.23	25.56
CO emissions at 50 hp	60.93	28 ^d	26.73
Total CO (g) per 8-hr shift ^e	426	94	146
<u>NO_x EMISSIONS (g/hr)</u>			
NO _x emissions at 12.5 hp	131.35	131.80	143.04
NO _x emissions at 25 hp	150.63	186.63	216.63
NO _x emissions at 37.5 hp	167.70	231.10	274.55
NO _x emissions at 50 hp	212.70	247 ^d	323.62
Total NO _x (g) per 8-hr shift ^e	674	796	924
<u>HC (HYDROCARBON) EMISSIONS (g/hr)</u>			
HC emissions at 12.5 hp	37.45	5.54	7.52
HC emissions at 25 hp	25.19	4.92	6.63
HC emissions at 37.5 hp	17.36	4.15	6
HC emissions at 50 hp	13.03	5 ^d	6
Total HC per 8-hr shift ^e	137	25	33

^aSquish-lip engine design.

^b3-cylinder, indirect-injection engine.

^c4-cylinder, indirect-injection engine.

^d100% rated hp, <50 HP.

^eForklift operating cycle of 0.1 hr at 50 hp, 0.2 hr at 37.5 hp, 3 hr at 25 hp, 1 hr at 12.5 hp, 2 hr at idle, and 1.7 hr with engine off.

^fAdapted from Daugherty et al. 1983.

The better results found with the 3-cylinder Deutz over the 4-cylinder one (parts otherwise identical) show the advantage of using an engine as nearly optimally adjusted to the load cycle as possible.

It would be of considerable interest to have 8-hr duty cycle results for the same engines tested in the report but using emission control devices such as the oxy-catalysts and catalyst particulate traps discussed in Section 4.1.

Bradow (1982) has conducted load-cycle simulation of diesels used in vehicles and has measured particulate and organic emissions and the mutagenic activities of the particulate extracts. For an automotive diesel comparable to the engines that would be used in the Army's forklifts, there was little variation in the emissions with different simulated driving patterns. With truck diesels, however, the changes were more marked.

Henein (1973) has considered design and operating parameters of diesels with respect to emissions. His results correlate with those already discussed. An advantage of the indirect injection engine brought out by him is that because of the greater turbulence generated, this engine can use later timing than can the direct injection one, resulting in lower levels of noise and smoke. NO_x emissions from the indirect injection engine were also found to be less sensitive to engine speed than they are from the direct injection engine.

Marshall (1978) found a change in the balance between NO and NO_2 with speed. In five engines tested, the NO_2 concentrations were from 10 to 30% of total NO_x , the NO_2 fraction being maximal at light load (maximum air-to-fuel ratio) and decreasing with increasing load. NO_2 is more hazardous than NO; this result therefore indicates that long periods of idling in the bunker should be avoided. Further, with respect to formation of NO, Sheehy (1980) has shown that at 100% load-intermediate speed, NO emission is significantly less than at 25% load-intermediate speed, 50% load-rated speed, and 75% load-rated speed.

Marshall and Hurn (1973) have studied the influence of exhaust rebreathing on emissions from a selection of gasoline and diesel engines. For the case of restricted ventilation, unacceptably high levels of CO (greater than 50 ppm) in the air of the working space resulted from exhaust rebreathing rates as low as 8% exhaust in the engine's intake. Operation in a nonventilated space resulted in highly critical CO levels (fatal in a few minutes) at times corresponding to 1 to 3 pounds of fuel consumed per 1,000 ft^3 of space. Tests involving spaces of various sizes showed that volume of the air space per se is not important -- the primary variable is fuel consumed per unit volume of air space.

4.4 DIESEL ENGINE CERTIFICATION AND MAINTENANCE

4.4.1 Certification Program of the Mine Safety and Health Administration

The Mine Safety and Health Administration (MSHA) of the U.S. Dept. of Labor has a program by which it approves mobile diesel-powered equipment for use in underground noncoal mines (see MSHA 1982; and Holtz 1960 for review). The major part of this procedure is engine testing under controlled conditions to determine exhaust gas composition for the entire operating range of the engine (power output, speed, and fuel-to-air ratio). The test results are used to calculate the ventilation required for safe operation of the engine in mines after the engine is adjusted for maximum acceptable fuel injection rate. The only specific requirement in these tests is that the concentration of carbon monoxide in the exhaust not exceed 0.30%. Under conditions of maximum power output, the CO concentration could exceed 0.30% because of the richness of the fuel/air mixture. If this occurs, a readjustment of the fuel injection rate is required, and this then becomes the maximum allowable fuel injection rate at sea level (for higher attitudes, lower rates are specified, see Section 4.2.3). The exhaust gases are then analyzed for carbon dioxide, carbon monoxide, oxides of nitrogen (NO_x) and aldehydes, and the safe ventilation rate is calculated as 2 times the rate necessary to reduce the concentrations of these components to safe levels (0.5% for CO₂, 0.01% for CO, 25 ppm for NO_x and 10 ppm for aldehydes) (see Section 4.2.4).

MSHA certification also includes testing of the exhaust gas conditioner, a required engine accessory designed to reduce exhaust gas temperature to less than 170°F under any operating condition. Most conditioners operate by adiabatic cooling of the exhaust by evaporation of water. This is achieved by bubbling the gas through a water reservoir. The conditioner also acts as a spark arrestor.

Certification also involves the examination and approval of the electrical system, electrical or hydraulic starting systems, and exhaust dilution system. The latter system is a required engine accessory which provides for the rapid mixing of the exhaust with the ventilation air, thereby avoiding local buildup of any exhaust pollutant. This is usually achieved by venting the exhaust near the radiator or by using special diffusers that inject the dilution air into the exhaust.

As noted by Holtz (1960), a fire extinguisher and a readily accessible fuel shutoff valve for emergency use are necessary for engine certification as well as sealing of the drain plug of the fuel tank to prevent leaking and sealing of the fuel metering and governor adjustments to discourage and detect tampering. Certification also requires the use of diesel fuel with minimum specifications for the flash point (not less than 140°F) and sulfur content (0.5% maximum).

4.4.2 Engine Maintenance

Proper maintenance of a diesel engine used in a confined space is extremely important to ensure that undesirable levels of exhaust

pollutants are avoided. For certification of diesel equipment for use in underground noncoal mines, MSHA requires that operators rigidly follow the inspection and maintenance procedures recommended by the engine manufacturers (MSHA 1982). The MSHA notes that the most important engine components and accessories to maintain in proper working order include the fuel injection system, the fuel pump, the air intake system, and the engine exhaust system. Malfunctioning of the fuel injectors can result in the release of excess amounts of smoke, carbon monoxide, and aldehydes. The fuel pump must be maintained at the MSHA-approved setting because higher fuel injection rates will result in dangerous levels of carbon monoxide in the exhaust. Abnormal reduced pressure in the air intake system, as a result of malfunctioning air cleaners, can result in reduced engine performance and excess emissions. The exhaust system must also be inspected regularly to ensure proper cooling and mixing of exhaust gases with the ventilation air and to detect leaks in the system. Engine certification by MSHA defines the maximum allowable pressure differential between the intake and exhaust systems. Higher pressure drops would be indicative of faulty operation and hazardous emissions. The importance of using only qualified personnel to do engine repair and maintenance, and the necessity of using replacement parts identical to those in the original equipment are stressed by MSHA (1982).

All diesel engines should have periodic attention, according to the manufacturer's specifications, with respect to changing of filters; maintaining water temperatures or running temperatures; renewing the oil; and even to decarbonizing, grinding and setting of valves and replacing piston rings. The importance of cleanliness of the oil and fuel is emphasized by Gibbons and Wolf (1980). If excess oil gets into the cylinder, contaminants in it may "plate out" on combustion space surfaces and there intermingle with fuel and fuel contaminants, causing an increase in emission products. According to the authors, such contaminants, in addition to the partially understood phenomenon of carbonizing, dynamically affect combustion in the known and partially known phenomena of wall-quenching, flame-quenching, and fuel-droplet quenching. Such contaminants may also act as "condensation nuclei" and as chemical reactants within the fluid dynamics of combustion.

The authors, using electrostatic precipitation to remove particles and contaminants from the fuel and oil, found the following benefits: reduced noise, reduced particulate emissions, reduced NO_x , reduced carbon, reduced downtime, and longer periods between oil drains. Wear and corrosion of mechanical elements, for instance, fuel pump and injector elements, was noted.

Clean (water-free; grit-free) fuel is a necessity to avoid trouble with the injector pump or pumps, and particularly with the injectors. Some diesel engines have injectors that can be easily changed without need for calibration; this is a desirable feature to maintain smooth running of the engine and the lowest possible level of emissions.

As mentioned in EPA (1980b), maintenance of trap-oxidizers, if fitted, is not expected to be a problem. The performance of the oxidizers was shown actually to improve with use.

As stated by Johnson (1980), it is generally thought that emission of NO and NO₂ will decrease during the life of a diesel engine while emission of particulate matter will increase. Hydrocarbons, smoke, and oil consumption will probably increase, causing an increase in both the total particulate and the soluble organic fractions.

4.5 MONITORING/MODELING CONSIDERATIONS

4.5.1 Use of Key Markers for Monitoring

Except for the governmental regulations of the Mine Safety and Health Administration (MSHA), which apply only to underground mining operations, there are no specific governmental standards for limiting occupational exposures to diesel exhaust. Furthermore, the standards established by MSHA are only for the CO, NO_x, and total aldehyde components of the exhaust. Occupational exposures to CO, regardless of source, are also limited by the Occupational Safety and Health Administration (OSHA). NO_x is indirectly regulated by OSHA since the two major components of NO_x, NO, and NO₂, have OSHA exposure limits. Although there is no OSHA standard for total aldehydes, individual compounds, such as formaldehyde, acrolein, acetaldehyde, and crotonaldehyde, are regulated. In addition, there are OSHA exposure limits for about 75 other compounds found in diesel exhaust (Table B-3). For most of these compounds the concentrations reported in diesel exhaust are so low that even under worst-case conditions, they would not be expected to reach their OSHA limits before the levels of the primary pollutants become dangerously high. Of the primary pollutants, CO is considered to be a greater immediate hazard than NO_x because of its exponential rate of increase in nonventilated spaces (Hurn 1975). However, there may be circumstances where NO_x emission rates are above their maximum acceptable level even when CO emission rates are within safe limits (Marshall 1978). Thus, both NO_x and CO may require monitoring unless design standards are used to ensure that the emission rates do not exceed the maximum acceptable levels.

Even if emissions of NO_x and CO were negligible, exposure to diesel exhaust in a confined space situation could still be hazardous because of the gradual buildup of CO₂. To ensure that the level of CO₂ does not exceed its TLV of 0.5%, a minimum amount of ventilation would be needed. Marshall (1978) reported that for a very efficient engine (bsfc \cong 0.4 lb/hp·hr) this would amount to 40 cfm/rated hp. Under these conditions the maximum acceptable emission rate for NO_x would be about 2.5 g/hr/rated hp and for CO about 5.5 g/hr/rated hp. If these rates are exceeded, the ventilation rate would have to be adjusted accordingly or some other method of control utilized.

Johnson (1980) found that CO₂ concentration is a good approximate indicator of overall diesel pollutant levels. In onsite studies conducted in an underground mine, a significant correlations were found between CO₂ and air concentrations of CO, NO, NO₂, and particulates (PM). The following least squares regression equations were calculated (all values in ppm except CO₂ which is % by volume and PM which is in mg/m³):

$$\begin{aligned} \text{CO} &= 55.05 \text{ CO}_2 + 0.18 \\ \text{NO} &= 37.92 \text{ CO}_2 - 0.06 \\ \text{NO}_2 &= 9.06 \text{ CO}_2 - 0.71 \\ \text{PM} &= 7.9 \text{ CO}_2 + 0.40. \end{aligned}$$

The data for NO gave the closest linear fit with CO₂, while that for CO gave the most amount of scatter with the range of CO values being as great as 18 ppm and the 95% confidence interval about 6 ppm. Using these equations it is possible to calculate the CO₂ concentration under conditions where each of the pollutants reached its MSHA or OSHA exposure limit. For a CO level of 50 ppm (MSHA) the CO₂ concentration would be 0.91%; for NO_x equal to 25 ppm (MSHA), CO₂ would be 0.66%; and for NO₂ equal to 5 ppm (OSHA), CO₂ would be 0.63%. All of these values are above the 0.5% TLV for CO₂. Consequently, the latter would be the limiting exposure factor.

In tests conducted in a dieselized coal mine, Daniel (1983) also compared exhaust pollutant concentrations with CO₂ levels and arrived at "pollutant characteristic curves," the slopes of which were 28 ppm CO/percent CO₂, 85.6 ppm NO/percent CO₂, and 6.1 ppm NO₂/percent CO₂. The CO₂ concentrations were measured values minus background levels. For pollutant occupational exposure limits, the corresponding CO₂ levels would be 1.8% for 50 ppm CO, 0.29% for 25 ppm NO_x, and 0.82% for 5 ppm NO₂ (short-term exposure limit) or 0.48% for 3 ppm NO₂ (8-hr TWA). In this case the nitrogen oxides would be the most limiting exposure factors. Daniel also notes that the relatively low CO measurements were due to the use of a catalytic convertor, without which the CO concentration would reach 50 ppm at about 0.5% CO₂. From the pollutant characteristic curve for particulates, Daniel calculated a slope of 21.9 mg/m³/percent CO₂. In the absence of a specific occupational exposure limit for diesel particulates Daniel used the 2 mg/m³ TWA for respirable combustible dust (RCD) to arrive at a limiting CO₂ concentration of 0.09%. If this same value of 2 mg/m³ is used in Johnson's equation for particulate matter, the corresponding CO₂ value is 0.8%.

Both Johnson (1980) and Daniel (1983) determined the correlation between CO₂ and SO₂ and sulfates not from actual in situ measurements, but from sulfur balance equations. Johnson used different fuel sulfur levels and varying rates of conversion to arrive at the following:

$$\begin{aligned} \text{SO}_4 &= 173.47 (S) (1-n) \text{ CO}_2 \\ &\text{and} \\ \text{SO}_2 &= 44.27 (S) (n) \text{ CO}_2 \end{aligned}$$

where SO₂ is in ppm, SO₄ in mg/m³, CO₂ in % by volume, S is percent sulfur by mass in fuel, n is the fraction conversion of fuel sulfur to SO₂, and (1-n) is the fraction of fuel sulfur converted to sulfates and present as particulates.

Using the first equation, and setting SO_4 equal to 1 mg/m^3 , which is the TLV for sulfuric acid (see Section 3.6), and S equal to 0.25%, which is near the average for No. 2 diesel fuel, then CO_2 can be calculated for different levels of conversion of fuel sulfur to SO_4 . If only 5% of the fuel sulfur is converted to SO_4 , the CO_2 level would be 0.47% at a SO_4 level of 1 mg/m^3 . With the same fuel sulfur level of 0.25%, and a 95% conversion of S to SO_2 , the second equation can be used to calculate that a similar level of CO_2 (0.45%) would be reached at an SO_2 concentration of 5 ppm, which is the TLV for this compound. Both CO_2 values are very close to the 0.5% TLV for CO_2 itself, and with different SO_2/SO_4 conversion rates they may be either above or below the CO_2 TLV (e.g., for a 10% conversion, the CO_2 level corresponding to a 1 mg/m^3 sulfuric acid level would be only 0.23%, but for a 1% conversion rate the corresponding CO_2 level would be 2.3%). Using these same equations, Daniel (1983) found that the "pollutant characteristic curve" for SO_2 would have a slope of 7.9 ppm SO_2 /percent CO_2 , and with a 2 ppm TWA for SO_2 , the CO_2 concentration would be 0.25%. Similarly, a sulfate level of 1 mg/m^3 (TWA for sulfuric acid) corresponded to a CO_2 level of about 0.27%.

Both Johnson (1980) and Daniel (1983) concluded that because of the linear relationship between CO_2 and the other exhaust pollutants, CO_2 could be used as a monitor for estimating air quality. It was stressed by both, however, that the relationship between CO_2 and the other exhaust pollutants is site specific and can vary with changes in engine or operational parameters, and therefore must be reverified on a periodic basis. More work is needed to determine if a more generalized model can be constructed based on emission rates, air volume, and ventilation rates which could then be applied to any confined space situation. One other factor which should be included in such a model is the concentration of aliphatic aldehydes, and, more specifically, that of formaldehyde and acrolein. Both of these compounds can cause significant health effects, and both can occur in diesel exhaust at levels near or above their exposure limits. Holland (1978) analyzed underground mine air for diesel exhaust pollutants and found that formaldehyde was the most sensitive indicator of a hazardous condition in that it most frequently exceeded its TLV. However, it was noted that in one mine acrolein was the only hazardous exhaust pollutant and in another it was only carbon dioxide which exceeded its TLV. Thus, under certain conditions any of these compounds may be the major factor in limiting exposures to diesel exhaust and/or to establishing minimum ventilation rates.

4.5.2 Criteria for Diesel Engine Use in Confined Space Workplaces

Current regulations governing the use of diesel engines in underground mines stipulate that atmospheric concentrations of carbon dioxide, carbon monoxide, and nitrogen oxides not exceed standard threshold tolerance limits (MSHA 1982). More comprehensive and integrated health effects models have been proposed. As reviewed by Fontana (1982), Ian W. French and Associates, under contract to Energy, Mines and Resources Canada, developed a "Health Effects Index" (H.E.I.) or ventilation

criteria (V) which is based on TLVs and measured air concentrations of CO, NO, SO₂, NO₂, and respirable, combustible dust (RCD):

$$V = \frac{CO}{50} + \frac{NO}{25} + \frac{RCD}{2} + 1.5 \frac{SO_2}{3} + \frac{RCD}{2} + 1.2 \frac{NO_2}{3} + \frac{RCD}{2}$$

It was suggested that a criterion value of 3.0 to 4.0 be considered a moderate health hazard to be avoided by the use of personal protective equipment such as respirators. A "V" level greater than 4.0 would indicate the need for an increase in air ventilation to reduce pollutant levels. Fontana (1982) reported on the results of 15 mine surveys for which the calculated "V" values ranged from 0.9 to 3.1. He notes that because of limitations in sampling and analytical technique, there may be an overall margin of error of $\pm 50\%$ in the calculated "V" values.

In an earlier study, Johnson (1980) determined the correlation between each of the Health Effects Index components and the carbon dioxide level in an underground mine in which diesel equipment was being used (see Section 4.5.1) and arrived at the following calculation:

$$V = 22.5 CO_2 + 0.18$$

Sixty-six percent of the "V" value was attributed to the effects of particulate matter, 16% to NO₂, 7% to NO, 6% to SO₂, and 5% to CO. Sulfates, as part of the particulates, accounted for only 1.5% (on the basis of a 0.06% fuel sulfur level). The CO₂ concentrations corresponding to the critical "V" values of 3 to 4 were calculated to be 0.13% and 0.17%. Johnson (1980) describes the methodology whereby monitoring of CO₂ could be used as an indicator and warning signal for adverse pollutant levels in the mine. He notes, however, that certain pollutants, such as smoke, total particulates, and total hydrocarbons, which are not included in the index, would still have to be monitored separately. To this group should also be added aliphatic aldehydes, particularly formaldehyde and acrolein.

Daniel (1983) also applied the Health Effects Index (referred to as the "Air Quality Index") to an underground mining situation. The criterion value ranged from 1.36 to 4.51. Seventy-nine percent of the value was attributed to respirable combustible dust plus sulfate, 7.7% to SO₂, 6.6% to NO, 4.8% to NO₂ and 1.0% to CO. By comparing the pollutant concentrations with the corresponding levels of CO₂ (see Section 4.5.1), Daniel calculated that if the total CO₂ concentration in the mine (exhaust level plus background level) was at or below 0.133%, then the resulting "Air Quality Index" would remain below 4.0. It was noted that in the two cases where the index exceeded 4.0, there was a one-third reduction in the normal mine ventilation rate, which would have caused the rise in pollutant levels. Daniel concluded that on a site

specific basis CO₂ could be used as a key marker to maintain the air quality of an underground mine at a safe level.

The German Democratic Republic's draft air purity standard for the use of internal combustion engines in enclosed work areas has been described by Prietsch et al. (1979):

$$\frac{C_{CO}}{MAK_{CO}} + \frac{C_{NO}}{MAK_{NO}} + \frac{C_{NO_2}}{MAK_{NO_2}} + \frac{C_{H'CHO}}{MAK_{H'CHO}} + \frac{C_{CO_2}}{MAK_{CO_2}} = X \leq 0.8$$

where: C_{CO}, C_{NO} . . . are the workplace concentrations and MAK_{CO}, MAK_{NO} . . . are the maximum acceptable concentrations. The criterion value "X" can be calculated for either entire workshift exposure periods, in which case the MAKs used are the occupational exposure limits for 8.75 ± 0.75 hr periods, or for short-term exposure periods, in which case the MAKs must all be for the same time period. It should be noted that, unlike the H.E.I. the criterion "X" does not include sulfur dioxide and particulates but does take into consideration the concentration of CO₂. Direct measurement of the workplace concentrations of each of the pollutants (with the total measurement error not to exceed +25%), is used to calculate "X". However, Prietsch et al. (1979) also describe a method whereby "X" can be estimated, on theoretical grounds, by calculating the pollutant concentrations on the basis of: (1) engine emissions (E_n; g/hr); (2) a time factor (TF) to account for starting and stopping the engine; (3) air exchange per unit volume of work area (AE); (4) pollutant exchange within the work area (PE); (5) air volume of the workplace (AV); and (6) a special factor which takes into account the effects of room size on localized concentration differences (LCD):

$$C_{N,i} = C_{N,0} + \frac{LCD}{PE \cdot AE \cdot AV} \sum_{i=1}^i TF_i \cdot E'_{n,i}$$

where C_{N,0} is the initial value of the pollutant concentration in the workplace (if nonzero). Prietsch et al. (1979) give complete details for determining each of the variables in the formula, and they provide examples where the formula can be used to modify engine use or air ventilation rates so that the criterion "X" stays at or below the standard of 0.8. For confined space workplaces in which there is little ventilation, the use of such a criteria formula (possibly with inclusion of sulfate and particulate variables) would provide a general assessment of the overall limitations on the operation of diesel powered equipment.

5. SUMMARY AND CONCLUSIONS

5.1 IDENTIFICATION AND CATEGORIZATION OF DIESEL EMISSIONS

Diesel exhaust is a complex mixture of gases, vapors, liquid aerosols, and solid particulate matter. The gaseous compounds consist of low molecular weight hydrocarbons and inorganics such as carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, and sulfur dioxide. Higher molecular weight hydrocarbons and other organic compounds (aliphatics and aromatics) are present as vapors, along with water and inorganic compounds. With cooling of the exhaust, some of these compounds may condense into liquid aerosols. The solid particulate matter of diesel exhaust consists of elemental carbon and adsorbed, high molecular weight, polycyclic organic compounds.

The various components of diesel exhaust can be categorized according to whether or not they are regulated by federal law. Emissions of carbon monoxide, nitrogen oxides (NO_x), total hydrocarbons, and total particulate matter are regulated by the U.S. Environmental Protection Agency (EPA) (Table B-1). These standards are not directly applicable to occupational exposure situations; however, the regulations of the Mine Safety and Health Administration (MSHA), which have been established for diesel equipment use in underground mines, can also be used for other confined space workplaces. The MSHA diesel emission standard for CO in noncoal, nongassy mines is 100 ppm after dilution. The NO_x standard is 25 ppm (measured as NO_2) and the total aldehyde standard is 10 ppm after dilution and measured as formaldehyde. These emission standards also function as maximum exposure limits. In addition, there is a 0.5% by volume upper limit for CO_2 in mine air and a 20% by volume lower limit for oxygen. There are no MSHA standards for total hydrocarbons, total particulates, or any other component of diesel exhaust.

Occupational exposure to diesel exhaust is not regulated by the Occupational Safety and Health Administration (OSHA); however, various components of diesel exhaust are regulated, including CO (50 ppm, 8-hr TWA), NO (25 ppm, 8-hr TWA), NO_2 (5 ppm, ceiling value), SO_2 (5 ppm, 8-hr TWA), sulfuric acid (1 mg/m^3 , 8-hr TWA), formaldehyde (3 ppm, 8-hr TWA), and acrolein (0.1 ppm, 8-hr TWA). More than 70 other compounds found in diesel exhaust are also OSHA regulated (Table B-3). Although it is generally assumed that the concentrations of most of these compounds are below the OSHA standards (Table B-6), more quantitative data are needed to verify this assumption. Hundreds of other compounds having no OSHA standards have also been found in diesel exhaust, mainly in association with particulate matter (Table B-5). For only a few of these are there guidelines for maximum exposure limits as recommended by the National Institute of Occupational Safety and Health (NIOSH) or the American Conference of Industrial Governmental Hygienists (ACGIH). Consequently, there are hundreds of identified, and possibly thousands of yet to be identified, chemicals in diesel exhaust for which there are no occupational exposure limits or guidelines. Of specific concern are those polycyclic aromatic compounds that are known or suspect carcinogens.

5.2 CHARACTERIZATION OF POTENTIAL DIESEL EXHAUST EMISSION HAZARDS

5.2.1 Carbon Monoxide

Carbon monoxide is present in diesel exhaust as a result of incomplete fuel combustion. The three most commonly used methods for measuring CO are nondispersive infrared absorption spectroscopy, gas chromatography, and detector tube analysis. The major health effects associated with CO exposure result from its combining with hemoglobin to form carboxyhemoglobin, which reduces the availability of oxygen to body tissues. If concentrations are sufficiently high, exposures to CO can affect work performance and can cause headache, fatigue, drowsiness, nausea, vomiting, and coma.

In confined space situations where a diesel engine is being operated with little or no ventilation, the CO concentration in the air will, after a period of time, increase exponentially, and, in a very short time, the OSHA and MSHA exposure limits would be exceeded. This would occur more rapidly if the engine was operating under heavy load. In such cases the CO level would have to be reduced by increasing ventilation or by using emission control devices.

5.2.2 Nitrogen Oxides

Nitrogen oxides (NO_x) form in combustion engines as a result of the oxidation of nitrogen under conditions of high temperature and pressure. The two principal nitrogen oxides in diesel exhaust are nitric oxide (NO) and nitrogen dioxide (NO_2). NO is the predominant component of NO_x , with maximum formation occurring with 5 to 10% excess air. Rapid dilution of exhaust gases slows the oxidation of NO to NO_2 . Nitrogen oxides are analyzed spectrophotometrically following conversion of NO to NO_2 .

The major health effect resulting from exposure to NO is hypoxemia, which is caused by the reaction of NO with hemoglobin to form methemoglobin. Cyanosis and labored breathing are early signs of NO exposure. While NO is not a major respiratory irritant, NO_2 directly affects the pulmonary system. Changes in effective lung compliance, increases in expiratory and inspiratory volume, and increases in pulse and respiratory rate are several observed effects. In addition, exposure to NO_2 can cause a reduced level of resistance to respiratory infection. In experimental animals acute doses of NO_2 result in pathological changes in pulmonary tissues and edema.

In confined spaces with inadequate ventilation, the operation of a diesel engine produces a gradual buildup of NO_x to a peak level, after which the concentration slowly declines. It is generally thought that the peak NO_x levels would not represent as great an immediate health hazard as the exponential rise in the CO concentration.

5.2.3 Hydrocarbons

Hydrocarbons are present in diesel exhaust as a result of incomplete fuel combustion. The gaseous hydrocarbons consist of aliphatic, alicyclic, and aromatic compounds. The predominant light hydrocarbons are ethylene (7-83 ppm), acetylene (1-38 ppm), and propylene (2-24 ppm). Heavier components consisting of indenenes, acenaphathenes, and benzothiophenes account for the oily kerosene odor of diesel exhaust and may be present at concentrations of 40 to 400 $\mu\text{g}/\text{m}^3$. The smoky-burnt odor is due to alkenones, dienones, furan, furfural, benzene derivatives, benzofurans, indanone, indenones, and naphthaldehydes. These are present at concentrations of 0.1 to 10 $\mu\text{g}/\text{m}^3$.

The standard method for monitoring for total hydrocarbons is the flame ionization detector. For detailed analysis, gas chromatography and/or mass spectrometry is necessary.

The short chain aliphatic hydrocarbons in diesel exhaust are relatively nontoxic and occur at concentrations substantially below the OSHA exposure standards. Some of the alicyclic and aromatic compounds are strong irritants, and a few can cause severe systemic effects at high doses, but, at the concentrations occurring in diesel exhaust, they probably do not represent a significant health hazard to workers occupationally exposed.

5.2.4 Particulates

Diesel exhaust particulates consist of elemental carbon particles and adsorbed polycyclic organic compounds. Particulate mass concentration can be determined gravimetrically following filtration or electrostatic precipitation. Particulate emissions increase during rapid acceleration and when the engine is operating under heavy loads.

There is little direct evidence for health effects associated with the carbon component of diesel particulates, but comparison with the effects of carbon black suggests that chronic long-term exposure could result in pulmonary changes such as pneumoconiosis and fibrosis. Further studies are needed to determine if long-term occupational exposure to diesel exhaust may produce similar changes.

Recent studies have shown that organic solvent extracts of diesel particulates are mutagenic in various assay systems, and several of the polycyclic organic compounds found on particulates are known mutagens and carcinogens. These data have raised the question as to whether there might be an increased carcinogenic risk associated with chronic exposure to diesel exhaust. No epidemiological or laboratory studies have demonstrated such an increased risk, and limited experimental data suggest that this might be due to the reduced bioavailability and inactivation of the polycyclic compounds following phagocytosis by the alveolar macrophages.

5.2.5 Aldehydes

Aldehydes are formed in diesel engines as a result of the incomplete combustion of the fuel. The predominant aliphatic aldehydes found in diesel exhaust are formaldehyde and acrolein. Formaldehyde is measured in diesel exhaust with the chromotropic acid and pararosaniline analytical methods. The 4-hexylresorcinol method with spectrophotometric detection is specific for acrolein.

Aldehydes are strong irritants to the skin, eyes, and mucous membranes. They can also cause increased pulmonary resistance, increased susceptibility to lung infection, and pathological changes in respiratory tract tissues following acute exposures. The more reactive aldehydes, such as acrolein and formaldehyde, are mutagenic in microbial assay systems and have been shown to be carcinogenic in experimental animals. In the latter case, chronic inhalation of high concentrations resulted in a significant increase in nasal and laryngeal carcinomas.

Because they are such strong irritants, aldehydes may be an immediate health hazard in confined space workplaces in which diesel engines are operating. With inadequate ventilation, air concentrations may easily exceed the 8-hr TWA of 1 ppm for formaldehyde and 0.1 ppm for acrolein. Limiting total aldehyde concentrations to 10 ppm in undiluted exhaust and maintaining a 50-to-1 ventilation rate would ensure that these levels were not exceeded. In view of the carcinogenicity of acrolein and formaldehyde to experimental animals, efforts should be made to reduce occupational exposures to the lowest level possible, at least until a maximum safe exposure level is established.

5.2.6 Sulfates and Sulfuric Acid

Diesel fuel contains as much as 1% sulfur. During the combustion process, the sulfur is oxidized to form sulfur dioxide (SO₂). The average concentration of SO₂ in diesel exhaust is about 40 ppm. The SO₂ can be further oxidized to sulfuric acid and various inorganic sulfates. Sulfuric acid accounts for more than 90% of the water-soluble sulfates in diesel exhaust.

There is currently a national air quality standard for SO₂ of 0.03 ppm annual arithmetic mean and 0.14 ppm 24-hr average; however, there are no national standards for sulfuric acid or total sulfates. Of the 50 states, only 4 have air quality standards for sulfates, and these range from 10 µg/m³ to 30 µg/m³ for a 24-hr average. There are no exposure limits for occupational exposures to total sulfates, but the OSHA 8-hr TWA for sulfuric acid is 1 mg/m³.

The two analytical methods that are routinely used for determining total water-soluble sulfate levels in atmospheric samples are the turbidimetric method and the methylthymol blue method. The barium chloranilate/uv spectrophotometric method, however, is most often used in analyzing diesel exhaust for sulfates.

Sulfates, particularly sulfuric acid, are strong respiratory and pulmonary irritants. Irritant potency is dependent not only on chemical species and concentration but also on the size of the aerosols or particulates and on the susceptibility of the exposed species. Low doses can cause minor changes in pulmonary dynamics; high or chronic doses can cause bronchial and alveolar lesions and severe pulmonary damage. However, sulfuric acid and sulfates have not been found to be mutagenic, carcinogenic, or teratogenic.

Whether sulfuric acid levels in diesel exhaust pose a health hazard in confined space workplaces is dependent on several factors, including (1) fuel sulfur content; (2) rate of oxidation of fuel sulfur to SO₂; (3) the rate of conversion of SO₂ to sulfuric acid; (4) ventilation rate; (5) air volume of the confined space; and (6) engine operating parameters. For average fuel sulfur levels of 0.25%, the sulfuric acid level in undiluted diesel exhaust can be above the OSHA exposure limit of 1 mg/m³. For a concentration of 5 mg/m³, a ventilation rate of 0.07 m³/min per rated horsepower, or more, would be needed to reduce the air concentration to the OSHA limit. In the absence of adequate ventilation, in an enclosed space of 10,000 cu. ft., the sulfuric acid level may reach the OSHA limit in only a matter of minutes, depending on the engine size and engine load.

5.2.7 Odor, Noise, and Smoke Emissions

The characteristic odor of diesel exhaust is due to various gaseous alicyclic and aromatic hydrocarbons that are formed as a result of incomplete fuel combustion. The oily-kerosene component of diesel odor has been attributed to indanes, indenenes, benzenes, tetralins, and naphthalenes. There is also a smoky-burnt odor component that is due to more oxidized derivatives such as indanone, indeneone, phenols, naphthols, furan, and furfural. The smoky-burnt odor component is derived from the liquid chromatographic oxygenate fraction (LCO). The LCO concentration in diesel exhaust averages 5 mg/m³, with individual components present at concentrations of 1 to 10 µg/m³.

Actual health effects of odor components are ill-defined because the chemicals are present in such small amounts. In confined space workplaces, diesel exhaust odor may be very annoying, but there is no evidence that this would represent a significant health hazard.

Diesel engine noise is another factor which must be considered in using diesels in confined space workplaces. For some engines, noise levels exceed current federal standards. Unless noise levels are reduced by modifying engine design, hearing protection would have to be used by personnel operating such equipment in confined spaces.

Diesel exhaust smoke is most apparent during rapid acceleration and when the engine is operating under heavy load. Exhaust smoke becomes visible when concentrations reach about 5 mg/ft³. Diesel soot, which consists of a large amount of elemental carbon as well as adsorbed organics, results from incomplete fuel combustion. Smoke opacity is measured with a light extinction meter. In confined space workplaces,

excessive amounts of diesel smoke may cause reduced visibility and worker discomfort. Although long-term health effects resulting from occupational exposures have not been established, there is the potential for adverse pulmonary effects.

5.3 CONFINED SPACE WORKPLACE CONCERNS

5.3.1 Control of Diesel Emissions

Diesel emissions can be controlled by fuel/fuel-air modifications, engine and fuel injection design modification, and exhaust after treatment modification.

Fuel composition can have a significant effect on exhaust composition. Smoke and particulate emissions can be reduced through the use of fuels with a lower cetane number, lower density, and/or lower aromatic content. However, lower cetane fuels may also result in increases in CO and aldehyde emissions. The fuel sulfur content will directly affect the concentration of SO₂ in the exhaust, and this, in turn, will determine the concentration of water-soluble sulfates and sulfuric acid. Because conversion of SO₂ to SO₄ is catalyzed by diesel particulates, the greater the concentration of particulates, the greater the amount of sulfuric acid formed.

Fuel additives can also affect diesel emissions. Smoke reducing additives can reduce smoke emissions, but, in some cases, this may actually be accompanied by an increase in the total particulate mass emission rate. Fuel additives that function as cetane improvers may increase the amount of black smoke released during periods of acceleration or when the engine is under heavy load.

NO_x emissions can be reduced by exhaust gas recirculation; however, this may be accompanied by increases in hydrocarbons, smoke, and CO emissions.

For diesel engines operating under steady state conditions, turbocharging can reduce smoke and CO emissions, but excessive smoke may be released during periods of acceleration because of a delay in the turbocharger response.

Fuel fumigation, or the addition of a small portion of the fuel charge to the intake air before the air enters the combustion chamber, can reduce both engine noise and exhaust smoke, but, under full load conditions, hydrocarbon emissions may increase substantially. Alcohol/water fumigation reduces NO_x emissions and particulates, but causes an increase in exhaust CO and unburned hydrocarbons.

Water addition, through the use of water/fuel emulsions or by fumigation or direct injection, can reduce NO_x and particulate emissions, but under certain circumstances there may be increases in hydrocarbon and CO emissions.

The two basic engine design modifications that can affect diesel emissions involve the fuel injection process. In direct injection engines, the fuel enters the combustion chamber directly, but, in indirect injection systems, it enters a small prechamber where mixing and ignition occur before the total air/fuel charge is released into the main chamber. Because of greater fuel/air mixing and more complete combustion, indirect injection engines generally have reduced emissions. Direct injection engines can also be modified to increase fuel/air mixing, as with a swirl chamber at the top of the piston, which will also reduce emissions.

Diesel emission control devices fall into four categories: (1) catalysts; (2) scrubbers; (3) particulate traps, and (4) reactors. Oxidation catalysts can be used to oxidize hydrocarbons and CO to water and CO₂. They have no effect on NO_x but can cause a more rapid rate of conversion of SO₂ to SO₃ and sulfuric acid. Reduction of hydrocarbon concentration would have the secondary effect of reducing diesel odor levels. Water scrubbers can be used to control particulates as well as hydrocarbons and SO₂. Particulate traps are filters on which exhaust particulates and sulfuric acid are collected. They may be modified so that high temperatures are generated to oxidize the collected material and thereby regenerate the trap. Reactors are high temperature reaction chambers that can further oxidize the exhaust components. They may also be fitted with oxidation catalysts to promote the reaction.

5.3.2 Effect of Ambient Conditions on Diesel Emissions

Temperature, humidity, and barometric pressure can affect diesel exhaust emissions because of their interactive effects on the density of the intake air and the resulting change in the fuel-to-air ratio in the combustion chamber. Thus, high temperatures, low humidities, and low air pressures reduce air density and, thereby, increase the fuel-to-air ratio. For engines operating at maximum power output, this can result in a substantial increase in emissions of incomplete combustion by-products.

Ventilation rate is a key ambient factor in affecting exhaust composition and in determining the buildup of exhaust pollutants in an enclosed space. Without ventilation there will be a linear decrease in the oxygen level, a linear increase in CO₂, an exponential increase in CO, and an increase, followed by a slow decrease, in NO_x. Under such conditions the CO level may exceed the OSHA exposure limit in only a few minutes. For a very efficient engine the minimum ventilation rate (based on dilution of CO₂ to an acceptable level) would be 40 cfm/rated horsepower. For any exhaust pollutant the minimum required ventilation rate can be calculated from the exhaust flow, the concentration of the pollutant in the exhaust, and the maximum exposure limit for the compound.

5.3.3 Engine Operating Parameters

The most critical engine operating parameters that affect exhaust emissions are engine speed and load. Rapid acceleration is the primary

cause of emissions of particulates, CO, and hydrocarbons. This is especially true when the engine is operating near maximum load. NO emissions decrease at maximum load due to the reduction in available oxygen. Thus, operation of diesels at intermediate speeds and loads should result in the lowest levels of exhaust pollutants.

5.3.4 Diesel Engine Certification and Maintenance

The Mine Safety and Health Administration has a certification program for diesel powered equipment used in underground mines. According to MSHA, the most important factors for minimizing diesel exhaust emissions are: (1) a properly functioning engine; (2) restrictions on maximum power output; (3) rapid and immediate dilution of exhaust with ventilation air; and (4) a minimum ventilation rate for each engine so that each primary exhaust component is at or below the maximum acceptable level (0.5% for CO₂, 0.01% for CO, 25 ppm for NO_x, and 10 ppm for total aldehydes).

Proper maintenance of diesel engines is necessary to ensure that undesirable levels of exhaust pollutants are avoided in confined space workplaces. The most important engine components and accessories to maintain in proper working order include the fuel injection system, the fuel pump, the air intake system, and the engine exhaust system. Any malfunction that alters the fuel/air ratio in the combustion chamber could substantially change the exhaust composition.

5.3.5 Monitoring/Modeling Considerations

The Mine Safety and Health Administration uses a minimum mandatory ventilation rate to ensure that diesel exhaust pollutants do not exceed acceptable levels in underground mines. In the absence of such standards, air quality in a confined space workplace in which a diesel engine is being operated must be determined from environmental monitoring and relating concentrations of specific exhaust components to short- and long-term occupational exposure limits. Attempts have been made to correlate pollutant concentrations to carbon dioxide, a stable combustion by-product, to arrive at a single key marker for monitoring. Further study is needed to determine if this technique can have wide applicability.

In the absence of monitoring data, the buildup of exhaust pollutants in a confined space can be calculated from engine emission rates, work cycle estimations, air ventilation rates, air volume of the enclosed space, and potential for mixing or stratification of the exhaust in the workplace atmosphere. In storage areas, the volume of material stored will affect the latter factor, as well as residual air volume and possibly ventilation patterns and rates. Consequently, unless it can be shown that such models provide an ample margin of safety, workplace monitoring may still be necessary.

6. LITERATURE CITED

- Abernethy, D.J., J.H. Frazelle, and C.J. Boreiko. 1982. Effects of ethanol, acetaldehyde and acetic acid in the C3H/10T $\frac{1}{2}$ Cl 8 cell transformation system. Environ. Mutagen. 4(3):331.
- ACGIH. 1982. American Conference of Governmental Industrial Hygienists. TLVs. Threshold Limit Values for Chemical Substances in Work Air Adopted by ACGIH for 1982. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.
- ACGIH. 1983. American Conference of Governmental Industrial Hygienists. TLVs. Threshold Limit Values for Chemical Substances in Work Air Adopted by ACGIH for 1983. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.
- Alarie, Y., W.M. Busey, A.A. Krumm, and C.E. Ulrich. 1973. Long-term continuous exposure to sulfuric acid mist in cynomolgus monkeys and guinea pigs. Arch. Environ. Health 27:16-24.
- Alarie, Y.C., A.A. Krumm, W.M. Busey, C.E. Ulrich, and R.J. Kantz, Jr. 1975. Long-term exposure to sulfur dioxide, sulfuric acid mist, fly ash, and their mixtures. Results of studies in monkeys and guinea pigs. Arch. Environ. Health 30:254-62.
- Alexandersson, R., G. Hedenstierna, and B. Kolmodin-Hedman. 1982. Exposure to formaldehyde: effects on pulmonary function. Arch. Environ. Health 37(5):279-284.
- Amdur, M.O. 1969. Toxicological appraisal of particulate matter, oxides of sulfur and sulfuric acid. J. Air Pollut. Control Assoc. 19:638-644.
- Amdur, M.O. and M. Corn. 1963. The irritant potency of zinc ammonium sulfate of different particulate sizes. Am. Ind. Hyg. Assoc. J. 24:326-33.
- Amdur, M.O., M. Dubriel, and D.A. Creasia. 1978a. Respiratory response of guinea pigs to low levels of sulfuric acid. Environ. Res. 15:418-423.
- Amdur, M.O., J. Bayles, V. Ugro, and D.W. Underhill. 1978b. Comparative irritant potency of sulfate salts. Environ. Res. 16:1-8.
- Amdur, M.O., R.Z. Schulz, and P. Drinker. 1952a. Toxicity of sulfuric acid mist to guinea pigs. Am. Med. Assoc. Ind. Hyg. Occup. Med. 6:318-329.

- Amdur, M.O., L. Silverman, and P. Drinker. 1952b. Inhalation of sulfuric acid mist by human subjects. Arch. Ind. Hyg. Occup. Med. 6:305-313.
- Amdur, M.O. and D.W. Underhill. 1968. The effect of various aerosols on the response of guinea pigs to sulfur dioxide. Arch. Environ. Health 16:460-68.
- Ames, R.G., M.D. Attfield, J.L. Hankinson, F.J. Hearl, and R.B. Reger. 1982. Acute respiratory effects of exposure to diesel emissions in coal miners. Amer. Rev. Resp. Dis. 125(1):39-42.
- Anderson, I. 1978. Formaldehyde in the indoor environment - health implications and the setting of standards. In P.O. Fanger, ed. Proc. Int. Indoor Clim. Symp. pp. 65-87. Danish Build Res. Inst., Horsholm, Den.
- Andon, J., H.M. Siegel, J.H. Johnson, D.G. Leddy, and S. Smaby. 1979. An Initial Assessment of the Literature on the Measurement, Control, Transport, Transformation and Health Effects of Unregulated Diesel Engine Emissions. Final Report, DOT-HS804 010. National Highway Traffic Safety Administration, Washington, DC. 516 pp.
- Appleman, L.M., R.A. Woutersen, and V.J. Feron. 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. Toxicology 23(4):293-307.
- Arthur D. Little Inc. 1969. Chemical Identification of the Odor Components in Diesel Engine Exhaust. Final Report, ADL-C-70131-F; ADL-C070132-F. Cambridge, MA. 101 pp. (Abstract).
- Avol, E.L., M.P. Jones, R.M. Bailey, N.-M.N. Chang, M.T. Kleinman, W.S. Linn, K.A. Bell, and J.D. Hackney. 1979. Controlled exposures of human volunteers to sulfate aerosols. Health effects and aerosol characterization. Am. Rev. Respir. Dis. 120(2):319-327.
- Baines, T.M., J.H. Somers, and C.A. Harvey. 1979. Heavy duty diesel particulate emission factors. J. Air Pollut. Control Assoc. 29:616-621.
- Baines, T.M., J.H. Somers, and K.H. Hellman. 1982. Effects of fuel variables on diesel emissions. J. Air Pollut. Control Assoc. 32:810-813.
- Rascom, R.C., L.C. Broering, and D.E. Wulfhorst. 1971. Design Factors that Affect Diesel Emissions. SAE Paper No. 710484. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).
- Bird, R.P., P.H. Draper, and P.K. Basrur. 1982. Effect of malonaldehyde and acetaldehyde on cultured mammalian cells. Mutat. Res. 10:237-246.

- Bosecker, R.E. and D.F. Webster. 1971. Precombustion Chamber Diesel Emissions - A Progress Report. SAE Paper No. 710672. Society of Automotive Engineers, Warrendale, PA.
- Boreiko, C.J., D.B. Couch, and J.A. Swenberg. 1980. Mutagenic and carcinogenic effects of formaldehyde. In R.R. Rice, D.L. Costa, and K.M. Schaich, eds. Genotoxic Effects of Airborne Agents. pp. 353-367. Plenum Press, NY.
- Braddock, J.N. 1981. Impact of Low Ambient Temperatures on 3-Way Catalyst Car Emissions. SAE Paper No. 810280. Society of Automotive Engineers, Warrendale, PA.
- Braddock, J.N. 1982. Impact of Low Ambient Temperature on Diesel Passenger Car Emissions. SAE Paper No. 820278. Society of Automotive Engineers, Warrendale, PA.
- Braddock, J.N. and R.L. Bradow. 1975. Emission Patterns of Diesel-Powered Passenger Cars. SAE Paper No. 750682. Society of Automotive Engineers, Warrendale, PA.
- Bradow, R.L. 1982. Diesel particle and organic emissions: engine simulation, sampling, and artifacts. Dev. Toxicol. Environ. Sci. 10:33-47.
- Brooks, A.L., R.K. Wolff, R.E. Royer, C.R. Clark, A. Sanchez, and R.D. McClellan. 1980. Biological availability of mutagenic chemicals associated with diesel exhaust particulates. In W.E. Peapelko, R.M. Danner, and N.A. Clarke, eds. Health Effects of Diesel Emissions. EPA-600/9-80-057a. U.S. Environmental Protection Agency, Washington, DC.
- Brownstein, D.G. 1980. Reflex-mediated desquamation of bronchiolar epithelium in guinea pigs exposed acutely to sulfuric acid aerosol. Am. J. Pathol. 98:577-590.
- Bushtueva, K.A. 1957. The toxicity of H₂SO₄ aerosol. Gig. i. Sanit. 22(2):17-22. (English translation: B.S. Levine. 1960. Survey of U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. vol. 1. U.S. Public Health Service, Washington D.C.
- Bykowski, B.B. 1981. Characterization of Diesel Emissions as a Function of Fuel Variables. Report No. EPA/460/3-81-015. Southwest Research Institute, San Antonio, TX.
- Callahan, T.J., T.W. Ryan, III, G.B. O'Neal, and R.W. Waytulonis. 1983. Control of Diesel Exhaust Emissions in Underground Coal Mines - Single-Cylinder Engine Optimization for Water-in-Fuel Microemulsions. SAE Paper No. 830553. Society of Automotive Engineers, Warrendale, PA.

- Campbell, K.I., E.L. George, and I.S. Washington, Jr. 1980. Enhanced susceptibility to infection in mice after exposure to dilute exhaust from light duty diesel engines. In W.E. Pepelko, R.M. Danner and N.A. Clarke. Health Effects of Diesel Engine Emissions. EPA-600/9-80-057b. U.S. Environmental Protection Agency, Washington, DC.
- Carpenter, C.P., H.F. Smith, Jr., and U.C. Pozzani. 1949. The assay of acute vapor toxicity, and the grading and interpretation of results on 96 chemical compounds. J. Ind. Hyg. Toxicol. 31:343-346.
- Cavender, F.L., J.L. Williams, W.H. Steinhagen, and D. Woods. 1977. Thermodynamics and toxicity of sulfuric acid mists. Toxicol. Environ. Health 2:1147-1159.
- Chaney, S., W. Blomquist, K. Muller, and G. Goldstein. 1980. Biochemical changes in humans upon exposure to sulfuric acid aerosol and exercise. Arch. Environ. Health 35:211-16.
- Chang, S.-G., R. Brodzinsky, R. Toossi, S.S. Markowitz, and T. Novakov. 1979. Catalytic oxidation of SO₂ on carbon in aqueous suspensions. In Proc. Carbonaceous Particles in the Atmosphere. Lawrence Berkeley Laboratory, March 20-22, 1978. Rept. No. LBL-9037. Lawrence Berkeley Laboratory, Berkeley, CA.
- Charles, J.M., D.E. Gardner, D.L. Coffin, and D.B. Menzel. 1977. Augmentation of sulfate ion absorption from the rat lung by heavy metals. Toxicol. Appl. Pharmacol. 42(3):531-538.
- Charles, J.M., W.G. Anderson, and D.B. Menzel. 1977. Sulfate absorption from the airways of the isolated perfused rat lung. Toxicol. Appl. Pharmacol. 41(1):91-99.
- Charlson, R.J., A.H. Vanderpool, D.S. Covert, A.P. Waggoner, and N.C. Ahlquist. 1974. H₂SO₄/(NH₄)₂SO₄ background aerosol: optical detection in the St. Louis region. Atmos. Environ. 8:1257-1267.
- Chen, K.C. and J.J. Vostal. 1981. Aromatic hydrocarbon hydroxylase activity induced by injected diesel particulate extract versus inhalation of diluted diesel exhaust. Abstracts, Twentieth Annual Meeting of the Society of Toxicology. The Toxicologist 1(1):73.
- Clark, C.R., T.R. Henderson, R.E. Royer, A.I. Brooks, R.O. McClellan, W.F. Marshall, and T.M. Naman. 1982. Mutagenicity of diesel exhaust particle extracts: influence of fuel composition in two diesel engines. Fundam. Appl. Toxicol. 2:38-43.
- Clayton, G.D. and F.E. Clayton, eds. 1981. Patty's Industrial Hygiene and Toxicology. 3rd ed. Vol. 2B. Toxicology, pp. 3175-3431. John Wiley and Sons, New York.

Claxton, L.D. 1980. Mutagenic and carcinogenic potency of diesel and related environmental emissions: Salmonella bioassay. In W.E. Pepekko, R.M. Danner, and N.A. Clarke, eds. Health Effects of Diesel Emissions. EPA-600/9-80-057a. U.S. Environmental Protection Agency, Washington, DC.

Cohen, I.R. and A.P. Altshuller. 1961. A new spectrophotometric method for the determination of acrolein in combustion gases and in the atmosphere. Anal. Chem. 33:726-733.

Craig, N.L., A.B. Harker, and T. Novakov. 1974. Determination of the chemical states of sulfur in ambient aerosols by x-ray photoelectron spectroscopy. Atmos. Environ. 8:15-21.

Creech, G., R.T. Johnson, and J.O. Stoffer. 1982. A comparison of three different high-performance liquid chromatography systems for the determination of aldehydes and ketones in diesel exhaust. Part I. J. Chromatogr. Sci. 20(2):67-72.

Daimler-Benz, AG. 1976. Position paper on the features of light duty diesel vehicles; submitted to the State of California Air Resources Board, October 7/8, 1976. (as cited in Smaby and Johnson 1979).

Dainty, E.D., J.P. Mogan, and A. Lawson. 1981. The impact on underground ventilation of the reduction of diesel emissions toxicity by water-in-oil fuel emulsification. CIM Bull. 74(835):91-97.

Dalby, W.E. 1982. Formaldehyde and tumors in hamster respiratory tract. Toxicology 24(1):9-14.

Daniel, J.H. 1983. Diesels in Underground Mining: A Review and an Evaluation of an Advanced Air Quality Monitoring Methodology. Thesis. University of Idaho, Moscow, IA.

Daugherty, J.M., R.A. Leimert, P.J. Murchland, D.M. Nagel, and R.H. Sievers. 1983. Review and Assessment of Reduced Emissions/Clean Burning Diesel Engines for Integration in the Army Inventory. Final Tech. Rept. No. SAI 84-170-WA. Science Applications Inc., McClean, VA.

Dorfler, P.K. 1975. Compress Supercharging of Vehicle Diesel Engines. SAE Paper No. 750335. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).

Dreisbach, R.H. 1980. Acetaldehyde, metaldehyde, and paraldehyde. In Handbook of Poisoning: Prevention, Diagnosis and Treatment. pp. 177-180. Lange Medical Publications, Los Altos, CA.

DUETZ. 1982. Klocker-Homboldt-Duetz Ag. Air-cooled diesel engines. FL 912, BF 6L913. Klocker-Homboldt-Duetz Ag, Cologne, West Germany.

Egle, J.L. 1972. Effects of inhaled acetaldehyde and propionaldehyde on blood pressure and heart rate. Toxicol. Appl. Pharmacol. 23:131-135.

EHA. 1978. Environmental Health Associates. Health Effects of Diesel Exhaust Emissions: A Comprehensive Literature Review, Evaluation and Research Gaps Analysis. Environmental Health Associates, Berkeley, CA. NTIS-PB-282795.

Ehrlich, R. 1980. Interaction between environmental pollutants and respiratory infections. Environ. Health perspect. 35:89-99.

EPA. 1971. U.S. Environmental Protection Agency. National Primary Air Quality Standards for Sulfur Dioxide. Code of Federal Regulations, Title 42, 4.10.1-410.5. (Federal Register 36(84):8186-8201).

EPA. 1973. U.S. Environmental Protection Agency. Air Pollution, Air Quality Criteria for Nitrogen Oxides, No. 15. NTIS Publication PB-240 575.

EPA. 1975. U.S. Environmental Protection Agency. Position Paper on Regulation of Atmospheric Sulfates. EPA-450/2-75-007. U.S. Environmental Protection Agency. Office of Air Quality Planning and Standards, Research Triangle Park, NC.

EPA. 1980a. U.S. Environmental Protection Agency. Regulatory Analysis of the Light Duty Diesel Particulate Regulations for 1982 and Later Model Year Light-Duty Diesel Vehicles. U.S. Environmental Protection Agency, Washington, DC.

EPA. 1980b. U.S. Environmental Protection Agency. Standard for Emission of Particulate Regulation for Diesel-Fueled Light Duty Vehicles and Light-Duty Trucks. Federal Register 45(45):14496-14525.

EPA. 1980c. U.S. Environmental Protection Agency. Control of air pollution from new motor vehicles and motor vehicle engines: gaseous emission regulations for 1984 and later model year light-duty trucks. Federal Register 45(188):63734-63784.

EPA. 1980d. U.S. Environmental Protection Agency. Control of air pollution from new motor vehicles and new motor vehicle engines: final high altitude emission standards for 1982 and 1983 model year light-duty motor vehicles. Federal Register 45(197):66984-67015.

EPA. 1983. U.S. Environmental Protection Agency. High altitude emission standards for 1984 and later model year light duty trucks: final rule. Federal Register 48(8):1418. January 12, 1983.

Faulds, A.J., Z. Waszczylo, and K.C. Westaway. 1981. Polynuclear aromatic hydrocarbons in the underground mine environment. CIM Bulletin 74(835):84-90.

Fenters, J.D., J.N. Bradof, C. Aranyi, K. Ketels, R. Ehrlich, and D.E. Gardner. 1979. Health effects of long term inhalation of sulfuric acid mist-carbon particle mixtures. Environ. Res. 19(2):244-257.

- Ferm, V.H. and S.J. Carpenter. 1967a. Developmental malformations resulting from administration of lead salts. Exp. Mol. Pathol. 7:208-213.
- Ferm, V.H. and S.J. Carpenter. 1967b. Teratogenic effect of cadmium and its inhibition by zinc. Nature 216:1123.
- Feron, V.J. and A. Kruyssen. 1977. Effects of exposure to acrolein vapor in hamsters simultaneously treated with benzo(a)pyrene or diethylnitrosamine. J. Toxicol. Environ. Health 3:379-394.
- Feron, V.J., A. Kruyssen, H.P. Til, and H.P. Immel. 1978. Repeated exposure to acrolein vapor: subacute studies in hamsters, rats and rabbits. Toxicology 9(1-2):47-57.
- Feron, V.J., A. Kruyssen, and R.A. Woutersen. 1982. Respiratory tract tumors in hamsters exposed to acetaldehyde vapor alone or simultaneously to benzo(a)pyrene or diethylnitrosamine. Eur. J. Cancer Clin. Oncol. 18(1):13-31.
- Florin, I., L. Rutberg, M. Curvall, and C.R. Enzell. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames' test. Toxicology 15(3):219-232.
- Fontana, A. 1982. Health effects index - practical tool for atmospheric evaluation in highly dieselized underground mining operations. CIM Bull. 75(842):69-72.
- Frey, J.W. and M. Corn. 1967. Physical and chemical characteristics of particulates in a diesel exhaust. Am. Ind. Hyg. Assoc. J. 23:468-478.
- Freedman, R.W. and F.A. Sharp. 1982. Determination of Sulfates in Diesel Exhaust. R.I. 8683. U.S. Bureau of Mines, Washington, DC. (Chem. Abstr. 97:114406v).
- Frisch, L.E., J.H. Johnson, and D.G. Leddy. 1979. Effect of fuels and dilution ratio on diesel particulate emissions. SAE Paper 790417. Society of Automotive Engineers, Warrendale, PA.
- Fukazawa, S., Y. Fujiwara, and S. Tosaka. 1982. An experimental study on exhaust smoke in diesel engines: Reduction of soot particulates using oxidation catalysts. Hokkaido Kogyo Daigaku Kenkyu Kiyo 10:17-25. (Chem. Abstr. 97:77972d).
- Gage, J.C. 1970. The subacute inhalation toxicity of 109 industrial chemicals. Brit. J. Indust. Med. 27:1-18.
- Gardner, D.E., F.J. Miller, J.W. Illing, and J.M. Kirtz. 1977. Increased infectivity with exposure to ozone and sulfuric acid. Toxicol. Lett. 1:59-64.

Gibbons, R.A. and B.A. Wolff. 1980. Optimizing diesel combustion: improving fuel economy, engine life, and reducing particulate and NO_x emissions with electrostatic fluid processors. In W.E. Pepelko, R.M. Danner and N.A. Clarke, eds. Health Effects of Diesel Engine Emissions. EPA-600/9-80-057a. U.S. Environmental Protection Agency, Washington, DC.

Gibson, J.E., ed. 1983. Formaldehyde Toxicity. Hemisphere Publishing Corp., Washington, DC.

Grieseimer, R.A., A.G. Ulsamer, J.C. Arcos, J.R. Beall, A.E. Blair, T.F. Collins, F.J. DeSerres, R.B. Everson, and J.F. Gamble. 1982. Report of the federal panel on formaldehyde. Environ. Health Perspect. 43:139-168.

Grigg, H.C. 1976. The Role of Fuel Injection Equipment in Reducing 4-Stroke Diesel Engine Emissions. SAE Paper No. 760126. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).

GSA. 1980. General Services Administration. 1980. Federal specifications, fuel oil, diesel. VV-F-800C.

Hare, C.T. 1975. Methodology for Determining Fuel Effects on Diesel Particulate Emissions. U.S. Environmental Protection Agency, Rept. No. EPA-650/2-75-056. Southwest Research Institute, San Antonio, TX.

Hare, C.T. and R.L. Bradow. 1977. Light-duty diesel emission correction factors for ambient conditions. SAE (Tech. Pap.) 770717. 23 pp. Society of Automotive Engineers, Warrendale, PA.

Hare, C.T. and R.L. Bradow. 1979. Characterization of heavy-duty diesel gaseous and particulate emissions, and effects of fuel composition. SAE (Tech. Pap.) 790490. 27 pp. Society of Automotive Engineers, Warrendale, PA.

Harris, J.E. 1981. Potential Risk of Lung Cancer from Diesel Engine Emissions. National Academy Press, Washington, DC.

Heisey, J.B. and S.S. Lestz. 1981. Performance and Emissions Characteristics of Aqueous Alcohol Fuels in DI Diesel Engine. National Aeronautics and Space Administration, Lewis Research Center, Cleveland, OH. Report DOE/NASA/0091-2.

Henein, N.A. 1973. Diesel engines combustion and emissions. In G.S. Springer and D.J. Patterson, eds. Engine Emissions: Pollutant Formation and Measurement. Chapter 6. Plenum Press, New York, NY.

Henein, N.A. and J.A. Bolt. 1969. The Effect of Some Fuel and Engine Factors on Diesel Smoke. SAE Paper No. 690557. Society of Automotive Engineers, Warrendale, PA.

Henschler, D. No date. Toxikologisch - arbeitsmedizinische Begründung von Mak-werte. Verlag Chemie. (As cited by Verschueren 1977).

- Hites, R.A., Y. Ming-Li, and W.G. Thilly. 1980. Compounds associated with diesel exhaust particulates. In M. Cooke and A.J. Dennis, eds. Polynuclear Aromatic Hydrocarbons: Chemical Analysis and Biological Fate. Battelle Press, Columbus OH.
- Holland, W.D. 1978. Determination of Breathing Zone Concentrations of Contaminants from Emissions from Diesel Powered Vehicles in Underground Mines. Bureau of Mines Report No. BUMINES-OFR-24-80. 127 pp. IFE Environmental Analysis Laboratories, Richmond, VA.
- Holtz, J.C. 1960. Safety with mobile diesel-powered equipment underground. Bureau of Mines Rept. No. 5616. U.S. Department of the Interior, Washington, DC.
- Holtzman, S.G. and E.D. Schneider. 1974. Comparison of acetaldehyde and ethanol: depression of motor activity in mice. Life Sci. 14:1243-1250.
- Horstman, D., M. Hazucha, E. Hank, and R. Stacy. 1982. Effects of sub-chronic sulfuric acid aerosol on human pulmonary function. Arch. Environ. Health 37(3):136-141.
- Horvath, S.M., L.J. Folinsbee, and J.F. Bedi. 1982. Effects of sulfuric acid mist exposure on pulmonary function. Environ. Res. 28(1):123-130.
- Huisinigh, J.L., D.L. Coffin, R. Bradow, L. Claxton, A. Austin, R. Zweidinger, R. Walter, J. Sturm, and R.J. Jungers. 1981. Comparative mutagenicity of combustion emissions of a high quality No. 2 diesel fuel derived from shale oil and a petroleum derived No. 2 diesel fuel. In W.H. Griest, M.R. Guerin, and D.L. Coffin, eds. Health Effects Invest. Oil Shale Dev. pp. 201-207. Ann Arbor Science, Ann Arbor, MI.
- Hurn, R.W. 1975. Diesel emissions measurement and control. Proc. of the Symp. on the Use of Diesel-Powered Equipment in Underground Mining, Pittsburgh, PA, January 30-31, 1973. pp. 47-57. Bureau of Mines Information Circular 8666. U.S. Department of the Interior, Washington, DC.
- Hurni, H. and H. Ohder. 1973. Reproduction study with formaldehyde and hexamethylenetetramine in beagle dogs. Food Cosmet. Toxicol. 11:459-462.
- IARC. 1979. International Agency for Research on Cancer. Acrolein. In IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 19, pp.479-495. International Agency for Research on Cancer, Lyon, France.
- IARC. 1982. International Agency for Research on Cancer. Formaldehyde. In IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vol. 29, pp. 345-389. World Health Organization, Geneva, Switzerland.

- Igali, S. and L. Gazso. 1980. Mutagenic effects of alcohol and acetaldehyde on Escherichia coli. Mutat. Res. 74(3):209-210.
- Ikeda, A., Y. Horiguchi, and K. Koyoshi, 1980. Studies on the biological effects of aldehydes. Kanagawa-ken Taiki Osen Chosa Kenkyu Hokoku 22:193-196 (Chem. Abstr. 94:42241a)
- ILO. 1983. International Labor Office. Encyclopedia of Occupational Health and Safety. 3rd ed, pp. 390-392, 626-631. International Labor Office, Geneva, Switzerland.
- Iwai, T., K. Furui, A. Yoshida, and M. Tashiro. 1976. Measurement of irritating odor from direct injection diesel engines and its reduction methods. 16th Int. Automob. Tech. Congr. Paper No. 2-11. pp. 93-99.
- Jensen, O.M. and S.K. Andersen. 1982. Lung cancer risk from formaldehyde. Lancet 1(8277):913.
- Johnson, J. 1975. Diesel engine design, performance, and emission characteristics. Proc. of the Symp. on the Use of Diesel-Powered Equipment in Underground Mining, Pittsburgh, PA, January 30-31, 1973. Bureau of Mines Information Circular 8666. U.S. Department of the Interior, Washington, DC.
- Johnson, J.H. 1980. Overview of monitoring and control methods for diesel pollutants in underground mines using diesel equipment. CIM Bull. 73(819):73-87.
- Johnson, R.T., S.E. Friberg, and J.O. Stoffer. 1982. Alcohol/Petroleum Systems as Fuels for Diesel Engines. DOE/CS/50026-1. U.S. Department of Energy, Washington, DC. 158 pp.
- Johnson, S.A., D.G. Graczyk, R. Kumar, and P.T. Cunningham. 1981. Analytical Techniques for Ambient Sulfate Aerosols. ANL 81-12. Argonne National Laboratory, Argonne, IL.
- Kane, L.E. and Y. Alarie. 1977. Sensory irritation to formaldehyde and acrolein during single and repeated exposures in mice. J. Am. Ind. Hyg. Assoc. 38(10):509-522.
- Kane, L.E. and Y. Alarie. 1978. Evaluation of sensory irritation from acrolein-formaldehyde mixtures. J. Am. Ind. Hyg. Assoc. 39(4):270-274.
- Kane, L.E. and Y. Alarie. 1979. Interactions of sulfur dioxide and acrolein as sensory irritants. Toxicol. Appl. Pharmacol. 48(2):305-315.
- Kankaanpaa, J., F. Elovaara, K. Hemminki, and H. Vainio. 1979. Embryotoxicity of acrolein, acrylonitrile and acrylamide in developing chick embryos. Toxicol. Lett. 4(2):93-96.
- Kellogg, W.W., R.D. Cadle, E.R. Allen, A.Z. Lazrus, and E.A. Martell. 1972. The sulfur cycle. Science 175:587-596.

- Kerr, M.D., T.J. Kulle, B.P. Farrell, L.R. Sauder, J.L. Young, D.L. Swift, and R.M. Borushok. 1981. Effects of sulfuric acid aerosol on pulmonary function in human subjects: an environmental chamber study. Environ. Res. 26(1):42-50.
- Khatri, N.J., J.H. Johnson, and D.G. Leddy. 1978. Characterization of the hydrocarbon and sulfate fractions of diesel particulate matter. Report CONF-780208-20, 24 pp. Society of Automotive Engineers, Inc., Warrendale, PA.
- Kruyssen, A., V.J. Feron, and H.P. Til. 1975. Repeated exposure to acetaldehyde vapor. Studies in Syrian golden hamsters. Arch. Environ. Health 30:449-452.
- Lapedes, D.N., ed. 1978. McGraw-Hill Dictionary of Scientific and Technical Terms. 2nd ed. McGraw-Hill Book Company, New York, NY.
- Lautenberger, W.J., E.V. Kring, and J.A. Morello. 1980. A new personal badge monitor for organic vapors. Am. Ind. Hyg. Assoc. J. 41(10):737-747.
- Lawson, A. 1981. Progress in the control of underground diesel emissions. CIM Bull. 74(835):68-73.
- Lawter, J.R. and D.A. Kendall. 1977. Effects of diesel engine emissions on coal mine air quality. Final report, BuMinesOFR-46-78, 286 pp. Bureau of Mines, Washington, DC.
- Leikauf, G., D.M. Spektor, R.E. Albert, and M. Lippmann. 1981. Effects of submicrometer sulfuric acid exposures on bronchial mucociliary particle clearance in healthy nonsmoking adults. Proc. Ann. Meet. Air Pollut. Control Assoc. Vol. 74, paper 81-11-8, 8 pp.
- Levins, P.L. 1981. Review of Diesel Odor and Toxic Vapor Emissions. Technical Report. DOT-TSC-NHTSA-81-9. Arthur D. Little, Cambridge, MA. U.S. Department of Transportation Rept. No. DOT-HS-805831.
- Lewis, T.R., W.J. Moorman, W.F. Ludman, and K.I. Campbell. 1973. Toxicity of long-term exposure to oxides of sulfur. Arch. Environ. Health 26:16-21.
- Lewkowsky, J.P., M. Malanchuk, L. Hastings, A. Vinegar, and G.P. Cooper. 1979. Effects of chronic exposure of rats to automobile exhaust, H₂SO₄, SO₂, Al₂(SO₄)₃, and CO. In S.D. Lee and J.B. Mudd. Assessing Toxic Effects of Environmental Pollutants. pp. 187-217. Ann Arbor Science, Ann Arbor, MI.
- Lewtas, J., ed. 1982. Toxicological Effects of Emissions from Diesel Engines. Elsevier Biomedical, New York, NY.
- Linnell, R.H. and W.E. Scott. 1962a. Diesel exhaust composition and odor studies. Jour. Air Pollut. Control Assoc. 12(11):510-515.

- Linnell, R.H. and W.E. Scott. 1962b. Diesel exhaust analysis. Arch. Environ. Health 5:616-625.
- Litton Bionetics Inc. 1975. Mutagenic Evaluation of Compound FDA 73-42, Ammonium Sulfate Granular, Food Grade. FDA Rept. No. FDABA-GRAS-382. Litton Bionetics, Inc. Kensington, MD. NTIS-PB-245 506.
- Lutz, D., E. Eder, T. Neudecker, and D. Henschler. 1982. Structure-mutagenicity relationship in α,β -unsaturated carbonylic compounds and their corresponding allylic alcohols. Mutat. Res. 93(2):305-315.
- Maddalone, R.F., R.L. Thomas, and P.W. West. 1976. Measurement of sulfuric acid aerosol and total sulfate content of ambient air. Environ. Sci. Technol. 10(1):162-168.
- Marshall, W.F. 1975. Emission Control for Diesels Operated Underground: Catalytic Converters. BERC/RI-75/8. Bartlesville Energy Research Center, Bartlesville, OK. 12 pp.
- Marshall, W.F. 1978. NO₂ Levels in Diesel Exhaust. BERC/TRP-78/1. Bartlesville Energy Research Center, Bartlesville OK. 8 pp.
- Marshall, W.F. and R.D. Fleming. 1971a. Diesel Emissions Reinventoried. Bureau of Mines, RI 7530. U.S. Bureau of Mines, Washington, D.C.
- Marshall, W.F. and R.D. Fleming. 1971b. Diesel emissions as Related to Engine Variables and Fuel Characteristics. SAE Paper No. 710836. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).
- Marshall, W.F. and R.W. Hurn. 1973. Hazard from Engines Rebreathing Exhaust in Confined Space. Report No. BuMines-RI-7757. 22 pp. Energy Research and Development Administration, Bartlesville, OK.
- McClellan, R.O., A.L. Brooks, R.G. Cuddihy, R.K. Jones, J.L. Mauderly, and R.K. Wolff. 1982. In J. Lewtas, ed. Toxicological Effects of Emissions from Diesel Engines. Elsevier Biomedical, New York, NY.
- McCormick, J.J., R.M. Zator, B.R. DaGue, and V.M. Maher. 1980. Studies on the effects of diesel particulate on normal and xeroderma pigmentosum cells. In W.E. Peapelko, R.M. Danner and N.A. Clarke, eds. Health Effects of Diesel Engine Emissions. EPA-600/9-80-057b. U.S. Environmental Protection Agency, Washington, DC.
- McKay, H.A.C. 1971. The atmospheric oxidation of SO₂ in water droplets in presence of NH₃. Atmos. Environ. 5:7-14.
- Mogan, J.P., A. Lawson, D.R. Stewart, and E.D. Dainty. 1977. Diesel exhaust treatment, present and future. Canadian Explosive Atmospheres Laboratory, Mining Research Laboratories, October, 1977. (as cited in Smaby and Johnson 1979).

Mogan, J.P., K. Katsuyama, and E.D. Dainty. 1981. The emission control system on the Volvo BM861 U underground truck. CIM Bull. 74(835):63-67.

Morrow, P.E., M.J. Utell, F.R. Gibb, and R.W. Hyde. 1979. Studies of pollutant aerosol simulants in normal and susceptible human subjects. Aerosols Sci., Med. Technol. Biomed. Influence Aerosol - Conf. 7th, pp. 11-20.

Morton, J.D. 1980. Biological Effects of Short, High-Level Exposure to Gases: Nitrogen Oxides. Phase Report. Environ. Control, Inc., Rockville, MD. DAMD-17-C-9086.

MSHA. 1982. Mine Safety and Health Administration. Mobile Diesel-Powered Equipment for Noncoal Mines. Code of Federal Regulation Title 30, Part 32, Sec. 32.1-32.10, revised July 1, 1982.

Murayama, T., N. Miyamoto, K. Igeta, and T. Kobayashi. 1982. Combustion Characteristics under Acceleration in Diesel Engines. JSAE Review, Nov. 1982:9-16.

Murray, F.J., B.A. Schwetz, K.D. Nitschke, A.A. Crawford, J.F. Quast, and R.E. Staples. 1979. Embryotoxicity of inhaled sulfuric acid aerosol in mice and rabbits. J. Environ. Sci. Health 13:251-266.

NATO. 1973. North Atlantic Treaty Organization. Committee on the Challenges of Modern Society. Air Pollution: Air Quality Criteria for Nitrogen Oxides PB-240-575. National Technical Information Service, Springfield, VA. 229 pp.

NCI. 1951-1978. National Cancer Institute. Survey of compounds which have been tested for carcinogenic activity. NIH Publ. No. 80-453 (formerly Public Health Service Publication No. 149). National Institutes of Health, Washington, DC.

Nesnow, S. and J.L. Huisingh. 1980. Mutagenic and carcinogenic potency of extracts of diesel and related environmental emissions: summary and discussion of results. In W.E. Pepekko, R.M. Danner, and N.A. Clarke, eds. Health Effects of Diesel Emissions EPA-600/9-80-057a. U.S. Environmental Protection Agency, Washington, DC.

Neudecker, T., D. Lutz, E. Eder, and D. Henschler. 1981. Crotonaldehyde is mutagenic in a modified Salmonella typhimurium mutagenicity testing system. Mutation Res. 91:27-31.

Nightingale, T.E. 1980. Biological Effects of Short, High-Level Exposure to Gases: Carbon Monoxide. Enviro Control, Inc., Rockville Md. DAMD 17-79-C9086. 117 pp.

NIOSH. 1972. National Institute for Occupational Safety and Health. Criteria for a Recommended Standard...Occupational Exposure to Carbon Monoxide. PB 212-629. National Technical Information Service, Springfield, VA.

NIOSH. 1976. National Institute for Occupational Safety and Health. Criteria for a Recommended Standard...Occupational Exposure to Oxides of Nitrogen (Nitrogen dioxide and nitric oxide). NIOSH Report No. 768-149. U.S. Department of Health, Education and Welfare, Washington, DC.

NIOSH. 1978. National Institute for Occupational Safety and Health. Criteria for a Recommended Standard...Occupational Exposure to Carbon Black. NIOSH Report No. 78-204, U.S. Department of Health, Education and Welfare, Washington, DC.

NIOSH. 1979. National Institute for Occupational Safety and Health. Ten NIOSH Analytical Methods, Set-5. NIOSH Report No. SCP-FU-5. National Institute for Occupational Safety and Health, Washington, DC. 283 pp.

NIOSH. 1982. National Institute for Occupational Safety and Health. Summary of NIOSH Recommendations for Occupational Health Standards. Draft Report. Division of Standards Development and Technology Transfer, National Institute of Occupational Safety and Health, Cincinnati, OH.

NRC. 1976. National Research Council. Vapor-Phase Organic Pollutants. National Academy of Sciences, Washington, DC.

NRC. 1977a. National Resource Council. Carbon Monoxide. National Academy of Sciences, Washington, DC.

NRC. 1977b. National Research Council. Nitrogen Oxides. U.S. Environmental Protection Agency Rept. No. 600/1-77-013. National Academy of Sciences, Washington, DC. NTIS Publication PB-264 872.

NRC. 1981a. National Research Council- Health Effects Panel of the Diesel Impacts Study Committee. Health Effects of Exposure to Diesel Exhaust. National Academy Press, Washington, DC.

NRC. 1981b. National Research Council. Formaldehyde and Other Aldehydes. National Academy Press, Washington, DC.

Obe, G. and B. Beek. 1979. Mutagenicity activity of aldehydes. Drug Alcohol Depend. 4(1-2):91-94.

O'Neal, G.B., H.F. Dietzmann, T.W. Ryan III, and R.W. Waytulonis. 1983. Control of Diesel Exhaust Emissions in Underground Coal Mines - Steady-State and Transient Engine Tests with a Five Percent Water-in-Fuel Microemulsion. SAE Paper No. 830555. Society of Automotive Engineers, Warrendale, PA.

OSHA. 1982. Occupational Safety and Health Administration. Toxic and Hazardous Substances. Code of Federal Regulations, Title 29, Subpart 2, Section 1910.1000.

- O'Shea, K.S. and M.H. Kaufman. 1979. The teratogenic effects of acetaldehyde: Implications for the study of fetal alcohol syndrome. J. Anat. 128:65-76.
- Pattle, R.E., F. Burgess, and H. Cullumbine. 1956. The effects of a cold environment and of ammonia on the toxicity of sulfuric acid mist to guinea pigs. J. Pathol. Bacteriol. 72:219-232.
- Perez, J.M. 1981. Measurement of unregulated emissions: some heavy duty diesel engine results. Environ. Int. 5:217-228.
- PHS. 1969. Public Health Service. Air Quality Criteria for Particulate Matter. National Air Pollution Control Administration. Washington, DC.
- PHS. 1970. Public Health Service. Air Quality Criteria for Hydrocarbons. National Air Pollution Control Administration. Publ. No. AP-64. Washington, DC.
- Pitts, J.N. Jr., D.M. Lokensgard, W. Harger, T.S. Fisher, V. Mejia, J.J. Schuler, G. Scorziell, and Y.A. Katzenstein. 1982. Mutagens in diesel exhaust particulates. Identification and direct activities of 6-nitrobenzo[a]pyrene, 9-nitroanthracene, 1-nitropyrene, and 5H-phenanthro[4,5-bcd]pyran-5-one. Mutat. Res. 103:241-249.
- Pool, B.L. and M. Wiessler. 1981. Investigations on the mutagenicity of primary and secondary α -acetoxynitrosamines with Salmonella typhimurium activation and deactivation of structurally related compounds by S-9. Carcinogenesis 2(10):991-997.
- Prietsch, W., M. Naumann, and H. Adolph. 1979. Requirements for air purity in using internal combustion engines in enclosed work areas. Technik 34:401-406.
- Rall, D.P. 1974. Review of the health effects of sulfur oxides. Environ. Health Perspect. 8:97-121.
- Reckner, L.R., W.E. Scott, and W.F. Biller. 1965. The composition and odor of diesel exhaust. API Proc., Div. of Refining (III), 45:133-47.
- Reeves, A.L., D. Deitch, and A.J. Vorwald. 1967. Beryllium carcinogenesis. I. Inhalation exposure of rats to beryllium sulfate aerosol. Cancer Res. 27(3):439-445.
- Ricardo, H. and H.S. Glyde. 1941. The High-Speed Internal-Combustion Engine. 3rd ed. Blackie and Son, Limited, London.
- Russell, M.F. 1977. Recent CAV Research into Noise, Emissions, and Fuel Economy of Diesel Engines. SAE Paper No. 770257. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).

- Ryan, T.W. III, J.D. Storum, B.R. Wright, and R. Waytulonis. 1981. The effects of fuel properties and composition on diesel exhaust emissions - a review. SAE Paper 810953. Society of Automotive Engineers, Warrendale, PA.
- Sackner, M.A., G.A. Chapman, J. Ciple, M. Kwoka, M. Reinhart, M. Brito, R. Schreck, and R.L. Dougherty. 1981. Effects of brief and intermediate exposures to sulfate submicron aerosols and sulfate injections on cardiopulmonary function of dogs and tracheal mucous velocity of sheep. J. Toxicol. Environ. Health 7(6):951-972.
- Sackner, M.A., D. Ford, and R. Fernandez. 1978. Effects of sulfuric acid aerosol on cardiopulmonary function of dogs, sheep, and humans. Am. Rev. Respir. Dis. 118:497-510.
- SAE. 1982. Society of Automotive Engineers. Continuous Hydrocarbon Analysis of Diesel Emissions. SAE Recommended Practice SAE J215 JAN80. SAE Handbook: 1982. Society of Automotive Engineers, Warrendale, PA.
- Sandmeyer, E.F. 1981a. Aliphatic hydrocarbons. In G.D. Clayton and F.F. Clayton, eds. Patty's Industrial Hygiene and Toxicology. 3rd ed., vol. 28, Chapter 45, pp. 3175-3220. John Wiley and Sons, New York.
- Sandmeyer, E.F. 1981b. Alicyclic hydrocarbons. In G.D. Clayton and F.F. Clayton, eds. Patty's Industrial Hygiene and Toxicology. 3rd ed., vol. 28, Chapter 46, pp. 3221-3251. John Wiley and Sons, New York.
- Sandmeyer, E.F. 1981c. Aromatic hydrocarbons. In G.D. Clayton and F.F. Clayton, eds. Patty's Industrial Hygiene and Toxicology. 3rd ed., vol. 28, Chapter 47, pp. 3253-3431. John Wiley and Sons, New York.
- Santodonato, J., B. Dibasu, and P. Howard. 1978. Health Effects Associated with Diesel Exhaust Emissions. Literature Review and Evaluations. Health Effects Research Lab., Research Triangle Park, NC. Report No. EPA/600/178/063. 165 pp.
- Schepers, G.W.M., T.M. Durkan, A.B. Delahant, and F.T. Creedon. 1957. The biological action of inhaled beryllium sulfate. Arch. Indust. Health 15:32-58.
- Schlesinger, R.R., M. Lippmann, and R.E. Albert. 1978. Effects of short-term exposures to sulfuric acid and ammonium sulfate aerosols upon bronchial airway function in the donkey. J. Am. Ind. Hyg. Assoc. 39(4):275-286.
- Schmid, B.P., E. Goulding, K. Kitchin, and M.K. Sanyal. 1981. Assessment of the teratogenic potential of acrolein and cyclophosphamide in a rat embryo culture system. Toxicology 22(3):235-243.
- Schultz, R.F. 1976. Ceramics in the ERDA Highway Vehicle Heat Engine Systems Program. SAE Paper No. 760238. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).

Schwartz, L.W., P.F. Moore, D.P. Chang, B.K. Tarkington, D.L. Dungworth, and W.S. Taylor. 1977. Short-term Effects of Sulfuric Acid Aerosols on the Respiratory Tract. A Morphological Study in Guinea Pigs, Mice, Rats, and Monkeys. In S.D. Lee, ed. Biochemical Effects of Environmental Pollutants. pp. 257-271. Ann Arbor Science, Ann Arbor, MI.

Schwartz, L.W., Y.C. Zee, B.K. Tarkington, P.F. Moore, and J.W. Osebold. 1979. Pulmonary responses to sulfuric acid aerosols. In S.D. Lee and J.B. Mudd, eds. Assessing the Toxic Effects of Environmental Pollutants. Ann Arbor Science, Ann Arbor, MI.

Sercombe, E.J. 1975. Exhaust purifiers for compression ignition engines. Platinum Metals Rev. 19(1). (as cited in Smaby and Johnson 1979).

Shamah, E. and T.O. Wagner. 1973. Fuel Quality or Engine Design: Which Controls Diesel Emissions? SAE Paper No. 730168. Society of Automotive Engineers, Warrendale, PA.

Sheehy, J.W. 1980. Evaluation of Exhaust Emissions Data for Diesel Engines used in Underground Mines. Report No. DHHS/PUB/80-146. National Institute for Occupational Safety and Health, Cincinnati, OH. 50 pp.

Sherwood, R.J. and D.M.S. Greenhalgh. 1960. A personal air sampler. Ann. Occup. Hyg. 2:127-132.

Siak, J.S. and K.A. Strom. 1981. Mutagenicity of diesel particulates obtained from alveolar macrophages from exposed animals. Abstract. Twentieth Annual Meeting of the Society of Toxicology. The Toxicologist 1(1):74.

Silbaugh, S. A., R.K. Wolff, W.K. Johnson, J.L. Mauderly, and C.A. Macken. 1981a. Effects of sulfuric acid aerosols on the pulmonary function of guinea pigs. J. Toxicol. Environ. Health 7:339-352.

Silbaugh, S.A., J.L. Mauderly, and C.A. Macken. 1981b. Effects of sulfuric acid and nitrogen dioxide on airway responsiveness of the guinea pig. J. Toxicol. Environ. Health 8:31-45.

Sim, V.M. and R.E. Pattle. 1957. Effect of possible smog irritants on human subjects. J. Am. Med. Assoc. 165:1908-1913.

Sittig, M. 1979. Hazardous and Toxic Effects of Industrial Chemicals. Noyes Data Corporation, Park Ridge, NJ.

Sittig, M. 1981. Handbook of Toxic and Hazardous Chemicals. Noyes Publications, Park Ridge, NJ.

Skog, E. 1950. A toxicological investigation of lower aliphatic aldehydes. I. Toxicity of formaldehyde, acetaldehyde, propionaldehyde and butyraldehyde; as well as of acrolein, and crotonaldehyde. Acta Pharmacol. 6:299-318.

Smaby, S.A. and J.H. Johnson. 1979. Control Technology. In J. Andon, H.M. Siegel, J.H. Johnson, D.G. Leddy, S. Smaby, J.N. Pitts, A.M. Winer, and K.A. Van Cauwenberghe. An Initial Assessment of the Literature on the Measurement, Control, Transport, Transformation and Health Effects of Unregulated Diesel Engine Emissions. Part B. Final Report, DOT-HS804 010. National Highway Traffic Safety Administration, Washinton, DC.

Smythe, R.J. and F.W. Karasek. 1973. The analysis of diesel engine exhausts for low-molecular-weight carbonyl compounds. J. Chromatogr. 86:228-231.

Spindt, R.S., G.J. Barnes, and J.H. Somers. 1971. The Characterization of Odor Components in Diesel Exhaust Gas. SAE Paper No. 710605. Society of Automotive Engineers, Warrendale, PA.

Spindt, R.S., et al. 1974. Polynuclear aromatic content of heavy duty diesel engine exhaust gases. First annual report. Gulf Research and Development Co. EPA Contract No. 68-01-2116. (As cited in Smaby et al. 1979.)

Springer, K.J. and T.M. Baines. 1978. Emissions from Diesel Versions of Production Passenger Cars. SAE Paper No. 770818. Society of Automotive Engineers, Warrendale, PA.

Springer, K.J. and R.C. Stahman. 1977. Diesel Car Emissions - Emphasis on Particulate and Sulfate Emissions. SAE Paper No. 770254. Society of Automotive Engineers, Warrendale, PA.

Springer, K.J. and R.C. Stahman. 1978. Unregulated Emissions from Diesels Used in Trucks and Buses. SAE Paper No. 770258. Society of Automotive Engineers, Warrendale, PA.

Sreenathan, R.N., R. Padmanabhan, and S. Shingh. 1982. Teratogenic effects of acetaldehyde in the rat. Drug Alcohol Depend. 9(4):339-350.

Stang, J.H. 1978. Designing Adiabatic Engine Components. SAE Paper No. 780069. Society of Automotive Engineers, Warrendale, PA. (as cited in Smaby and Johnson 1979).

Stephens, E., E. Darley, O. Taylor, and C. Scott. 1961. Photochemical reaction products in air pollution. J. Air and Water Pollut. 4:79.

Stewart, D.R., J.P. Mogan, and E.D. Dainty. 1975. Some characteristics of particulate emissions in diesel exhaust. Can. Min. Metall. Bull. April, 1975. (as cited in Smaby and Johnson 1979).

Stokinger, H.E. 1975. Toxicology of diesel emissions. Proc. of the Symp. on the Use of Diesel-Powered Equipment in Underground Mining. Pittsburgh, PA, January 30-31, 1973. pp. 147-157. Bureau of Mines Information Circular 8666. U.S. Department of the Interior, Washington, DC.

Stumpp, G. and W. Banshaf. 1978. An Exhaust Gas Recirculation System for Diesel Engines. SAE Paper No. 780222. Society of Automotive Engineers, Warrendale, PA. (as cited in Samby and Johnson 1979).

Sutton G.W. 1975. Industrial hygiene survey of U.S. mines. Proc. of the Symp. on the Use of Diesel-Powered Equipment in Underground Mining. Pittsburgh, PA, January 30-31, 1973. pp. 171-219. Bureau of Mines Information Circular 8666. U.S. Department of the Interior, Washington, DC.

Swenberg, J.A., W.D. Kerns, R.I. Mitchell, E.J. Gralla and K.L. Pavkov. 1980. Induction of squamous cell carcinomas of the rat nasal cavity by inhalation exposure to formaldehyde vapor. Cancer Research 40:3398-3401.

SWRI. 1983. Southwest Research Institute. Clean burning diesel engines. Monthly progress report to the U.S. Army Fuels and Lubricants Research Laboratory. May 9, 1983. Southwest Research Institute, San Antonio, TX.

Taigel, P.G. 1951-52. Diesel Locomotives in Mines: The Production of Toxic Gases by the Diesel Engine. Trans. Inst. Min. Eng., London 111:85-108.

Tanner, R.L. and L. Newman. 1976. The analysis of airborne sulfate. A critical review. J. Air Poll. Control Assoc. 26(8):737-747.

Task Group on Lung Dynamics. 1966. Deposition and retention models for internal dosimetry of the human respiratory tract. Health Phys. 12:173-207.

Tejada, S.B., J. Sigsby, and R. Bardow. 1979. Determination of soluble sulfates in automobile exhaust by automated HPLC modification of the barium chloranilate method. In Analytical Procedures for Categorizing Unregulated Pollutant Emissions from Motor Vehicles. pp. 454-472. EPA Rept. No. 600-2-79-017. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Thomas, M.D., R.H. Hendricks, F.D. Gunn, J. Critchlow. 1958. Prolonged exposure of guinea pigs to sulfuric acid aerosol. Arch. Ind. Health 17:70-80.

Torpey, P.M., M.J. Whitehead, and M.J. Wright. 1971. Experiments in the control of diesel emissions. In Conf. on Air Pollution Control in Transport Engines. Inst. of Mech. Eng., November, 1971. (as cited in Samby and Johnson 1979).

Treon, J.F., F.R. Dutra, J. Cappel, H. Sigmon, and W. Younker. 1950. Toxicity of sulfuric acid mist. Arch. Indust. Hyg. Occup. Med. 2:716-734.

Truex, T.J., W.R. Pierson, and D.E. McKee. 1980. Sulfate in diesel exhaust. Environ. Sci. Technol. 14(9):1118-1121.

- Vogh, J.W. 1969. Nature of odor components in diesel exhaust. J. Air Pollut. Control Assoc. 19:773-777.
- Vostal, J.J. 1980. Health aspects of diesel exhaust particulate emissions. Bull. N.Y. Acad. Med. 56:914-934.
- Wark, K. and C.F. Warner. 1976. Control of sulfur dioxide. In Air Pollution - Its Origin and Control. Chapter 7, pp. 327-335. IEP - Dun Donnelly Publisher, New York.
- Weber-Tschopp, A., T. Fischer, R. Gierer, and E. Grandjean. 1977. Experimental irritating effects of acrolein on man. Int. Arch. Occup. Environ. Health 40(2):117-130.
- WHO. 1977. World Health Organization. Environmental Health Criteria 4: Oxides of Nitrogen. World Health Organization, Geneva, Switzerland.
- WHO. 1979a. World Health Organization. Environmental Health Criteria 13: Carbon Monoxide. World Health Organization, Geneva, Switzerland. 125 pp.
- WHO. 1979b. World Health Organization. Environmental Health Criteria 8: Sulfur Oxides and Suspended Particulate Matter. World Health Organization, Geneva, Switzerland. 107 pp.
- Williams, R.L. 1982. Diesel particulate emissions: composition, concentration, and control. In J. Lewtas, ed. Toxicological Effects of Emissions from Diesel Engines. pp. 15-32. Elsevier Science Publishing Co., Inc., NY.
- Williams R.L. and D.P. Chock. 1980. Characterization of diesel particulate exposure. In W.E. Pepekko, R.M. Danner, and N.A. Clarke, eds. Health Effects of Diesel Emissions. EPA-600/9-80-057a, U.S. Environmental Protection Agency, Washington, DC.
- Wolff, R.K., S.A. Silbaugh, D.G. Brownstein, R.L. Carpenter, and J. L. Mauderly. 1979. Toxicity of 0.4 and 0.8 μ m sulfuric acid aerosols in the guinea pig. J. Toxicol. Environ. Health 5:1037-1047.
- Wood, W.P., A.W. Castleman, Jr., and I.N. Tang. 1975. Mechanisms of aerosol formation from SO₂. J. Aerosol Sci. 6:367.

APPENDIX A
REVIEW OF KEY LITERATURE

APPENDIX A-1

REVIEW AND ANALYSIS OF PERTINENT REFERENCES
ON THE REGULATORY ASPECTS OF
DIESEL ENGINE EMISSIONS

ACGIH. 1982. American Conference of Governmental Industrial Hygienists. TLVs. Threshold limit values for chemical substances in work air adopted by ACGIH for 1982. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.

Review:

Threshold limit values are given for the following regulated components of diesel engine emissions: carbon monoxide, 50 ppm 8-hr TWA and 400-ppm STEL (short-term exposure limit); formaldehyde, 2-ppm ceiling value, nitric oxide 25-ppm 8-hr TWA and 35-ppm STEL, and nitrogen dioxide 3-ppm 8-hr TWA and 5-ppm STEL. TLVs for unregulated diesel exhaust components include those for acetaldehyde (100-ppm 8-hr TWA and 150-ppm STEL), acrolein (0.1-ppm TWA and 0.3-ppm STEL), crotonaldehyde (2-ppm TWA and 6-ppm STEL), sulfur dioxide (2-ppm TWA and 5-ppm STEL), and sulfuric acid (1 mg/m³ TWA). There are no ACGIH recommendations for limiting exposures to total aldehydes, total hydrocarbons, or total particulates in diesel exhaust.

Analysis:

The STEL values given by ACGIH are 15-min time weighted averages which supplement the 8-hr TWA values. The STELs should not be exceeded at any time during a work day even if the 8-hr TWA is within the recommended limits. The STELs are designed to ensure that workers do not suffer from contact irritation, chronic or irreversible tissue change, or narcosis as a result of short-term exposures.

EPA. 1980c. U.S. Environmental Protection Agency. Control of air pollution from new motor vehicles and motor vehicle engines. Gaseous emission regulations for 1984 and later model year light-duty trucks. Federal Register 45(188):63734-63784.

Review:

This ruling establishes new emission standards for 1984 and later light-duty trucks (under 6000 lb). The new standard for hydrocarbons will be 0.8 g/mi, and that for CO, 10 g/mi. CO emissions during vehicle idle, as expressed in terms of raw exhaust concentration, will be limited to 0.47%. The NO_x standard for 1984 and later is 2.3 g/mi.

Analysis:

This ruling also contained specific 1985 standards for light duty trucks operated at high altitudes; however, these standards were modified in a later ruling (see EPA 1983).

EPA. 1980d. U.S. Environmental Protection Agency. Control of air pollution from new motor vehicles and new motor vehicle engines: Final

high-altitude emission standards for 1982 and 1983 model year light-duty vehicles. Federal Register 45(197):66984-67015.

Review:

The emission standards for 1982 and 1983 model light-duty trucks operated at altitudes above 4000 ft are: 2.0 g/mi of total hydrocarbons, 26 g/mi of carbon monoxide, and 2.3 g/mi of NO_x. No standard was established for particulates.

Analysis:

The standards are designed to provide the same proportional decrease in emissions as those in effect for light-duty trucks operated at low altitudes.

EPA. 1983. U.S. Environmental Protection Agency. High-altitude emission standards for 1984 and later model year light-duty trucks: Final rule. Federal Register 48(8):1418. January 12, 1983.

Review.

This final ruling establishes the 1984 and later year emission standards for light-duty trucks operated at high altitudes. The standards will be: 1.0 g/mi for total hydrocarbons, 14 g/mi for carbon monoxide, and 2.3 g/mi for NO_x. No standard was set for particulates.

Analysis:

The high altitude standards were derived from the low altitude standards by using a proportional factor such that the same percentage reduction in emissions would be achieved. The NO_x standard is the same regardless of altitude because NO_x emissions from uncontrolled vehicles normally decrease with increasing altitude.

MSHA. 1982. Mine Safety and Health Administration. Mobile diesel-powered equipment for noncoal mines. Code of Federal Regulations, Part 32, revised July 1, 1982. Mine Safety and Health Administration, Washington, DC.

Review:

In order to safeguard the health of underground miners, the Mine Safety and Health Administration (MSHA) has established emission standards for diesel-powered equipment. For noncoal underground mining the standard for total aldehydes is 10 ppm (after exhaust dilution and measured as formaldehyde equivalents). There are no standards for specific aldehydes. For carbon monoxide the MSHA standard is 2500 ppm in undiluted diesel exhaust and 100 ppm after dilution. The latter value is

also the maximum permissible occupational exposure limit recognized by MSHA. In mines having 0.25% or more flammable gas, the emission standard is 100 ppm and the exposure limit is 50 ppm. For total nitrogen oxides the MSHA standard is 25 ppm after dilution, and the exposure limit is also 25 ppm. For mines containing flammable gas the emission standard is 200 ppm in undiluted exhaust and 25 ppm in diluted exhaust, and the occupational exposure limit is 25 ppm. MSHA does not have any emission standards or exposure limits for total hydrocarbons or total particulate in diesel exhaust.

Analysis:

In order to ensure that its emission standards and exposure limits are met by industry, MSHA has established an engine certification program. For each engine certified MSHA has determined the minimum mine ventilation rate that must be maintained to keep the regulated emissions at safe levels. Although no specific data are available, it is likely that these ventilation requirements would also have the effect of reducing the concentrations of other unregulated, pollutants, such as total hydrocarbons, particulates, and SO₂, to safe levels.

NIOSH. 1982. National Institute of Occupational Safety and Health. Summary of NIOSH recommendations for Occupational Health Standards. Draft report. National Institute of Occupational Safety and Health, Cincinnati, OH.

Review:

NIOSH periodically revises Criteria Documents and prepares special Hazard Reviews for the Occupational Safety and Health Administration of the Department of Labor. This document summarizes in tabular form recent changes in NIOSH recommendations, lists the current OSHA standards, and briefly describes the major health effects of each of the chemicals. There are no NIOSH recommendations for limiting occupational exposures to total aldehydes, total hydrocarbons, or diesel engine particulates. For carbon monoxide the recommended 10-hr TWA is given as 35 ppm (40 mg/m³ with a ceiling value of 200 ppm (229 mg/m³). No recommendation is given for exposures to total nitrogen oxides; however, for NO₂ the 15 min ceiling value is given as 1 ppm or 1.8 mg/m³, and for NO the 10-hr TWA is given as 25 ppm.

Analysis:

The NIOSH recommendation for carbon monoxide is based on potential health effects relating to the heart; that for NO is based on adverse effects to the blood; and that for NO₂ is based on potential effects on the respiratory system. NIOSH does not have recommendations for limiting exposures to diesel exhaust, nor has it considered the potential interactive health effects of various components of diesel exhaust.

OSHA. 1982. Occupational Safety and Health Administration, U.S. Department of Labor. Toxic and Hazardous Substances. U.S. Code of Federal Regulations, Title 29, revised July 1, 1982. Washington, DC.

Review:

The OSHA standards for occupational exposure to air contaminants are given in this part of the U.S. Code of Federal Regulations. Of the regulated components of diesel engine emissions, only carbon monoxide (CO), nitric oxide (NO), and nitrogen dioxide (NO₂) have OSHA exposure limits; 8-hr TWAs are 55 ppm for CO and 25 ppm for NO. There is no 8-hr TWA for NO₂; however, OSHA has a 5-ppm ceiling value for this compound. Unregulated components of diesel emissions for which there are OSHA exposure limits include acetaldehyde (200-ppm 8-hr TWA), acrolein (1-ppm 8-hr TWA), crotonaldehyde (2-ppm 8-hr TWA), formaldehyde (3-ppm 8-hr TWA, 5-ppm ceiling value, 10-ppm 30-min maximum peak above the ceiling value), sulfur dioxide (5-ppm 8-hr TWA), and sulfuric acid (1 mg/m³ 8-hr TWA).

Analysis:

In most cases the OSHA exposure limits are based on 8-hr time weighted averages; consequently, short-term exposures of 30 min or less can be considerably higher before the TWAs are exceeded. There are, however, no data indicating that the maximum safe 30-min exposure limits are directly proportional to the 8-hr TWAs.

APPENDIX A-2

**REVIEW AND ANALYSIS OF PERTINENT REFERENCES
ON THE IDENTIFICATION AND QUANTIFICATION
OF CHEMICALS IN DIESEL ENGINE EMISSIONS**

DEUTZ. 1982. Klocker-Humboldt-Deutz AG. Air-cooled diesel engines, FL 912, BF 6L913. Klocker-Humboldt-Deutz AG, Cologne, West Germany.

Review

This is a manufacturer's brochure which describes two series of Deutz diesel engines. Both series come in direct injection or two-stage combustion models. Emission rates of primary pollutants from the direct injection model of both engine types are compared graphically with those from another Deutz two-stage combustion model (F 8L 413W), a gasoline-powered and a natural-gas-powered engine. The emission rates of CO were reported to be 4.0 g/hp-hr for the direct injection engines and 1.8 g/hp-hr for the for the two stage combustion engine. For total hydrocarbons the rates were 2.5 g/hp-hr (direct injection) and 0.4 g/hp-hr (two stage combustion), and for NO_x the rates were 8.5 g/hp-hr (direct injection) and 4.7 g/hp-hr (two stage combustion).

Analysis

The data as presented do not allow for a comparison of emission rates at different engine speeds and loads. SWRI (1983) has tested the Deutz F 3L 912W diesel and has reported that at very low loads the emission rates can be quite high (e.g., at 2% load CO was 14.79-86.05 g/hp-hr; HC was 5.14-19.24 g/hp-hr; and NO_x was 27.11-126.30 g/hp-hr); however, at high loads the rates drop to below those reported by the manufacturer for the F 8L 413W engine (e.g., at 100% load, CO was 0.55-0.94 g/hp-hr; HC was 0.14-0.25 g/hp-hr, and NO_x was 2.41-3.09 g/hp-hr). Thus, the rate of buildup of air pollutants in a confined space situation would depend considerably on engine operating conditions.

Faulds, A.J., Z. Waszczylo, and K.C. Westaway. 1981. Polynuclear aromatic hydrocarbons in the underground mine environment. CIM Bulletin. 74(835):84-90.

Review

Air in underground mines in which diesel engines were being used was sampled by means of Hi Vol filters. The particulate matter on the filters was extracted with benzene and methanol, and the extracts were analyzed for polynuclear aromatic hydrocarbons (PAHs) by means of thin layer chromatography and spectrofluorometry. Thirty PAHs were separated from the samples, and eleven of these were identified and quantified. Concentrations were as high as 429.70 ng/m³ for pyrene, 72.75 ng/m³ for fluoranthene, 40.33 ng/m³ for benzo(a)anthracene, 38.87 ng/m³ for benzo(e)pyrene, 1.47 ng/m³ for perylene, 7.49 ng/m³ for benzo(k)fluoranthene, 8.37 ng/m³ for benzo(a)pyrene, 15.39 ng/m³ for benzo(ghi)perylene, 1.98 ng/m³ for dibenzo(def,mno)chrysene, 12.65 ng/m³ for naphtho(1234,def)chrysene, and 3.99 ng/m³ for benzo(rst)pentaphene. Seven of these compounds are known carcinogens.

Analysis

The authors note that the concentration of PAHs in the mines was related to diesel activity, type of diesel engine and/or type of fuel used, amount of ventilation, and type of mine from which the air samples were taken. Adding a PTX catalytic converter to the exhaust system of a diesel, however, did not appear to affect the PAH levels markedly. It was also noted that the concentrations found were lower than those reported for other industrial operations and even for some atmospheric samples taken over some large cities.

Hites, R.A., Y. Ming-Li, W.G. Thilly. 1980. Compounds associated with diesel exhaust particulates. In M. Cooke and A.J. Dennis, eds. Polynuclear Aromatic Hydrocarbons. Chemical Analysis and Biological Fate. Battelle Press, Columbus, OH.

Review

A particulate sample was filtered from the exhaust of a 350 in³ Oldsmobile diesel engine, and then extracted with methylene chloride for 16 hr. The crude extract was fractionated on a silicic acid column using seven different eluates. Chemical analyses (gas chromatography/flame ionization detection) were conducted on two of the fractions; one was eluted with a 1:1 mixture of hexane and toluene and another with pure toluene. Polynuclear aromatic hydrocarbons (PAHs) were found in the hexane/toluene fraction, and oxygenated polycyclic compounds such as aldehydes and ketones were found in the other fraction. The PAHs were compounds containing three to five aromatic rings. Phenanthrene and alkylated phenanthrenes were the major components. Fluoranthene, pyrene and their methyl homologs, and alkyl fluorenes were also detected. Phenylanthralene and its C₁, C₂, and C₃ alkyl homologs were also tentatively identified in this fraction. The aldehydes found in the toluene fraction included naphthaldehyde and its alkyl homologs, phenanthrene-carboxaldehydes and their alkyl homologs, biphenylcarboxaldehydes and their C₁ and C₂ alkyl homologs and pyrenecarboxaldehyde and fluoranthene-carboxaldehyde. The most common ketones found in the toluene fraction were fluorenone and alkylated fluorenones. Also found were the diketones 9,10-anthracenedione and methyl-9,10-anthracenedione.

Analysis

This study does not provide quantitative data on PAHs in diesel particulates except in indicating which compounds or groups of compounds were relatively more abundant. The benzopyrenes, well-known mutagens, were detected at only very low levels; however, the mutagenic methylated phenanthrenes and methylated fluorenes were relatively more common.

Lawson, A. 1981. Progress in the control of underground diesel emissions. CIM Bulletin 74(835):68-73.

Review

This paper reviews several methods for reducing emissions from diesel engines. The effect of an Engelhard PTX catalyst on emissions of carbon monoxide, hydrocarbons, and particulates at different engine speeds and torques is illustrated graphically for an unidentified engine. Without the catalyst CO emissions ranged from about 27 to 63 g/hr, but with the catalyst they were below 10 g/hr. Total hydrocarbon emissions, when uncontrolled, ranged from about 4 to 15 g/hr, but were reduced to 2 to 7 g/hr by the catalyst. Total particulates (cyclohexane insoluble and soluble organic components) were not substantially altered by the catalyst, however, there was a progressive increase in the conversion of SO₂ to SO₄ with increasing catalyst temperature and fuel/air ratio. Percent conversion rose from about 7% at temperatures below 200°C to above 40% at temperatures above 350°C.

The effectiveness of a fiberglass filter in reducing particulate emissions was evaluated on a Deutz F 6L 714 and a Deutz F 8L 413 engine. With the first engine the exhaust gas was cooled upstream of the filter by water injection. The trapping efficiency of the filter ranged from 70 to 90%. In the second engine the efficiency was 60 to 80%.

Exhaust gas recirculation tests with a Deutz engine indicated that NO_x emissions could be reduced by 60%; however, there were also substantial increases in particulate, CO, and gaseous HC emissions. Twenty percent exhaust recirculation at full load resulted in a 700% increase in particulates, a 600% increase in CO, and a 100% increase in gaseous hydrocarbons.

Use of a 15% water/fuel emulsion resulted in a 40 to 50% reduction in NO_x and particulates when tested in a Deutz F 6L 714 diesel; however, when tested in a Detroit Diesel 8V 71N, unacceptably large amounts of water were needed before effective reductions in NO_x were achieved.

When the water/fuel emulsion was tested in a Deutz F 6L 714 diesel fitted with a catalyst and an exhaust filter, NO was reduced by about 35%, total hydrocarbons by about 90%, CO by about 90%, and particulates by over 95%. When the same engine was fitted with an exhaust gas recirculation system (20% of exhaust recirculated), a catalyst, and an exhaust filter and run on standard diesel fuel, all emission components were reduced by 50% or more, and over 95% of the H₂SO₄ was removed by the filter.

Analysis

This review, which covers the results of several earlier studies, clearly demonstrates that a combination of control techniques is needed to effectively reduce the levels of diesel emissions without at the same time causing a large increase in any one component such as NO_x or H₂SO₄. Further studies of this type, on direct injection and two-stage combustion diesel engines, would be very useful in determining the maximum effectiveness of such controls.

Levins, P.L. 1981. Review of Diesel Odor and Toxic Vapor Emissions. Report No. DOT-HS-805 831. U.S. Department of Transportation, Washington, DC.

Review

This report reviews the pre-1978 information available on the analysis and characterization of diesel engine exhaust components, including odor, irritants, toxic chemicals, and polynuclear aromatic hydrocarbons. Diesel exhaust may contain 100-3000 ppm CO (normally 300-500 ppm); 2.4-8.7% CO₂; 100-1200 ppm NO_x (generally 500-700 ppm and consisting mainly of NO with 10-50 ppm NO₂); 40 ppm SO₂ (including 2-4 ppm as sulfuric acid); <0.05 to 10.7 ppm light hydrocarbons (C₁-C₇), consisting primarily of ethylene, acetylene, and propylene; 1-30 ppm total aldehydes (typical values 15-20 ppm, including 3-7 ppm formaldehyde and 0.3-3 ppm acrolein); 100 µg/m³ total phenols; 25 mg/m³ odorants; polynuclear aromatic hydrocarbons, including phenanthrenes, pyrenes, fluoranthenes, pyrenes, chrysenes, and perylenes; and particulates containing aromatics and polynuclear aromatics and their oxidized derivatives.

Analysis

This report provides a good summary of diesel emissions data but does not contain information on specific engines or operating conditions.

Marshall, W.F. 1975. Emission Control for Diesels Operated Underground: Catalytic Converters. Report BERC/RI-75/8. Bartlesville Energy Research Center, Bartlesville, OK.

Review

Three catalyst systems (two platinum and one nickel) were tested in one or more of four different diesel engines [2-stroke, direct injection; 4-stroke (air cooled) indirect injection; 4-stroke, Lanova combustion system; 4-stroke, indirect injection using one or more of four different fuels (sulfur content 0.15, 0.30, 0.88, and 1.04%). The platinum catalysts were much more efficient than the nickel catalyst in oxidizing CO and HC. Both CO and HC were significantly reduced when exhaust gas temperature exceeded 600°F. The platinum catalyst was most effective when used in a pelletized form as compared with a monolithic construction. Exhaust gas odors were substantially reduced by the platinum catalysts; however, emissions of SO₃ increased almost tenfold when the engine was run at full power and exhaust temperatures exceeded 1000°F. SO₃ concentration varied with sulfur content of the fuel used and approached 120 ppm when the sulfur levels were 0.88 and 1.0%.

Analysis

Because of the increased emissions of SO₃ with the use of catalytic converters, additional exhaust controls, such as water scrubbers or exhaust filters would be needed to avoid a dangerous buildup of sulfur oxides.

Marshall, W.F. 1978. NO₂ Levels in Diesel exhaust. BERC/TPR-78/1. Bartlesville Energy Research Center, Bartlesville, OK.

Review

Nitrogen dioxide was measured in the exhaust from five diesel engines operated over a wide range of speeds and loads. The measurements were made with an online chemiluminescence analyzer. The engines tested were 50 to 150 bhp, 2-stroke or 4-stroke, and direct injection or two-stage combustion. A catalyst and water scrubber were also tested on one engine. NO₂ concentrations varied from less than 10% to about 30% of the total oxides of nitrogen (data not given). Maximum relative NO₂ (ratio of NO₂ to NO_x) emissions occurred at light loads and decreased with increasing load. The catalytic reactor and water scrubber decreased relative NO₂ emissions only slightly and had no significant effect on total NO_x levels. Relative NO₂ emissions were slightly lower in the two-stage combustion engines than in the direct injection engines. In all cases the NO₂ fraction in the exhaust was a function of exhaust gas temperatures, with increasing temperatures decreasing the relative NO₂ emissions.

Analysis

The authors calculated that, for the NO₂ levels in the emissions of the five tested engines to be reduced to the NO₂ threshold limit value (TLV) of 5 ppm, a ventilation rate of 12 to 52 cfm/rated hp would be needed. Since these rates are generally below those for achieving the TLV levels for NO (43-133 cfm/rated hp), CO (13-150 cfm/rated hp), and CO₂ (36-50 cfm/rated hp), it is assumed that NO₂ should not be a problem if one or more of these other air contaminants is used for monitoring and ventilation control.

Marshall, W.F., R.W. Hurn. 1973. Hazard from Engines Rebreathing Exhaust in Confined Space. Bureau of Mines Report of Investigations, BM-RI 7757. U.S. Bureau of Mines, Washington, DC.

Review

Diesel and gasoline engines were tested under conditions that simulated confined space operations. The diesel engines tested were a 2-stroke direct injection model and a 4-stroke indirect injection model. For each test, exhaust levels of CO, CO₂, and NO were determined. In a

nonventilated space, with the engines running at constant speed and load, there was a linear decrease in O₂ and a linear increase in CO₂ which were proportional to the fuel consumed per unit of confined space volume. CO concentration increased in a nearly linear manner for a short period (15-20 min) but then increased almost exponentially such that at 25 min it was nearly 5000 ppm and at 30 min almost 10,000 ppm. A CO level of 5000 ppm is fatal in a matter of a few minutes. The rate of CO buildup was only slightly dependent on engine type but highly dependent on power output. Critical levels of CO (5000 ppm) were reached at a time corresponding to 1.5 to 3.0 lb fuel consumed per 1000 ft³ of confined space volume. It was calculated that this would be about 10 min for an engine delivering 100 hp in a 5000 ft³ enclosure.

Both engines were also tested in such a way that part of the exhaust gas was directed to a chamber where it was mixed with fresh air and then piped back into the engine intake. The CO level in the exhaust of the two-stroke, direct injection engine at idle did not change when exhaust recirculation was increased (0-20%); at 1/2 or 3/4 power it increased slightly with exhaust recirculation of 10% and above; and at full power it increased almost exponentially with increasing exhaust recirculation (ca. 400 ppm CO at 0%, >800 ppm at 10% and ca. 1400 ppm at 15%). A similar pattern was seen in the tests in the indirect injection engine.

Using as a maximum allowable intake air composition a CO₂ level of 0.5% (TLV for CO₂), the authors calculated that the CO levels would be unacceptably high for the direct injection engine operating under full load. CO levels in the indirect injection engine would be satisfactorily low, but NO_x would be above the TLV.

Analysis

This study illustrates the critical need for careful monitoring of primary gaseous pollutants from diesel engines operated in confined spaces with little or no ventilation.

Sheehy, J.W. 1980. Evaluation of Exhaust Emissions Data for Diesel Engines Used in Underground Mines. DDHS (NIOSH) Publication No. 80-146. National Institute for Occupational Safety and Health, Cincinnati, OH.

Review

The emissions data evaluated in this report were collected by the Mine Enforcement and Safety Administration (now Mine Safety and Health Administration). Tests were conducted on a series of 4-, 6-, 8-, and 12-cylinder indirect injection diesel engines having no emission control devices. The results showed that NO emissions were higher, and CO emissions generally lower, in turbocharged after-cooled engines than in naturally aspirated engines (average NO levels were 4.8 and 5.7 g/bhp-hr, respectively; average CO levels were 1.5-7.6 g/bhp-hr in the naturally aspirated engines and 0.8 g/bhp-hr in one turbocharged after-cooled

engine tested). At 100% load CO emissions were similar in both types of engines. The number of engine cylinders had almost no effect on brake specific emissions of NO and CO; however, both components decreased considerably as horsepower increased. Furthermore, NO decreased with increasing fuel-to-air ratio up to the tested maximum of 0.050. CO emissions were lowest in the range of fuel-to-air ratios from 0.032 to 0.045. It was also found that brake specific emissions of NO when averaged over segments 3, 6, 9, and 10 of the EPA 13-mode test cycle were higher than NO levels at peak torque or at rated speed. For all engines tested average CO₂ emissions ranged from 580 to 812 g/bhp-hr.

Analysis

This study provides a very thorough analysis of the effects of engine speed, load, fuel/air ratio, and horsepower on the NO and CO emissions from indirect injection diesel engines of a size class that would be appropriate for use in fork lifts. Although the engines were not identified by manufacturer, the general conclusions reached would probably also apply to the two engines being considered for Army use.

SWRI. 1983. Southwest Research Institute. Clean Burning Diesel Engines. Monthly progress report to the U.S. Army Fuels and Lubricants Research Laboratory. May 9, 1983. Southwest Research Institute, San Antonio, TX.

Review

SWRI tested a Deutz F 3L 912W and a Perkins 4.203.2 diesel engine using the EPA 13-mode test cycle. Emissions data (raw exhaust) concentrations for the two engines were as follows (Deutz data given first, then data for the Perkins engine): 24-172, and 464-1280 ppm for HC, 54-371 and 195-1348 ppm for CO, 99-585 and 128-138 ppm for NO_x, 17.24-153.81 and 7.04-158.67 ppm for SO₂, 1.6-5.9 and 1.9-7.5 ppm for sulfates, 26-215 and 33-282 ppm for particulates, 0.07-0.38 and 0.08-3.17 ppm for formaldehyde, ND-0.07 and 0.06-1.55 ppm for acetaldehyde, ND-0.10 and ND-0.13 ppm for acrolein, ND-0.05 and ND-0.53 ppm for propionaldehyde, ND-0.05 and ND for acetone, ND-0.05 and ND-0.08 ppm for crotonaldehyde, ND-0.04 and ND-0.28 ppm for isobutyraldehyde, 0.05-0.34 and ND-0.20 ppm for methylethylketone, ND-0.06 and ND-0.15 ppm for benzaldehyde, and ND-0.01 and ND-0.48 ppm for hexanaldehyde. The ranges given above are for different engine speeds and loads. The data are also presented in the form of emission rates (g/hp-hr and g/hr).

Analysis

This report represents the preliminary results of bench tests on two clean burning diesel engines. The authors note that with a few exceptions, several generalizations can be made concerning the data. Both engines produced higher HC, CO, and NO_x at low load conditions, and with decreasing load when engine speed was constant. At constant load, HC and CO increased with increasing speed, but NO_x fluctuated

erratically. The Perkins engine had higher brake specific and mass emission rates of HC, CO, and NO_x than the Deutz engine. Particulate and SO₂ levels increased with increasing load when engine speed was constant, and sulfates increased with increasing load at constant, peak and torque and rated speeds. In most cases particulate brake specific emission rates were higher on the Perkins engine. SO₂ emissions were proportional to the fuel consumed. Formaldehyde, which accounted for over 50% of the aldehydes and ketones detected, was less than 0.5 ppm in the Deutz engine and less than 3.2 ppm for the Perkins. Brake specific aldehyde emissions increased with a decrease in load at constant speed in the Deutz engine, and, in general, aldehyde and ketone mass emission rates increased with an increase in speed when the load remained constant (except at low load for the Perkins). No organic sulfides were found in the exhaust of the Deutz engine but were present in that of the Perkins. Carbonyl sulfide, the predominant organic sulfide, showed an increased mass emission rate with increased engine speed at constant load, and a decreased mass emission rate with an increase in load at constant speed. Analysis of diesel odor indicated that the Perkins was generally higher than that of the Deutz.

APPENDIX A-3

REVIEW AND ANALYSIS OF PERTINENT
REFERENCES ON HEALTH EFFECTS OF ALDEHYDES

Alexandersson, R., G. Hedenstierna, and B. Kolmodin-Hedman. 1982. Exposure to formaldehyde: effects on pulmonary function. Arch. Environ. Health 37(5):279-284.

Review:

An epidemiological study was conducted on carpentry shop workers exposed to formaldehyde at a mean air concentration of 0.45 mg/m³. Formaldehyde concentrations were determined by liquid chromatography. When compared with controls, exposed workers had an increased incidence of eye, nose, and throat irritation, dyspnea during work, and chest oppression. By the end of the working day, exposed workers had significant reductions in lung function; including forced expiratory volume in 1 sec; percent change in forced expiratory volume; and maximum midexpiratory flow. Closing volume, expressed as percent of the expired vital capacity, increased significantly. The changes in lung function were similar in smokers and nonsmokers, and were attributed to bronchoconstriction. There was, however, little correlation between degree of lung function deterioration and level of exposure to formaldehyde.

Analysis:

The authors note that the employees were also exposed to terpenes and dust, but that the concentrations were too low to elicit the observed effects. The lack of correlation between exposure level and effect was attributed to either avoidance behavior of the employees when exposed to high formaldehyde concentrations, or to a dose-independent allergic or hyperreactive responses. There was no evidence of chronic adverse pulmonary effects in any of the workers. Other physiological tests, such as liver and kidney function, were not conducted.

Bird, R. P., P. H. Draper, and P. K. Basrur. 1982. Effect of malonaldehyde and acetaldehyde on cultured mammalian cells. Mutat. Res. 10:237-246.

Review:

Primary cultures of rat skin fibroblasts were exposed to acetaldehyde concentrations of 10⁻⁴ to 10⁻² M for 12, 24, or 48 hr in 2 mL of medium. The exposed cells showed a dose-dependent increase in the production of micronuclei. Twenty-four hour treatment with 10⁻³ M acetaldehyde resulted in a significant increase in the number of metaphases with chromosomal aberrations, including chromatid and chromosome breaks, gaps, deletions, and fragmentation. Four out of fifty metaphases contained translocation type aberrations.

Analysis:

This study substantiates the results of earlier ones which have shown that acetaldehyde is genotoxic in vitro. Potential genotoxic effects under in vivo conditions require further study.

Dalbey, W. E. 1982. Formaldehyde and tumors in hamster respiratory tract. Toxicol. 24:9-14.

Review:

Male Syrian golden hamsters were exposed to 10 ppm formaldehyde 5 hr/day, 5 days/wk for their entire lifetime. Exposed animals had significantly shorter survival times ($p < 0.05$) relative to unexposed controls. No tumors were observed in the respiratory tract of either exposed animals or controls. The incidence of rhinitis was 24% in exposed animals and 31% in controls, and the incidence of hyperplastic and metaplastic lesions in the nasal epithelium was 5% in the exposed animals and 0% in the controls. In a second experiment, hamsters were exposed once per week to 30 ppm formaldehyde (5 hr/day) for their entire lifetime. This exposure level did not result in any respiratory tract tumors. However, when the formaldehyde exposures were followed two days later by weekly injections of the carcinogen diethylnitrosamine (for 10 wk), the incidence of tracheal tumors per tumor-bearing-animal was higher than that for animals exposed to diethylnitrosamine alone.

Analysis:

The results of this study differ from those in which formaldehyde was found to cause tumors in the nasal cavity of rats. Furthermore the lower incidence of hyperplastic and metaplastic lesions in hamsters when compared to that in rats suggests that this species may be more resistant to the irritant effects of the aldehyde.

Egle, J. L. 1972. Effects of inhaled acetaldehyde and propionaldehyde on blood pressure and heart rate. Toxicol. Appl. Pharmacol. 23:131-135.

Review:

Male Wistar rats were anesthetized with sodium pentobarbital and then exposed to vapors of either acetaldehyde (0.5-30 $\mu\text{g}/\text{mL}$) or propionaldehyde (3.0-200 $\mu\text{g}/\text{mL}$) for 1-min intervals while changes in arterial blood pressure and heart rate were measured. At concentrations of 0.5 and 1.0 $\mu\text{g}/\text{mL}$, acetaldehyde caused no changes in either parameter. All higher concentrations caused a significant increase in blood pressure, and concentrations of 12 and 25 $\mu\text{g}/\text{mL}$ (but not 30 $\mu\text{g}/\text{mL}$) significantly increased heart rate. For propionaldehyde, significant increases in blood pressure were seen at exposure levels of 10 $\mu\text{g}/\text{mL}$ and above. Heart rate increased significantly at 20 and 30 $\mu\text{g}/\text{mL}$ but showed a significant decrease at 200 $\mu\text{g}/\text{mL}$.

Analysis:

The variability in the responses may have been due to the differences in how well anesthetized the animals were, and to the fact that the

sodium pentobarbital may have interacted with the aldehydes and altered the response.

Feron, V. J., A. Kruyse, and R. A. Woutersen. 1982. Respiratory tract tumors in hamsters exposed to acetaldehyde vapor alone or simultaneously to benzo(a)pyrene or diethylnitrosamine. Eur. J. Cancer Clin. Oncol. 18(1):13-31.

Review:

Hamsters were exposed to acetaldehyde vapors 7 hr/day, 5 days/wk, for 52 weeks. Exposure levels were 2500 ppm during the first 9 weeks, 2250 ppm during weeks 10-20, 2000 ppm during weeks 21-29, 1800 ppm during weeks 30-44, and 1650 ppm during weeks 45-52. The study was terminated after 81 weeks. Hamsters exposed to acetaldehyde had substantially lower body weights and higher relative kidney and liver weights than controls. There were no significant differences in hematological and biochemical parameters except for a slight increase in alkaline phosphatase activity in females exposed to the aldehyde. Pathological changes seen in exposed animals included rhinitis, hyperplasia and metaplasia of the nasal, laryngeal, and tracheal epithelium, and nasal and laryngeal carcinomas. The incidence of nasal carcinomas was 7% in males and 4% in females; that of laryngeal carcinomas was 26% in males and 20% in females. Only the incidence of laryngeal tumors was significantly different from that of the controls. No tracheal tumors were found in the exposed animals, and there were no changes in the bronchi or lungs that could be attributed to acetaldehyde.

Analysis:

This study demonstrates that chronic exposure to high doses of acetaldehyde results in the formation of nasal and laryngeal carcinomas; however, the high exposure levels required to produce the observed effects also caused considerable direct damage to the respiratory epithelia. Further study is needed to determine if chronic exposures to low levels of acetaldehyde would be carcinogenic.

Florin, I., L. Rutberg, M. Curvall, and C. R. Enzell. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames test. Toxicol. 15(3):219-232.

Review:

Four aldehydes were assayed for mutagenicity with Salmonella typhimurium strains TA 98, TA 100, TA 1535, and TA 1537. The tests were conducted with and without metabolic activation (rat liver S-9 fraction). When tested at 3 μ mole per plate, benzaldehyde, propionaldehyde and butyraldehyde were not mutagenic to any of the bacterial strains with and without metabolic activation. Acrolein was highly toxic to the bacteria

at 3 μ mole per plate. When retested at 0.3 μ mole per plate it was not mutagenic.

Analysis:

This study used the standard Ames procedure, a method which, with a few exceptions, provides a reliable estimate of potential mutagenicity. However, another study using a modified Ames assay has indicated that acrolein is mutagenic to TA 100 (see Lutz et al. 1982).

Holtzman, S. G. and E. D. Schneider. 1974. Comparison of acetaldehyde and ethanol: depression of motor activity in mice. Life Sci. 14:1243-1250.

Review:

The fine and gross motor activity of CF #1 (Carworth) mice was measured at 1-min intervals for 15 min after intravenous injection of various concentrations of acetaldehyde. Gross motor activity was significantly reduced by as little as 3.0 mg/kg and fine motor activity by 10 mg/kg in the first minute after administration. The median effective doses (ED₅₀) were 2.2 and 2.7 mg/kg for 50% depression of fine and gross motor activity, respectively.

Analysis:

This study demonstrates that behavior changes can result from acute iv exposure to acetaldehyde; however, there is insufficient information to determine if chronic exposure to low levels of acetaldehyde vapors might have a similar effect.

Jensen, O. M. and S. K. Anderson. 1982. Lung cancer risk from formaldehyde. Lancet 1:913.

Review:

An epidemiological study was conducted on the association between formaldehyde exposure and lung cancer among Danish physicians. Physicians occupationally exposed to formaldehyde did not have a greater risk of lung cancer relative to controls (8/23) than those never exposed (69/202).

Analysis:

Although the results are based on limited sample size, and allow for no correlation with exposure levels, they do support the results of other epidemiological studies.

Kane, L. E. and Y. Alarie. 1977. Sensory irritation to formaldehyde and acrolein during single and repeated exposures in mice. J. Am. Ind. Hyg. Assoc. 38(10):509-522.

Review:

Male Swiss Webster mice were exposed to a range of concentrations of formaldehyde and acrolein vapors, and the resulting changes in respiratory rate were recorded. Acrolein was measured spectrophotometrically after reaction with 4-hexylresorcinol, and formaldehyde was measured spectrophotometrically after reaction with 4,5-dihydroxy-2,7-naphthalene disulfonic acid.

For a single exposure to acrolein, a 50% reduction in respiratory rate occurred at a concentration of 1.7 ppm. For formaldehyde, this same effect occurred at 3.1 ppm. Mice were also exposed to each aldehyde for 3 hr each day for 4 consecutive days. Repeated exposures to high concentrations (>0.5 ppm) resulted in sensitization, whereas low concentrations (0.31 ppm for formaldehyde and 0.17 ppm for acrolein) resulted in an increased tolerance to acrolein, but no change in response to formaldehyde.

Analysis:

The use of a 50% level of reduction in respiratory rate appears to be a valid way to standardize measurements of response to aldehydes; however, it should be noted that the magnitude of the difference between the ED50 doses for these two aldehydes is much less than would be expected from other toxicological studies.

Kane, L. E. and Y. Alarie. 1978. Evaluation of sensory irritation from acrolein-formaldehyde mixtures. J. Am. Ind. Hyg. Assoc. 39(4):270-274.

Review:

Swiss-Webster mice, in groups of four, were exposed to mixtures of formaldehyde and acrolein vapors, and changes in respiratory rate were monitored by means of a body plethysmograph. The exposure periods were 10 min long, and the response was measured as maximum percent decrease in respiratory rate. A 50% decrease in rate occurred at 3.1 ppm formaldehyde. A similar median effect occurred at 1.7 ppm acrolein. For a series of mixtures of the two aldehydes, the responses seen were not additive, but rather followed a model of competitive agonism.

Analysis:

The authors note that their results show that total sensory response was not simply a function of total aldehyde present but rather a function of the specific concentrations of the acrolein and formaldehyde.

Although this study was based on only one exposure time (10 min), the results would most likely be applicable to longer periods.

Kane, L. E. and Y. Alarie. 1979. Interactions of sulfur dioxide and acrolein as sensory irritants. Toxicol. Appl. Pharmacol. 48(2):305-315.

Review:

Male Swiss-Webster mice were exposed to various mixtures of sulfur dioxide and acrolein vapors, and changes in respiratory rate were measured before, during, and after each exposure. The results were expressed in terms of maximum percentage decrease in respiratory rate. Changes in response pattern during and after the exposures were also recorded. A ratio dependent decrease in the respiratory rate was observed when both chemicals were present, but this response was always less than what would be expected if the effects were additive. There was a secondary response after the exposures had ended.

Analysis:

The authors noted that the observed effects might have been due to the formation of a less active acrolein bisulfite adduct, which disproportionated after the exposure ended, thereby releasing additional acrolein to cause a secondary response. The lowest concentration of acrolein used was 0.85 ppm, while that for SO₂ was 9 ppm. These levels caused a 17% reduction in respiratory rate (compared to a 48% expected decrease if the effects were additive).

Kruysse, A., V. J. Feron, and H. P. Til. 1975. Repeated exposure to acetaldehyde vapor. Studies in Syrian golden hamsters. Arch. Environ. Health 30:449-452.

Review:

The subacute inhalation toxicity of acetaldehyde was examined in hamsters exposed to 0, 390, 1,340, and 4,560 ppm for 6 hr per day, 5 days per week for a 90 day period. Exposures occurred at 22-24°C and with a relative humidity of 50-70%. Acetaldehyde was analyzed by gas chromatography.

Exposure to 390 ppm acetaldehyde resulted in no toxic effects. At 1340 ppm there was an increase in kidney weight in males and slight hyper- and metaplastic changes of the tracheal epithelium. The highest exposure level caused growth retardation, ocular and nasal irritation, increased numbers of erythrocytes, increased weights of heart and kidneys, and severe histopathological changes in the respiratory tract, including necrosis, inflammation, and hyperplasia and metaplasia of the

epithelium. The upper part of the respiratory tract was more severely damaged than the lower part.

Analysis:

The authors note that the severe injuries to the upper respiratory tract were not unlike the lesions produced by other irritants such as formaldehyde, sulfur dioxide, and peroxyacetyl nitrate. Thus, an evaluation of the potential health effects of chronic exposure to diesel emissions must take into account the potential additive effects of chemically unrelated respiratory irritants.

Lutz, D., E. Eder, T. Neudecker, and D. Henschler. 1982. Structure-mutagenicity relationship in α,β -unsaturated carbonylic compounds and their corresponding allylic alcohols. Mutat. Res. 93(2):305-315.

Review:

The mutagenicity of several carbonyl compounds was assayed by means of a modified, liquid cell suspension, Ames assay. The mutagenicity of acrolein was found to be extremely high (2400 histidine revertants per μ mole of chemical) in the absence of S-9 metabolic activation. However, when S-9 mix was added to the test system, the mutagenic rate dropped to zero. The related compound 2-methylacrolein gave a mutagenic rate of 460 revertants/ μ mole, and this was less affected by S-9 mix than was acrolein (the rate dropped to 220 revertants/ μ mole).

Analysis:

The authors note that the loss of mutagenicity of acrolein in the presence of S-9 mix may have been due to the extremely high cytotoxicity of the chemical and the use of only a single concentration of S-9 mix. They did not address the question as to whether this has any significance for the potential mutagenicity of acrolein in higher organisms.

Neudecker, T., D. Lutz, E. Eder, and D. Henschler. 1981. Crotonaldehyde is mutagenic in a modified Salmonella typhimurium mutagenicity testing system. Mutat. Res. 91:27-31.

Review:

A modified Ames assay was used to test the mutagenicity of crotonaldehyde to Salmonella typhimurium strain TA 100. Both the standard agar plate method and two cell suspension techniques were used. Crotonaldehyde concentrations ranged from 0.025 to 0.375 μ L per mL of incubation volume (0.5-1.5 μ mole/2 mL), and the exposures lasted 90 min. While the aldehyde was not mutagenic in the standard agar plate assay, it was mutagenic in both cell suspension systems. In one system the presence of

S-9 mix reduced the mutagenicity by more than 50%, and in both systems the dose-related increase in mutagenicity corresponded to a decrease in cell survival rate (see also Lutz et al. 1982).

Analysis:

The authors note that negative results were found for four other S. typhimurium strains (TA 98, TA 1535, TA 1537, TA 1538) tested in both the standard and modified Ames tests (data not given). Significantly, for TA 1535 the histidine reversion mechanism is identical to that which occurs in TA 100 (base substitution). The reason for the greater susceptibility of TA 100 is unknown. The reduction in the mutagenic effect by the S-9 mix was attributed to direct chemical interactions or enzyme-induced alterations in the aldehyde.

Obe, G. and B. Beck. 1979. Mutagenic activity of aldehydes. Drug Alcohol Depend. 4(1-2):91-94.

Review:

Human lymphocytes were treated in vitro with 1×10^{-4} to $1 \times 10^{-3}\%$ (v/v end concentration) of formaldehyde for 24-48 hr, following which the number of sister chromatid exchanges (SCE) per mitosis was determined. At the highest dose there were 12.07-16.75 SCEs per mitosis as compared with 4.19-4.58 for the controls.

Analysis:

This study demonstrates that the mutagenic activity of formaldehyde can occur in human cells in vitro; however, the data are insufficient to extrapolate to in vivo situations involving inhalation exposures. More information is needed concerning the actual levels of formaldehyde in the blood resulting from chronic occupational exposure.

Schmid, B. P., E. Goulding, K. Kitchin, and M. K. Sanyal. 1981. Assessment of the teratogenic potential of acrolein and cyclophosphamide in a rat embryo culture system. Toxicol. 22:235-243.

Review:

Rat embryos, within the yolk sacs, were removed from the uterus on day 10.5 of gestation and transferred to a culture medium containing pure rat serum with and without liver microsomes and NADPH and with various concentrations of acrolein. Exposures lasted 48 hr, after which morphological development was evaluated. Acrolein-treated embryos showed slight but significant inhibition of growth at 100 μM and 150 μM . Dose levels of 200 and 250 μM resulted in drastic inhibition of growth and differentiation but no morphological abnormalities.

Analysis:

The absence of acrolein-related teratogenic effects seen in this study on rat embryos relates to a specific gestational age and a limited exposure. Further data are needed for other gestational ages and exposure periods before it can be concluded that acrolein is not a teratogen under all possible circumstances.

Skog, E. 1950. A toxicological investigation of lower aliphatic aldehydes. I. Toxicity of formaldehyde, acetaldehyde, propionaldehyde and butyraldehyde; as well as of acrolein, and crotonaldehyde. Acta Pharmacol., 6, 299-318.

Review:

Rats, in groups of eight, were exposed to vapors of various aliphatic aldehydes, and median lethal concentrations were determined. Exposures were for 30 min, and observations continued over a 3-wk post-exposure period. The LC50 values were 1 mg formaldehyde/L air, 37 mg/L for acetaldehyde, 62 mg/L for propionaldehyde, 174 mg/L for butyraldehyde, 0.3 mg/L for acrolein, and 4 mg/L for crotonaldehyde. Mortality resulting from exposures to acetaldehyde, propionaldehyde, and butyraldehyde occurred during and shortly after the 30-min exposure periods. All three aldehydes have a strong narcotic effect, and the author concluded that paralysis of the respiratory centers was the major cause of the mortality. Mortality resulting from exposure to formaldehyde, acrolein, and crotonaldehyde occurred mainly at 6 or more hours after the exposures. These compounds had less of a narcotic effect and caused acute respiratory and pulmonary irritation.

Histological examinations revealed that the lungs were the major organs affected by the exposures to the aldehydes. In some cases the kidneys and liver were also affected. Formaldehyde caused hemorrhages and intra-alveolar and perivascular edema in the lungs; hyperemia, perivascular edema, and necrosis in the liver; and perivascular edema in the kidneys. Acetaldehyde produced hemorrhaging and edema in the lungs and hyperemia in the liver and kidneys. Propionaldehyde exposure resulted in bronchitis and broncho-pneumonias and hyperemia of liver and kidneys. Butyraldehyde caused perivascular edema in the lungs, vacuolation of the bronchial epithelium, and slight changes in the walls of the venae with smooth muscle cells swollen and vacuolated. In the liver degenerative cell changes were seen around the vena centralis, and in the kidneys there were nephrotic changes. Acrolein caused lung edema, hyperemia, hemorrhaging, and possible degenerative changes in the bronchial epithelium, and hyperemia in the heart, kidneys, and liver. Hyperemia and hemorrhages also occurred in the lungs, heart, liver, and kidneys of rats exposed to crotonaldehyde.

Analysis:

The study illustrates the similarities in the toxic effects of the lower aliphatic aldehydes. Especially significant is the fact that lungs are the organs most directly affected by acute exposures; however, for long-term chronic exposures the possibilities of kidney and liver damage may be of greater importance.

Sreenathan, R. N., R. Padmanabhan, and S. Shingh. 1982. Teratogenic effects of acetaldehyde in the rat. Drug Alcohol Depend. 9(4):339-350.

Review:

CF rats were given a single intraperitoneal dose of 50, 75, or 100 mg/kg of acetaldehyde on days 10, 11, or 12 of gestation, or three equal doses, one on each day, over the same gestational period. Fetuses were collected on day 21 and examined for malformations.

All acetaldehyde exposures resulted in a significant number of fetal resorptions, reduced fetal growth, and alterations in skeletogenesis. Fetal malformations included cataracts, low-set ears, micromelia, micrognathia, subcutaneous hemorrhages, edema, lordosis, hydrocephaly, digit anomalies (syndactyly), narrowing of caudal trunk, and hydranencephaly. Placental weight and umbilical cord length were also significantly reduced.

Analysis:

This study demonstrates that acetaldehyde is definitely embryotoxic and teratogenic in rats. The observed effects were generally dose-related, with single exposures to 50 mg/kg producing the fewest adverse effects. Further information is needed concerning the potential teratogenicity of very low chronic inhalation exposures.

Swenberg, J. A., W. D. Kerns, R. I. Mitchell, E. J. Gralla, and K. L. Pavkov. 1980. Induction of squamous cell carcinomas of the rat nasal cavity by inhalation exposure to formaldehyde vapor. Cancer Res. 40:3398-3401.

Review:

Fischer-344 rats (120 males and 120 females) were exposed to 0, 2, 6, or 15 ppm formaldehyde for 6 hr/day, 5 days/wk, for 18 months. Histopathological changes, which occurred at all exposure levels, were dose related and were restricted to the nasal cavity. These changes included mild to severe mucopurulent rhinitis, epithelial dysplasia, and squamous metaplasia. Additional histopathological effects, seen only at a dose level of 15 ppm, included squamous papillary hyperplasia, cellular

atypia, and squamous cell carcinomas. The incidence of carcinomas was 18%. In an identical study on mice, formaldehyde caused similar pathological lesions in the nasal cavity, but no carcinomas developed, even at the 15-ppm dose level.

Analysis:

The authors note that all the animals exposed to formaldehyde also had enzootic sialodacryoadenitis infections, and it was not known to what extent this affected the results.

Carcinomas developed only after prolonged exposures to the highest dose and after continuous severe damage to the nasal epithelium. Lower dose levels were not carcinogenic. These observations, if applicable to other species, including occupationally exposed humans, might be useful in assessing health risks, but further study is needed to establish that severe chronic damage to the respiratory tract is a necessary prerequisite for formaldehyde-induced carcinogenicity.

APPENDIX A-4

**REVIEW AND ANALYSIS OF PERTINENT
REFERENCES ON HEALTH EFFECTS OF SULFATES**

Amdur, M. O., L. Silverman, and P. Drinker. 1952b. Inhalation of sulfuric acid mist by human subjects. Arch. Ind. Hyg. Occup. Med. 6:305-313.

Review:

Healthy subjects were exposed to 0.35 to 5 mg/m³ of sulfuric acid mist (particle size 1 μm) for 5 to 15 min to determine the effects of exposure on respiration. Respiration was measured with a pneumotachograph. The acid mist in the exhaled air was trapped on molecular filter papers and extracted with distilled water, and the acid concentration determined by conductivity measurements. Retention of the inhaled acid averaged 77% (range 50-87%). A concentration as low as 0.35 mg/m³ caused changes in respiration. Inhalation of acid resulted in an increased respiration rate, a decrease in inspiratory and expiratory flow rates, and a decrease in tidal volume. These changes were considered to be of a reflex nature. Concentrations below 1 mg/m³ could not be detected by odor, taste, or irritations.

Analysis:

This study is important in that it demonstrates that alterations in respiration can occur at sulfuric acid concentrations below the current TLV (1 mg/m³). The calculation of acid retention in the respiratory tract did not take into account the possibility of acid neutralization by ammonia excreted in the lungs.

Amur, M. O., M. Dubriel, and D. A. Creasia. 1978. Respiratory response of guinea pigs to low levels of sulfuric acid. Environ. Res. 15:418-423.

Review:

Guinea pigs were exposed for 1 hr to sulfur acid atmospheric concentrations of 0.11 to 1.0 mg/m³ and any resulting effects on respiration were recorded. Exposure to aerosol particle sizes of either 0.3 or 1.0 μm at all tested concentrations resulted in a significant dose-related increase in pulmonary flow resistance. Pulmonary compliance was also significantly reduced by all exposures except those to 1.0-μm particles at concentrations of 0.11 and 0.4 mg/m³. Generally, the 0.3-μm particles produced a greater response at a given concentration than did the 1.0-μm particles. Resistance and compliance did not immediately return to normal values after the exposures were discontinued. Other respiratory parameters, such as tidal volume, respiratory frequency, and minute volume, were not altered.

Analysis:

This study shows that pulmonary function of laboratory animals can be affected by very low levels of sulfates and that the observed changes continue after the exposure ends. It also demonstrates that smaller size aerosol particles of sulfuric acid have a greater irritant effect than larger particles.

Avol, E. L., M. P. Jones, R. M. Bailey, N.-M.N. Chang, M. T. Kleiman, W. S. Linn, K. A. Bell, and J. D. Hackney. 1979. Controlled exposures of human volunteers to sulfate aerosols. Health effects and aerosol characterization. Am. Rev. Respir. Dis. 120(2):319-327.

Review:

Healthy and asthmatic adult men were exposed for 2-hr periods (with intermittent exercise) to a nominal concentration of 100 $\mu\text{g}/\text{m}^3$ of ammonium sulfate, ammonium bisulfate, and sulfuric acid (sulfate equivalents 100, 85, and 75 $\mu\text{g}/\text{m}^3$, respectively). The aerosol particles had a mass median aerodynamic diameter of 0.3 μm . Sulfate analyses were by the barium chloride turbidimetric method. Exposure chamber temperature was 31 C and relative humidity 40% or 85%. There were no significant changes in pulmonary function in any exposed group.

Analysis:

In the experiments with sulfuric acid an additional 50 μg of aerosol/ m^3 was added to the exposure chamber to allow for neutralization by background ammonia concentrations of 5 to 20 ppb. Both healthy and asthmatic individuals were tested. Each subject also provided information as to the severity of symptoms of potential respiratory distress; these included cough, substernal soreness, shortness of breath, wheezing, fatigue, headache, sore throat, nasal discharge, chest tightness, and sputum production. Asthmatics had an increased incidence of symptoms during exposures to 75 $\mu\text{g}/\text{m}^3$ of sulfuric acid but not during exposures to ammonium sulfate and ammonium bisulfate. For the particle size level tested (0.3 μm), this might be considered the lowest dose causing an effect, but even in this case there were no significant changes in pulmonary function.

Chaney, S., W. Blomquist, K. Muller, and G. Goldstein. 1980. Biochemical changes in humans upon exposure to sulfuric acid aerosol and exercise. Arch. Environ. Health 35:211-216.

Review:

Healthy men (average age 28.0 years) were exposed for 4 hr to 100 $\mu\text{g}/\text{m}^3$ (0.033 μM) sulfuric acid aerosol (0.5 μm mean mass diameter). At 30 min and 90 min after the start of the exposure all subjects exercised

for 15 min. Six biochemical blood parameters were measured before and after the exposure: glutathione, lysozyme, glutathione reductase, serum glutamic oxaloacetic acid transaminase, serum vitamin E, and 2,3-diphosphoglyceric acid. There were no significant changes in any of these parameters except for an increase in post exposure levels of glutathione reductase in both exposed and control subjects.

Analysis:

The authors noted that the lack of effect of $100 \mu\text{g}/\text{m}^3$ sulfuric acid on the measured blood parameters was most likely due to the neutralization of the acid by ammonia released in the respiratory tract. The normal range for ammonia concentrations was reported to be $29\text{--}2,200 \mu\text{g}/\text{m}^3$, and only $35 \mu\text{g}/\text{m}^3$ would be required to convert $100 \mu\text{g}/\text{m}^3$ of sulfuric acid to ammonium sulfate. Thus, the potential cytotoxicity of the acid to the epithelial lining of the respiratory tract would be very low until acid levels increased beyond the point where they would be neutralized by the ammonia and this may vary considerably from one individual to another.

Horstman, D., M. Hazucha, E. Haak, and R. Stacy. 1982. Effects of submicronic sulfuric acid aerosol on human pulmonary function. Arch. Environ. Health 37(3):136-141.

Review:

Eighteen healthy men (median age 24.4 years) were exposed for 4 hr in an environmental chamber to an average $108 \mu\text{g}/\text{m}^3$ of sulfuric acid (mass median aerodynamic diameter $0.5 \mu\text{m}$). Subjects exercised for 15 min at 1.5 and 3.5 hr into the exposure at an intensity equivalent to 75% of predicted maximal heart rate. Ventilation was monitored during the initial 14 min of each exposure. Pulmonary function was assessed prior to, and at 2 hr and 4 hr into each exposure. Exposure to sulfuric acid had no significant effects on minute volume, respiratory frequency, tidal volume, airway resistance, specific airway conductance, forced vital capacity (FVC), forced expiratory flow in 1 sec (FEV_{1.0}), FEV_{1.0}/FVC, mean expiratory flow rate between 25% and 75% FVC, and expiratory flow rates at 50% and 25% FVC.

Analysis:

The results of this study, like those of other recent ones, indicate that short-term exposures to submicronic particles of sulfuric acid would not have immediate adverse effects on pulmonary function in normal individuals.

Horvath, S. M., L. J. Folinsbee, and J. F. Bedi. 1982. Effects of sulfuric acid mist exposure on pulmonary function. Environ. Res. 28(1):123-130.

Review:

The effects of sulfuric acid particles (0.90-0.93 μm mass median diameter) on pulmonary function were examined in young male nonsmokers. Subjects were exposed to air and 233, 418, and 939 $\mu\text{g}/\text{m}^3$ of H_2SO_4 at 22 DB (dry belt) and 55% RH (relative humidity), or to air and 314, 600, and 1107 $\mu\text{g}/\text{m}^3$ of H_2SO_4 at 35 DB and 85% RH. Each exposure lasted 2 hr and included a sequence of 20 min of exercise (ventilation about 30 L/min) and 20 min of rest, repeated three times. The only parameter of pulmonary function which showed a significant change with H_2SO_4 exposure was forced expiratory volume at 1 sec. This was decreased at 939 $\mu\text{g}/\text{m}^3$ but not enough to be considered physiologically significant. At the highest exposure level more than half the subjects reported throat irritation, coughing, dizziness, fatigue, and headaches.

Analysis:

This study is similar to others in demonstrating that sulfuric acid levels below 1 mg/m^3 cause only minor changes in pulmonary function. However, at an exposure level of 1 mg/m^3 a number of subjects exhibited signs of respiratory irritation and fatigue that might impair work performance.

Kerr, H. D., T. J. Kulle, B. P. Farrell, L. R. Sander, J. L. Young, D. L. Swift, and R. M. Borushok. 1981. Effects of sulfuric acid aerosol on pulmonary function in human subjects: An environmental chamber study. Environ. Res. 26(1):42-50.

Review:

Twenty-eight normal subjects were exposed for 4 hr to 100 $\mu\text{g}/\text{m}^3$ of sulfuric acid aerosol of particle size 0.1 to 0.3 μm . The tests were conducted in an exposure chamber at 22.5 ± 0.5 C and at a relative humidity of $60 \pm 2\%$. Sulfuric acid concentrations in the chamber were measured as total soluble sulfate using the barium perchlorate-Thorin method. Pulmonary function tests, including forced vital capacity, airway resistance, and nitrogen elimination rate, were conducted prior to exposure and at 2-hr intervals thereafter. Quasistatic and dynamic compliance and pulmonary resistance were measured at 4 hr. No significant changes were found in any parameter of pulmonary function as a result of exposure to sulfuric acid.

Analysis:

This study is useful in showing that single 4-hr exposures to 100 $\mu\text{g}/\text{m}^3$ of sulfuric acid mist have no adverse effect on pulmonary function. Potential adverse effects of repeated exposures were not examined.

Litton Bionetics Inc. 1975. Mutagenic evaluation of compound FDA 73-42, ammonium sulfate granular, food grade. FDA BF-GRAS-382. U.S. Food and Drug Administration, Washington, DC.

Review:

The mutagenic activity of ammonium sulfate was tested in microbial assays with and without the addition of mammalian metabolic activation preparations. The yeast strain used were Saccharomyces cerevisiae, D4, and the bacteria used was Salmonella typhimurium TA-1535, TA-1537, and TA-1538. The ammonium sulfate, tested at concentrations ranging from 2.5 to 10%, exhibited no mutagenetic activity under any of the test conditions.

Analysis:

This rather thorough study, which utilized both positive and negative controls, as well as several different microbial indicators, is strong evidence in favor of the nonmutagenicity of ammonium sulfate.

Murray, F. J., B. A. Schwetz, K. D. Nitschke, A. A. Crawford, F. A. Quast, and R. E. Staples. 1979. Embryotoxicity of inhaled sulfuric acid aerosol in mice and rabbits. J. Environ. Sci. Health 13(3):251-266.

Review:

CF-1 mice and New Zealand white rabbits were exposed to 0, 5.7 ± 1.2 , or 19.3 ± 4.0 mg/m³ sulfuric acid (median particle diameter 1.6 μ m at 5.7 mg/m³ and 2.4 μ m at 19.3 mg/m³) for 7 hr/day during the period of major organogenesis (6th through 15th day of gestation for mice, and 6th through 18th day of gestation for rabbits). On days 18 and 29 for the mice and rabbits, respectively, the animals were sacrificed and examined for embryotoxicity and teratogenicity. Slight maternal toxicity was noted in that liver weight of mice was reduced at 19 mg/m³. In rabbits there was a decrease in body weight gain during the first few days of exposure to 19 μ g/m³ and a dose-related increase in the incidence of subacute rhinitis and tracheitis. Sulfuric acid had no adverse effect on litter size, live fetuses per litter, resorptions per litter, fetal sex ratio, or fetal body size. The offspring of exposed mice did not have a higher incidence of malformations than controls; however, at the highest exposure, two fetuses were conjoined ventrally through the head, neck, and thoracic regions, and both had cranial malformations. In rabbits there was no significant increase in fetal malformations, although there was an increased incidence of small nonossified areas of the skull bones.

Analysis:

Although the conclusions reached in this study appear to be valid for the described experimental conditions, additional studies are needed at higher exposure levels.

APPENDIX B

TABULAR SUMMARIES

TABLE B-1. GOVERNMENTAL EMISSION STANDARDS FOR PRIMARY COMPONENTS OF DIESEL ENGINE EXHAUST

Chemical	Agency	Emission rate	Concentration (ppm)	Comment	Reference
Aldehydes (total)	USEPA ^a	-	-	No standard Emissions, after dilution, from diesel engines used in noncoal underground mining (measured as formaldehyde equivalents)	MSHA 1982
	MSHA ^b	-	10		
Carbon monoxide	USEPA (1983)	a) 18 g/mile	-	a) Low altitudes	EPA 1980c,d
		b) 26 g/mile	-	b) High altitudes Light-duty trucks	
	USEPA (1984)	a) 10 g/mile	-	a) Low altitudes (at curb idle)	-
		b) 26 g/mile	4700	b) High altitudes Light-duty trucks	
	USEPA (1985)	a) 10 g/mile	-	a) Low altitudes (at curb idle)	-
		b) 26 g/mile	4700	b) High altitudes Light-duty trucks	
	MSHA	-	3000	Undiluted exhaust gas from diesel engines operating in noncoal mines	MSHA 1982
	MSHA	-	100	Exhaust gas, after dilution from diesel engines operating in noncoal underground mines	MSHA 1982
	MSHA	-	300	Undiluted exhaust gas from diesel engines operating in gassy noncoal mines (0.25% flammable gas)	MSHA 1982
	MSHA	-	100	Exhaust gas, after dilution, from diesel engines operating in gassy noncoal mines	MSHA 1982

TABLE B-1. (continued)

Chemical	Agency	Emission rate	Concentration (ppm)	Comment	Reference
	State of California	25 g/hp-h	15000	(15000 ppm for 140 cu in engine)	
Hydrocarbons (total)	USEPA (1983)	a) 1.7 g/mile b) 2.0 g/mile	- -	a) Low altitudes b) High altitudes Light-duty trucks	EPA 1980c, d
	USEPA (1984)	a) 0.8 g/mile b) 2.0 g/mile	- -	a) Low altitudes b) High altitudes Light-duty trucks	
	USEPA (1985)	a) 0.8 g/mile b) 2.0 g/mile	- -	a) Low altitudes b) High altitudes Light-duty trucks	
	MSHA	-	-	No standard for diesel engine emissions in underground mines	MSHA 1982
NO _x	State of California	5.0 g/HPh	275	Total CH + NO _x	
	USEPA	2.3 g/mile	-	1982-1985 model year light-duty trucks	EPA 1980c, d
	MSHA	-	200	Emissions, in undiluted exhaust gas, from diesel engines operating in gassy (0.25% flammable gas) noncoal mines (measured as NO ₂ equivalents)	MSHA 1982
	MSHA	-	25	Emissions, after dilution, in exhaust gas from diesel engines operating in gassy and nongassy noncoal mines	MSHA 1982
Particulates	State of California	5.0 g/HPh	-	Total NO _x + CH	
	USEPA	0.6 g/mile	-	1983-84 model light-duty diesel vehicles and trucks	EPA 1980b
	USEPA	0.26 g/mile	-	1985 model light-duty diesel trucks	
Sulfates	USEPA	-	-	No standards	
	MSHA	-	-	No standards	

^aU.S. Environmental Protection Agency.^bMine Safety and Health Administration.

TABLE B-2. GOVERNMENTAL EXPOSURE LIMITS FOR PRIMARY COMPONENTS OF DIESEL ENGINE EXHAUST

Chemical	Agency	Concentration		Comment	Reference
		ppm	mg/m ³		
Aldehydes (total)	OSHA ^a	-	-	No exposure limit	OSHA 1982
	MSHA ^b	-	-	No exposure limit for under- ground mines	MSHA 1982
Carbon monoxide	OSHA	50	55	8-hr time weighted average (TWA)	OSHA 1982
	MSHA	100	-	Maximum permissible in noncoal mines	MSHA 1982
	MSHA	50	-	Maximum permissible in gassy noncoal mines	MSHA 1982
	NIOSH ^c	35	40	10-hr TWA	NIOSH 1982
	NIOSH	200	229	Ceiling value (no minimum time)	NIOSH 1982
	ACGIH ^d	50	55	8-hr TWA	ACGIH 1982
	ACGIH	400	440	Short-term exposure limit (STEL)	ACGIH 1982
Hydrocarbons (total)	OSHA	-	-	No standard for total hydro- carbons	OSHA 1982
	MSHA	-	-	No standard for total hydro- carbons	MSHA 1982
NO _x	OSHA (NO)	25	30	8-hr TWA	OSHA 1982
	OSHA (NO ₂)	5	9	Ceiling value	OSHA 1982
	MSHA (NO _x)	25	-	Noncoal underground mines	MSHA 1982
	MSHA (NO _x)	12.5	-	Gassy noncoal underground mines	MSHA 1982
	NIOSH (NO)	25	30	10-hr TWA	NIOSH 1982
	NIOSH (NO ₂)	1	1.8	15-min ceiling value	NIOSH 1982
	ACGIH (NO)	25	30	8-hr TWA	ACGIH 1983
	ACGIH (NO)	35	45	STEL	ACGIH 1982
	ACGIH (NO ₂)	3	6	8-hr TWA	ACGIH 1982
	ACGIH (NO ₂)	5	10	STEL	ACGIH 1982
Particulates	OSHA	-	-	No standards	OSHA 1982
	MSHA	-	-	No standards	MSHA 1982
Sulfates	OSHA	-	-	No standards	OSHA 1982
	MSHA	-	-	No standards	MSHA 1982
Sulfuric acid	OSHA	-	1	8-hr TWA	OSHA 1982
	NIOSH	-	1	10-hr TWA	NIOSH 1982
	ACGIH	-	1	8-hr TLV	ACGIH 1982
	MSHA	-	-	No standards	MSHA 1982

^aOccupational Safety and Health Administration.

^bMine Safety and Health Administration.

^cNational Institute of Occupational Safety and Health; recommended occupational exposure limits.

^dAmerican Conference of Governmental Industrial Hygienists; occupational exposure guidelines.

TABLE B-3. EXPOSURE LIMITS FOR OSHA REGULATED COMPOUNDS
FOUND IN DIESEL EMISSIONS^a

Chemical name (CAS number)	Time weighted averages (TWA)	
	ppm	mg/m ³
Acetaldehyde (75-07-0)	200	360
Acetic acid, ethyl ester (141-78-6)	400	1400
Acetic acid, methyl ester (79-20-9)	200	610
Acetylene (74-86-2)	2500 ^b	2700
Acrolein (107-02-8)	0.1	0.25
Ammonia (7664-41-7)	50	35
Barium oxide (1304-28-5)	-	0.5
Benzenamine (62-53-3)	5 ^c	19
Benzenamine, 2-methyl- (95-53-4)	5 ^c	22
Benzene (71-43-2)	10 ^d	32
Benzene, 1-(1,1-dimethylethyl)- 4-methyl- (98-51-1)	10	60
1,4-Benzenediol (123-31-9)	-	2
Benzene, ethenylmethyl- (25013-15-4)	100	480
1,2-Benzene dicarboxylic acid, dibutyl ester (84-74-2)	-	5
1,2-Benzene dicarboxylic acid, dimethyl ester (131-11-3)	-	5
Benzene, dimethyl- (1330-20-7)	100	435
Benzene, ethyl- (100-41-4)	100	435

TABLE B-3 (continued)

Chemical name (CAS number)	Time weighted averages (TWA)	
	ppm	mg/m ³
Benzene, methyl- (108-88-3)	200 ^e	766
Benzene, (1-methylethenyl)- (98-83-9)	100	480
Benzene, (1-methylethyl)- (98-82-8)	50 ^c	245
Benzene, 1,1'-oxybis- (101-84-8)	1	7
Bicyclo(2.2.1)heptan-2-one, 1,7,7-trimethyl- (76-22-2)	-	2
1,1'-Biphenyl (92-52-4)	0.2	1
1,3-Butadiene (106-99-0)	1000	2200
2-Butanone (78-93-3)	200	590
Carbon black (7440-44-0)	-	3.5
Carbon dioxide (124-38-9)	5000	9000
Carbon disulfide (75-15-0)	20 ^f	63
Carbon monoxide (630-08-0)	50	55
Crotonaldehyde (4170-30-3)	2	6
2,5-Cyclohexadiene-1,4-dione (106-51-4)	0.1	0.4
Cyclohexane (110-82-7)	300	1050
Cyclohexane, methyl- (108-87-2)	500	2000
Cyclohexanol (108-93-0)	50	200
Cyclohexanone (108-94-1)	50	200

TABLE B-3 (continued)

Chemical name (CAS number)	Time weighted averages (TWA)	
	ppm	mg/m ³
Cyclohexanone, methyl- ^g (583-60-8)	100	460
Cyclohexene (110-83-8)	300	1015
2-Cyclohexen-1-one, 3,5,5- trimethyl- (78-59-1)	25	140
Diazomethane (334-88-3)	0.2	0.4
1,4-Dioxane (123-91-1)	100 ^c	360
Formaldehyde (50-00-0)	3 ^h	3.7
Formic acid (64-18-6)	5	9
Formic acid, ethyl ester (109-94-4)	100	300
2-Furancarboxaldehyde (98-01-1)	5 ^c	20
2-Furanmethanol (98-00-0)	50	200
Furan, tetrahydro- (109-99-9)	200	590
Heptane (142-82-5)	500	2000
Hexane (110-54-3)	500	1800
2-Hexanone (591-78-6)	100	410
Hexone (108-10-1)	100	410
Hydrocyanic acid (74-90-8)	10 ^c	11
Hydrogen sulfide (7783-06-4)	20 ⁱ	28
1,3-Isobenzofurandione (85-44-9)	2	12

TABLE B-3 (continued)

Chemical name (CAS number)	Time weighted averages (TWA)	
	ppm	mg/m ³
Methylacetylene (74-99-7)	1000	1650
Naphthalene (91-20-3)	10	50
Nickel carbonyl (13463-39-3)	0.001	0.007
Nitroethane (79-24-3)	100	310
Nitrogen oxide (10102-43-9)	25	30
Nitrogen dioxide (10102-44-0)	5 ^j	9
Nitromethane (75-52-5)	100	250
Octane (111-65-9)	500	2350
Oxiranemethanol (556-52-5)	50	150
Oxirane, (phenoxymethyl)- (122-60-1)	10	60
Oxirane, [(2-propenyloxy)- methyl]- (106-92-3)	10 ^j	45
n-Pentane (109-66-0)	1000	2950
2-Pentanone (107-87-9)	200	700
3-Penten-2-one, 4-methyl- (141-79-7)	25	100
Phenol (108-95-2)	5 ^c	19
Phenol, 2 methyl- (95-48-7)	5 ^c	22
Phenol, 3-methyl- (108-39-4)	5 ^c	22
Phenol, 4-methyl- (106-44-5)	5 ^c	22

TABLE B-3 (continued)

Chemical name (CAS number)	Time weighted averages (TWA)	
	ppm	mg/m ³
Phenol, 2-methyl-4,6-dinitro- (534-52-1)	-	0.2 ^c
Propane (74-98-6)	1000	1800
2-Propanone (67-64-1)	1000	2400
2-Propenoic acid, ethyl ester (140-88-5)	25 ^c	100
2-Propenoic acid, methyl ester (96-33-3)	10 ^c	35
2-Propenoic acid, 2-methyl-, methyl ester (80-62-6)	100	410
Pyridine (110-86-1)	5	15
Styrene (100-42-5)	100 ^k	433
Sulfur dioxide (7446-09-5)	5	13
Sulfuric acid (7664-93-9)	-	1

^aOSHA = Occupational Safety and Health Administration. Eight-hour time-weighted averages (TWA) unless otherwise noted. Numbers in () are Chemical Abstract Registry numbers.

^b10% of lower exposure limit.

^cCutaneous absorption can contribute to overall exposure.

^dAcceptable ceiling concentration 25 ppm (80 mg/m³); maximum 10-min peak, 50 ppm (160 mg/m³).

^eAcceptable ceiling concentration 300 ppm (1149 mg/m³); maximum 10-min peak/8 hr, 500 ppm (1915 mg/m³).

^fAcceptable ceiling concentration 30 ppm (94.5 mg/m³); maximum 30-min peak/8 hr, 100 ppm (315 mg/m³).

^gSpecific isomer in exhaust not identified. Data and CAS number given for o-methylcyclohexanone.

^hAcceptable ceiling concentration 5 ppm (6.2 mg/m³); maximum 10-min peak/8 hr, 10 ppm (12.4 mg/m³).

TABLE B-3 (Footnotes continued)

ⁱAcceptable ceiling concentration; maximum 10-min peak, 50 ppm (7 mg/m³) once per 8 hr only if no other exposure occurs.

^jMaximum permissible exposure limit, regardless of time of exposure.

^kAcceptable ceiling concentration 200 ppm (866 mg/m³); maximum 5-min peak/3 hr, 600 ppm (2598 mg/m³).

TABLE B-4. ACGIH^a AND NIOSH^b RECOMMENDED STANDARDS^c

Chemical Name	ACGIH	NIOSH
Ethylene (74-85-1) ^d	Noted as being an asphyxiant	
Ethane (74-84-0)	Noted as being an asphyxiant	
n-Butane (106-97-8)	TWA - 800 ppm, 1900 mg/m ³	
Benzo(a)pyrene (50-32-8)	Noted as an industrial substance suspect of carcinogenic potential for man	
Chrysene (218-01-9)		To be controlled as an occupational carcinogen
Valeraldehyde (110-62-3)	TWA - 50 ppm, 175 mg/m ³	
Propylene (115-07-1)	Noted as being an asphyxiant	
Methane (74-82-8)	Noted as being an asphyxiant	

^aAmerican Conference of Governmental Industrial Hygienists.

^bNational Institute for Occupational Safety and Health.

^cIncludes only those compounds found in diesel exhaust that do not have an OSHA standard.

^dChemical Abstract Registry Number.

TABLE B-5. CHEMICAL COMPOUNDS FOUND IN DIESEL EMISSIONS FOR WHICH NO FEDERAL OCCUPATIONAL EXPOSURE LIMITS OR AMBIENT AIR STANDARDS EXIST

Aceanthrone	Benzo(c)fluorene
Acenaphthenol	Benzofluorenone
Acenaphthylene	Benzo(a)fluorenone
Acphenanthrone	H-Benzo(a)fluorenone
Acetylaminofluorene	Benzo(furan)
1-Acetylnaphthalene	Benzo(e)perylene
Anthanthrene	Benzo(ghi)perylene
Anthracene	Benzo(phenanthracene)- carboxaldehyde
Anthracene carboxaldehyde	Benzo(c)phenanthrene
9-Anthracene carboxaldehyde	1-Benzopyran-4(4H)-one
Anthracene dicarboxylic acid anhydride	2H-1-Benzopyran-2-one
Anthracenedione	Benzo(e)pyrene
Anthracene quinone	Benzo(e)pyrene-4,5-dihydrodiol
9,10-Anthracene quinone	Benzo(e)pyrene-9,10-epoxide
Anthracenone	1,6-Benzo(a)pyrenequinone
Anthranthene	3,6-Benzo(a)pyrenequinone
Anthroic acid	6H-Benzo(cd)pyrenone
Anthrone	Benzo(ed)pyrenone
9-Anthrone	Binaphthalene
Anthroquinone	Binaphthyl
Barium sulfate	Biphenylcarboxaldehyde
Benz(j)aceanthrylene	1,1'-Biphenyl-4-carbox- aldehyde
Benz(e)acephenanthrylene	Biphenyldicarbonitrile
Benzacridine	Biphenylene
Benzaldehyde	1,1'-Biphenyl, 2-methoxy- [1,1'-Biphenyl]-2-ol
Benz(a)anthracene	[1,1'-Biphenyl]-4-ol
Benzanthracene dicarbox- aldehyde	Butanal
Benz(a)anthracene-7-12- dione	1-Butene
7H-Benz(de)anthracene-7-one	2-Butene
Benz(j)fluoranthene	Butyl-2-methylpropyl- phthalate
Benzo(a)anthracene carboxaldehyde	Butyl-naphthalene
Benzo(a)anthracenequinone	n-Butyraldehyde
7,12-Benzo(a)anthracene quinone	Carbon oxide sulfide
Benzoanthrone	o-Chlorophenol
7-Benzo(de)anthrone	Chrysene carboxaldehyde
Benzo(dh)anthrone	Chrysene quinone
Benzo(b)chrysene	Coronene
Benzo(c)cinnoline	Cyclodexanol
Benzo(ghi)fluoranthene	Cyclopentacorone
Benzo(k)fluoranthene	Cyclopentano(c,d)-pyrene
Benzo(j)fluoranthrene	Cyclopentano(c,d)pyrene-3,4- cis-diol
Benzo(k)fluoranthrene	Cyclopentano(c,d)pyrene-3,4- oxide
Benzofluorene	

TABLE B-5 (continued)

Cyclopentano(c,d)-pyrene- 3,4-trans-diol	Dihydroxyfluorene
4H-Cyclopenta(def)phenanthren- 4-one	Dihydroxymethoxybenzene
Cyclopentaphenanthren-5-one	Dihydroxymethylanthracene
Cyclopentapyrene	Dihydroxymethylfluorene
Cyclopenta(cd)pyrene	Dihydroxymethyl phenanthrene
Cyclopentenodibenzopyrene	Dimethylanthracene
Cyclopenteno(c,d)pyrene	9,10-Dimethylanthracene
Cyclopenteno(c,d)pyrene anhydride	Dimethylanthracene carboxaldehyde
Decalin	Dimethylanthrone
Decane	Dimethylbenz(a)anthracene
Dibenz(a,c)anthracene	Dimethylbenzonaphtho- thiophene
Dibenz(a,j)anthracene	Dimethylcyclopentacene- naphthylene
Dibenz(a,h)anthracene dicarboxaldehyde	Dimethyldecalin
Dibenz(c,g)carbazole	1,9-Dimethylfluorene
Dibenzo(def,mno)chrysene	Dimethylfluorenequinone
Dibenzo[b,e][1,4]-dioxin	Dimethylfluorenone
1,2,3,4-Dibenzofluoranthene	Dimethylhydroxyanthracene
Dibenzofluorene	Dimethylhydroxyfluorene
Dibenzo(a,g)fluorene	Dimethylhydroxyphenanthrene
Dibenzofuran	Dimethylindan
1,2,3-Dibenzopyrene	Dimethylnaphthalene- carboxaldehyde
Dibenzo(a,i)pyrene	Dimethylnaphthalene dicar- boxylic acid anhydride
1,2,9,10-Dibenzotetracene	Dimethylnaphthothiophene
Dibenzothiophene	Dimethylperhydrophen- anthrene
2,6-Di-tert-butyl-4-methyl- phenol	Dimethylphenanthrene
1,2-Dihydroacenaphthylene	Dimethylphenanthrene carboxaldehyde
9,10-Dihydrobenzo(e)pyrene	Dimethyl phenanthrone
4,5-Dihydrodihydroxy benzopyrene	Dimethyltetrahydro- naphthalene
Dihydrodihydroxy- fluoranthene	Dimethyltetralin
Dihydrodihydroxypyrene	2,7-Dinitrofluorene
5,6-Dihydrodiolbenzo(a)- anthracene	1,5-Dinitro-2-methyl-9,10- anthraquinone
1,6-Dihydrodiolbenzo(a)- pyrene	1,5-Dinitronaphthalene
4,5-Dihydrodiolbenzo(a)- pyrene	Dinitropyrene
2,3-Dihydroinden-1-one	Diphenylacenaphthylene
1,2-Dihydrotrimethyl- naphthalene	Diphenylacetylene
Dihydroxyanthracene	Diphenylbenzene
9,10-Dihydroxyanthracene	n-Docosane
Dihydroxydimethyl- anthracene	Dodecylcyclohexane

TABLE B-5 (continued)

n-Eicosane	Hydroxypyrene
Ethene	Hydroxytrimethylanthracene
Ethylfluorene	Hydroxytrimethylphen-
1-Ethyl naphthalene	anthrene
p-Ethylphenol	Hydroxyxanthene
Ethyl tridecane	3-Hydroxyxanthene-9-one
1,1'-(1,2-Ethynediyl)-	Hydroxyxanthrone
bisbenzene	1-Indanone
Fluoranthene carbox-	Indeno(1,2,3-cd)fluoranthene
aldehyde	Isoamylfluorene
Fluoranthenequinone	Isobutyraldehyde
Fluoranthene-2,3-dihydrodiol	Isobutylene
Fluoranthone	Isopentene
Fluoranthrene	2-Isopropyl naphthalene
9-Fluorene-a-one	o-Isopropylphenol
9H-Fluorene	Methoxybenzaldehyde
2-Fluorene carboxaldehyde	Methoxybiphenyl
9-Fluorene carboxaldehyde	2-Methoxy,1,1'biphenyl
Fluorenequinone	Methoxyfluorene
1,4-Fluorenequinone	Methoxyphenanthrene
9H-Fluorene-9-one	Methoxyxanthone
9H-Fluorene-9-one,2,4,7-	Methylanthracene
trinitro-	2-Methylanthracene
n-Heneicosane	9-Methylanthracene
n-Heptadecane	Methylanthracene
1-Heptadecene	carboxaldehyde
n-Hexadecane	2-Methyl-9-anthracene
Hexaldehyde	carboxaldehyde
Hexanol	Methyl-9-10-anthracenedione
Hydroxyanthracene	Methylanthracenequinone
Hydroxyanthroic acid	Methyl-9-10-anthracenequinone
2-Hydroxybenzaldehyde	2-Methyl-9-10-anthracene-
5-Hydroxybenzo(a)anthracene	quinone
6-Hydroxybenzo(a)pyrene	Methyl anthroic acid
7-Hydroxybenzo(a)pyrene	Methylanthrone
Hydroxychrysene	Methyl-9-anthrone
Hydroxycoronene	4-Methylbenzaldehyde
Hydroxyfluorene	Methylbenz(a)anthracene
9-Hydroxyfluorene	Methylbenzanthrone
Hydroxyfluorenone	Methylbenzoate
Hydroxynaphthalene dicar-	7-Methylbenzofuran
boxylic acid anhydride	Methylbenzoic acid
Hydroxynaphthalic acid	Methylbenzo(a)pyrene
Hydroxynaphthoic acid	Methylbenzo(e)pyrene
Hydroxynitrofluorene	10-Methylbenzopyrene
1-Hydroxyphenanthrene	3-Methylbutanal
Hydroxyphenanthrene	2-Methylbutane
Hydroxyphenanthroic acid	Methylcarbazole
Hydroxyphthalic acid	Methyl caronene

TABLE B-5 (continued)

3-Methylcholanthrene	Methylphenylcinnoline
Methylchrysene	Methylphthalic acid
Methylcyclohexanone	1-Methylpyrene
Methyl-4H-cyclopenta(def)-phenanthren-4-one	Methylpyrenequinone
Methyldecane	Methylquinoline
Methyldibenzothiophene	Methyltetralin
Methyldihydrofluoranthene	Methyltriphenylene
Methyldihydropyrene	Methylundecane
Methylfluoranthenequinone	Monomethylaniline
1-Methylfluorene	Naphthalene acetaldehyde
9-Methylfluorene	1-Naphthalene carboxaldehyde
Methylfluorenequinone	2-Naphthalene carboxaldehyde
Methylfluorenone	1-Naphthalene carboxylic acid
2-Methylfluorenone	2-Naphthalene carboxylic acid
Methyl-9-fluorenone	Naphthalene dicarboxaldehyde
Methylhydroxyanthracene	1,8-Naphthalene dicarboxylic acid
5-Methylhydroxybenzo-phenanthrene	Naphthalene dicarboxylic acid anhydride
Methylhydroxyfluorene	1H-Naphthalenequinone
Methylhydroxyphenanthrene	2-Naphthalenol
Methylindan	Naphtho(1234,def)chrysene
Methylnaphthaldehyde	Naphtho-2,3(b)furan-4,9-dione
6-Methyl-2-naphthaldehyde	Naphthopyrandione
Methylnaphthalene	Naphtho(1,8-cd)pyran-1,3-dione
Methylnaphthalene carboxaldehyde	1H,3H-Naphtho-1,8-cd-pyran-1,3-dione
Methylnaphthalene dicarboxylic acid anhydride	1-Naphtho(cd)pyrone
Methylnaphthalic acid	C4-Naphthothiophene
Methylnaphthoic acid	9-Nitroanthracene
Methylnitroanthracene	6-Nitrobenzo(a)pyrene
Methylnitropyrene	Nitrochrysene
Methylpentadecane	2-Nitrofluorene
Methylperylene	3-Nitro-9-fluorenone
1-Methylphenanthrene	Nitrogen pentoxide
2-Methylphenanthrene	Nitronaphthalic acid
3-Methylphenanthrene	Nitrophenanthrene
4-Methylphenanthrene	Nitropyrene
9-Methylphenanthrene	1-Nitropyrene
Methylphenanthrene carboxaldehyde	Nitropyrene
Methylphenanthrenequinone	N-Nitrosomorpholine
Methyl-9-10-phenanthrenequinone	n-Nonadecane
Methylphenanthroic acid	n-Octadecane
Methylphenanthrone	Octahydrophenanthrene
4-Methylphenylbenzo(c)-cinnoline	Odor component of diesel emissions
	n-Pentadecane

TABLE B-5 (continued)

Pentaphene	Pyrenone
1-Pentene	Pyrone
Perhydrophenanthrene	Riphenylene
Perinaphthindenone	Sulfur
Peroxyacetyl nitrate	Sulfur trioxide
Peroxypropionyl nitrate	n-Tetradecane
Perylene	1-Tetradecene
Phenanthrene	1,2,3,4-Tetrahydronaphthalene
Phenanthrene carbox- aldehyde	Tetrahydrophenanthrene
2-Phenanthrene carbox- aldehyde	Tetramethylnaphthalene
Phenanthrene-9-carbox- aldehyde	Thioxanthone
Phenanthrene dicarboxylic acid anhydride	9H-Thioxanthen-9-one
Phenanthrenequinone	Thioxanthen-9-one
9,10-Phenanthrenequinone	9-Thioxanthone
Phenanthroic acid	Trihydroxyanthraquinone
Phenanthrone	Trihydroxyfluorene
Phenanthroquinone	Trimethylantracene
Phenylbenzopyranone	Trimethylfluorene
Phenylethyl ketone	Trimethylfluorenone
1-Phenyl-2,4-hexadiyn-1-one	Trimethylindan
1-Phenylnaphthalene	Trimethylnaphthalene
2-Phenylnaphthalene	Trimethylnaphthalene carboxaldehyde
Phenylpyrocatechol	Trimethylnaphthothiophene
Phenylpyrrolopyridine	2,2,4-Trimethylpenta-1,3- dioldiisobutyrate (bkg)
Phthalic acid	Trimethylphenanthrene
Propanal	2,3,5-Trimethylphenol
Pyrene	Trimethyltetralin
Pyrene carboxaldehyde	Trinaphthenebenzene
1-Pyrene carboxaldehyde	Triphenylene carboxaldehyde
Pyrene quinone	Triphenylene quinone
3,10-Pyrene quinone	9H-Xanthen-9-one
Pyreno-3,4-dicarboxylic anhydride	9-Xanthone
	2,6-Xylenol
	3,5-Xylenol

TABLE B-6. CONCENTRATIONS OF COMPOUNDS
IDENTIFIED IN DIESEL EXHAUST^a

Compound	Concentration	Reference
Acetaldehyde (ppm)	3.2	Vogh 1969
	0.5-4.8	Iwai et al. 1976
	0.02-0.31	Springer and Stahman 1978
	0.27-0.67	Johnson et al. 1982
Acetone (ppm)	2.2-25.2	Linnell and Scott 1962a
	1.42-38.0	Linnell and Scott 1962b
	1-19	Reckner et al. 1965
	0.13-0.48	Springer and Stahman 1978
	0.07	Johnson et al. 1982
Acrolein (ppm)	4	Linnell and Scott 1962a
	0.84-17.4	Linnell and Scott 1962b
	5-10	Cohen and Altschuller 1961
	0.8-42	Reckner et al. 1965
	2.9	Vogh 1969
	0.2-0.3	Smythe and Karasek 1973
	2.1-7.5	Iwai et al. 1976
	2.1-8.4	EHA 1978
	0.06-1.4	Holland 1978
	0.06	Johnson et al. 1982
Aldehydes (total) (ppm)	18-56	Reckner et al. 1965
	3-97	Marshall and Fleming 1971
	9-35	Iwai et al. 1976
	13.8-58.8	EHA 1978
	1.3	Williams and Chock 1980
	4.2-4.7	Creech et al. 1982
	4.46	Johnson et al. 1982
Ammonia (ppm)	0.2	Williams and Chock 1980
Anthanthrene ($\mu\text{g}/\text{m}^3$)	0-0.89	Reckner et al. 1965
Anthracene ($\mu\text{g}/\text{m}^3$)	1.5-16	Reckner et al. 1965
	20-200	Holland 1978
Benzaldehyde (ppm)	0.3	Vogh 1969
	0-0.16	Springer and Stahman 1978
	0.2-1.5	Springer and Baines 1978
	0.05-0.07	Creech et al. 1982
	0.10-0.31	Johnson et al. 1982
Benzanthracene ($\mu\text{g}/\text{m}^3$)	0-17	Reckner et al. 1965
	<0.4-4	Holland 1978
Benzene (ppm)	0.3-1	Springer and Stahman 1978

TABLE B-6 (continued)

Compound	Concentration	Reference
Benzo(b)fluoranthene ($\mu\text{g}/\text{m}^3$)	0-14	Reckner et al. 1965
Benzo(k)fluoranthene ($\mu\text{g}/\text{m}^3$)	0.05-13	Reckner et al. 1965
Benzo(a)fluorenes ($\mu\text{g}/\text{m}^3$)	0.7-4.8	Reckner et al. 1965
Benzo(ghi)perylene ($\mu\text{g}/\text{m}^3$)	0-11 0.05	Reckner et al. 1965 Spindt et al. 1974
Benzo(a)pyrene ($\mu\text{g}/\text{m}^3$)	0.05-10.5 0.16 60-<600 43	Reckner et al. 1965 Spindt et al. 1974 Holland 1978 Williams and Chock 1980
Benzo(e)pyrene ($\mu\text{g}/\text{m}^3$)	0-87 0.03 40-<400	Reckner et al. 1965 Spindt et al. 1974 Holland 1978
1,3-Butadiene (ppm)	0.24-4.81 0.06-13	Linnell and Scott 1962b Reckner et al. 1965
Butane (ppm) (n-Butane)	<0.05-0.14 0.2	Linnell and Scott 1962b Reckner et al. 1965
1-Butene (ppm)	0.37-7.0	Linnell and Scott 1962b
1-Butene (+ iso-butene) (ppm)	0.1-41	Reckner et al. 1965
1-Butene, 2 methyl- (+ trans 2-pentene) (ppm)	0.1-3.8	Reckner et al. 1965
1-Butene, 3 methyl- (ppm)	0.1-2.1	Reckner et al. 1965
2-Butene (cis or trans) (ppm)	<0.005-1.56 0.06-2.2	Linnell and Scott 1962b Reckner et al. 1965
2-Butene, 2 methyl- (ppm)	0.2-3.3	Reckner et al. 1965
Butylene (ppm) iso-butylene	0.14-2.85	Linnell and Scott 1962b

TABLE B-6 (continued)

Compound	Concentration	Reference
Butyraldehyde (ppm)		
n-butyraldehyde	0.3	Vogh 1969
iso-butyraldehyde	0.08-0.56	Springer and Stahman 1978
	0.08-0.13	Creech et al. 1982
	0.10-0.13	Johnson et al. 1982
Carbon dioxide (ppm)	16900-142000	Linnell and Scott 1962a
	6400-91000	Reckner et al. 1965
	4000-25000	Holland 1978
Carbon monoxide (ppm)	173-4800	Linnell and Scott 1962a
	220-3000	Reckner et al. 1965
	153-5146	Marshall 1978
	10-300	Holland 1978
	188	Williams and Chock 1980
Chrysene ($\mu\text{g}/\text{m}^3$)	31	Reckner et al. 1965
	5-200	Holland 1978
Coronene ($\mu\text{g}/\text{m}^3$)	5.4	Reckner et al. 1965
Crotonaldehyde (ppm)	0.07-0.6	Smythe and Karasek 1973
	0.1-0.8	Iwai et al. 1976
	0.44-1.41	Springer and Baines 1978
	0.08-0.20	Springer and Stahman 1978
	0.02-0.04	Creech et al. 1982
	0.03-0.62	Johnson et al. 1982
Ethylene (ppm)	52-85.4	Linnell and Scott 1962a
	6.85-82.8	Linnell and Scott 1962b
	10-56	Reckner et al. 1965
	0.9-26.7	Springer and Stahman 1978
Ethane (ppm)	0.1-1.3	Springer and Stahman 1978
Fluoranthrene ($\mu\text{g}/\text{m}^3$)	9-120	Reckner et al. 1965
	1.9	Spindt et al. 1974
Formaldehyde (ppm)	15	Linnell and Scott 1962a
	2.98-26.7	Linnell and Scott 1962b
	4.6-40	Reckner et al. 1965
	18.3	Vogh 1969
	16-24	Smythe and Karasek 1973
	5.1-18	Iwai et al. 1976
	0-35	Holland 1978
	1.24-12.33	Springer and Baines 1978
	9.2-34.9	EHA 1978
	3.26-3.61	Creech et al. 1982
	1.99-3.56	Johnson et al. 1982

TABLE B-6 (continued)

Compound	Concentration	Reference
Hexaldehyde (ppm)	0.2	Vogh 1969
	0.05-0.09	Springer and Baines 1978
	0-0.04	Springer and Stahman 1978
	0.01	Creech et al. 1982
1-Hexene (ppm)	0.1-20	Reckner et al. 1965
Hydrocarbons (total) (ppm)	9.7-54.2	Linnell and Scott 1962b
	6-56	Reckner et al. 1965
	77-4852	Marshall 1978
	27	Williams and Chock 1980
Hydrogen cyanide (ppm)	0.2	Williams and Chock 1980
Indeno (1,2,3,cd) pyrene ($\mu\text{g}/\text{m}^3$)	0-11	Reckner et al. 1965
Methane (ppm)	0.1-5.6	Springer and Stahman 1978
Methyl ethyl ketone (ppm)	0.24-0.68	Johnson et al. 1982
Nitric oxide (ppm)	42-985	Linnell and Scott 1962a
	42-1850	Reckner et al. 1965
	<0.2-13.4	Holland 1978
Nitrogen dioxide (ppm)	38-408	Linnell and Scott 1962a
	46-822	Linnell and Scott 1962b
	25-150	Reckner et al. 1965
	0.5-20	Holland 1978
	15	Williams and Chock 1980
Particulates (mg/m^3)	33.3-75.3	Frey and Corn 1967
	6.7-143.7	Springer and Stahman 1978
	0.5-236	Williams and Chock 1980
Pentane (ppm) n-pentane iso-pentane	0.07-0.80	Linnell and Scott 1962b
	<0.05-0.24	Linnell and Scott 1962b
1-Pentene (ppm)	0.10-5.24	Linnell and Scott 1962b
1-Pentene (+ propyne) (ppm)	0.05-13	Reckner et al. 1965
1-Pentene, 4 methyl- (ppm)	0.2-2.3	Reckner et al. 1965

TABLE B-6 (continued)

Compound	Concentration	Reference
2-Pentene (ppm) cis-pentene	0.04-1.0	Reckner et al. 1965
Perylene ($\mu\text{g}/\text{m}^3$)	0.01-0.14	Reckner et al. 1965
Phenanthrene ($\mu\text{g}/\text{m}^3$)	46 8	Spindt et al. 1974 Holland 1978
Phenols ($\mu\text{g}/\text{m}^3$)	107	Spindt et al. 1974
Propadiene (ppm)	0.03-0.1	Reckner et al. 1965
Propane (ppm)	<0.05-0.77 0.2-2.1 0-1.5	Linnell and Scott 1962b Reckner et al. 1965 Springer and Stahman 1978
Propionaldehyde (ppm)	0.33-0.08 0.03-0.69	Creech et al. 1982 Johnson et al. 1982
Propylene (ppm)	1.12-23.9 0.2-86 0.7-15.1	Linnell and Scott 1962b Reckner et al. 1965 Springer and Stahman 1978
Pyrene ($\mu\text{g}/\text{m}^3$)	11-57 2.5 4-20	Reckner et al. 1965 Spindt et al. 1974 Holland 1978
Salicylaldehyde (ppm)	0.05	Johnson et al. 1982
Sulfates (mg/m^3)	0.31-2.34 0.34-7.0 4.5	Khatri et al. 1978 Springer and Stahman 1978 Williams and Chock 1980
Sulfur dioxide (ppm)	3.3-85 0.9-16 0-<0.5	Linnell and Scott 1962a Reckner et al. 1965 Holland 1978
Sulfuric acid (mg/m^3)	0.3-7.1 0.23-44.85	Springer and Stahman 1978 Holland 1978
Tolualdehyde (ppm) para-tolualdehyde	0.1 0.17	Creech et al. 1982 Johnson et al. 1982
Toluene (ppm)	0.1-1.0	Springer and Stahman 1978

TABLE B-6 (continued)

Compound	Concentration	Reference
Valeraldehyde (ppm)		
n-valeraldehyde	0.01-0.02	Creech et al. 1982
	0.1-1.16	Johnson et al. 1982
iso-valeraldehyde	0.01-0.03	Creech et al. 1982

^aData from selected references. Ranges given are for various operating conditions or engines tested. Test engines are primarily heavy-duty diesels, but results are also indicative of light-duty diesel emissions.

TABLE B-7. SUMMARY OF CARBON MONOXIDE EXPOSURE-EFFECT RELATIONSHIPS

Exposure (HbCO%)	Reported Effects
<u>Behavioral Changes</u>	
20	Essentially no impairment in time discrimination (using Beard-Wertheim task)
11.3	No vigilance decrement (using Horvath task)
9	No vigilance decrement (using Fodor-Winneke task); no change in reaction time
8.4	No vigilance decrement (using Groll-Knapp vigilance task)
7.6	Longer reaction times
7.3	Disturbance in certain perceptual and cognitive processes
5	Vigilance decrement
4.5	Longer reaction times
3.1	Initial vigilance decrement with subsequent normalization; no change in response latency
3	Vigilance decrement
2	Impaired performance in time discrimination
<u>Changes in Work Performance</u>	
6.3	Decrease in maximal work time
4.3	Decrease in a maximal oxygen uptake
4.0 (1.7 ^a)	Decrease in mean exercise time until exhaustion
2.5 (0.6 ^a)	Decrease in absolute exercise time in nonsmokers
<u>Aggravation of Symptoms in Patients with Cardiovascular Disease</u>	
5.1 (1.1 ^a)	Shortened time to angina response immediately after exposure
2.9 (1.1 ^a)	Shortened time to angina response 2 h after exposure
2.9 (1.1 ^a)	Exposure time to angina response
2.8 (1.1 ^a)	Decrease in mean exercise time until onset of intermittent claudications
2.7 (1.0 ^a)	Shortened time to angina response

^aHbCO% before exposure to CO.

Source: Adapted from WHO 1979a.

TABLE B-8. SUMMARY OF CLINICAL AND EPIDEMIOLOGIC STUDIES ON HUMAN EXPOSURE TO NITRIC OXIDE

Concentration in ppm	Duration of Exposure	Type of Exposure	Observed Effects	Remarks
112	3 min	Anesthesia accident	One patient showed signs of cyanosis and methemoglobinemia, followed 18 1/2 hours later by death. Autopsy indicated severe pulmonary edema. Second patient showed signs of cyanosis, but recovered fully following proper medical treatment.	Accidents due to contamination of nitrous oxide by nitric oxide, the analysis of which was not described.
3	Working lifetime	Occupational: Nitrogen fertilizer production	Exposed workers had higher carboxy- and methemoglobin levels in their blood compared with controls. Exposed workers developed pyroxidine deficiency.	Exposure to carbon monoxide, ammonia, and mixed oxides of nitrogen.
2-10	Unknown	Occupational: Arc welding	Slight increase in methemoglobin levels.	Exposure to mixed oxides of nitrogen.

Source: Adapted from NIOSH 1976, Table XIII-8, p. 178.

TABLE B-9. SUMMARY OF EFFECTS OF EXPOSURE TO NITRIC OXIDE
IN EXPERIMENTAL ANIMALS

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Observed Effects
5000-20000	Dog	Up to 50 min	Continuous	5000 ppm: Decreased arterial oxygen tension, rise in methemoglobin and arterial carbon dioxide tension. If exposure greater than 24 min, death occurred 7-120 min after exposure. 20000 ppm: Death in 15-50 minutes.
2500-5000	White mice	Up to 12 min	Continuous	Animals exposed at 5000 ppm died after 6-8 min. Animals exposed at 2500 ppm died after 12 min of exposure.
310-3500	White mice	Up to 8 hr	Continuous	LC50 = 320 ppm. All animals survived an 8-hr exposure at 310 ppm. At high concentrations, nitric oxide 4 times more toxic than nitrogen dioxide.
175-2100	Mice, guinea pig	Up to 6 hr	Continuous	Mice exposed at 2100 ppm for 30 min produced 80% methemoglobin. Exposure at 322 ppm for 6 hr produced 60% methemoglobin. No change in recovery of resting respiratory rhythm in guinea pigs at 175 ppm for 120-150 min.

Source: Adapted from NIOSH 1976, Table XIII-9, p. 179.

TABLE B-10. SUMMARY OF EPIDEMIOLOGIC AND EXPERIMENTAL STUDIES ON HUMAN EXPOSURE TO NITROGEN DIOXIDE

Concentration in ppm	Length of Exposure	Type of Exposure	Observed Effects	Remarks
Low Exposure = 0.106 High Exposure = 0.711	24 hr/day	Community: Ambient air near TNT plant	Higher incidence of acute respiratory disease in high exposure community compared with low exposure community, particularly in children below age 12. No difference in chronic respiratory disease between communities	Suspended nitrates and total suspended particulates higher in high exposure community compared with other communities. Concentrations of sulfur dioxide and other contaminants not reported
Less than 2.8	Unknown	Occupational: Printing shop and sulfuric acid plant	Dental erosion and gingivitis; emphysema and pulmonary tuberculosis; cardiovascular hypotonia and bradycardia; polycythemia rubra, granulocytosis, basophilia; decreased osmotic fragility of red blood cells, accelerated agglutination of the blood cells; reduced catalase index, reduced alkali reserve, reduced blood sugar	Workers probably exposed to sulfuric acid mists and sulfur dioxide at unknown concentrations
0.4-2.7	4-6 years	Occupational: Chemical works	Complaints of sporadic cough, mucopurulent expectoration, and dyspnea on exertion. Normal chest X-ray, spirometry, and blood pH. Carbon dioxide partial pressure and total carbonic acid in blood increased. Significant decrease in serum proteins and significant increase in urinary amino acids and glycoproteins	Conflicting results on the presence of chronic obstructive pulmonary disease. Total lack of environmental data

TABLE B-10 (Continued)

Concentration in ppm	Length of Exposure	Type of Exposure	Observed Effects	Remarks
0.5-5.0	15-60 min	Experimental: Continuous inhalation	Significant reduction in carbon monoxide diffusing capacity in 16 healthy male subjects exposed for 15 min at 5 ppm. Significant decrease in arterial oxygen partial pressure with corresponding increase in alveoloarterial oxygen pressure gradients in 14 chronic bronchitis patients exposed for 15 min at 5 ppm. Continued exposure to 60 min did not significantly change findings at 15 min. Increased airway resistance in 70 chronic bronchitis patients exposed at and above 1.5 ppm	
0.0-5.0	30 breaths or 15 min	Experimental: Continuous inhalation	Exposure at 1.5-5.0 ppm increased airway resistance. Significant decrease in arterial oxygen tension, and significant increase of end-expiratory arterial pressure at 4-5 ppm. No effects noted below 1.5 ppm	88 chronic bronchitis patients
4-5	10 min	Experimental: Continuous inhalation	Decrease in effective lung compliance with corresponding increase in expiratory and inspiratory maximum viscous response	5 healthy adult male subjects
2.0-10.3	Unknown	Occupational: Arc welding	Slight increase in methemoglobin levels in blood	Exposure to oxides of nitrogen

TABLE B-10 (Continued)

Concentration in ppm	Length of Exposure	Type of Exposure	Observed Effects	Remarks
4-20	Acute, duration not stated after exposure	Occupational: Open arc welding	Conjunctivitis and pharyngitis which subsided 18 hr after exposure	Exposure to oxides of nitrogen
62-158	3 separate exposures ranging from 10 min to 2 hr	Experimental: Continuous inhalation	62 ppm for 1 hour: Laryngeal irritation, but no other effects. 25-100 ppm for 2 hr: Marked mucosal irritation, increased pulse and respiratory rates. 158 ppm for 10 minutes: Coughing, irritation of nasal and laryngeal mucosa, lacrimation, headache, nausea, and vomiting. No delayed or long-term illness	Probable exposure to nitric oxide and air-borne nitric acid in addition to nitrogen dioxide
38-345	Working lifetime	Occupational: Shotfiring operations in coal miners	Forced Expiratory Volume (FEV 0.75) and vital capacity reduced, residual volume and total lung capacity increased relative to controls	Inappropriate control sample. Exposure to high levels of carbon monoxide and carbon dioxide in addition to "nitrous fumes"

Source: Adapted from NIOSH 1976, Table XIII-7, p. 176-177.

TABLE B-11. SUMMARY OF EFFECTS OF EXPOSURE TO NITROGEN DIOXIDE
IN EXPERIMENTAL ANIMALS

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
0.4	Guinea pigs	1 wk	Continuous	Protein level in lung lavage fluid	Animals exposed to nitrogen dioxide showed higher protein levels in lung lavage fluid than controls
0.5	Mice	3-12 months	6, 18, 24 hr/day	Alveolar size	Lung alveoli ex- panded in all mice exposed to nitrogen dioxide as compared with controls
0.5-1.0	Rats	1 hr at 1 ppm, 4 hr at 0.5 ppm	Continuous	Changes in mast cells of lung	Exposure at 1 ppm resulted in loss of cytoplasmic granules, rupture, and reduc- tion in number of mast cells. Exposure at 0.5 ppm for 4 hr resulted in de- granulation of mast cells

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
1.0	Rabbits	1 hr	Continuous	Changes in protein structure of lung tissue	Peak shift in absorbance spectrum in animals killed immediately after exposure. Absorbance spectrum returned to normal in animals killed 24-48 hrs after exposure
1	Guinea pigs	180 days	8 hr/day	Macro- and microscopic changes in the lung. Hematologic, urinary, and immunologic changes	Evidence of chronic respiratory disease such as bronchitis bronchopneumonia, extravasation of blood in lungs, and foci of emphysema. Urinary hydroxproline and acid mucopolysaccharides were increased. Decreased serum proteins, immunoglobins, and weight gain
1.3	Rats	3 months	12 hr/day	Reproductive effects	Significant changes in estrus, litter size, and fetal weights

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
2.5 and 3.5	Mice	2 hr	Continuous	Susceptibility to <u>Klebsiella pneumonnis</u>	Increased susceptibility at 3.5 ppm, but not at 2.5 ppm
0.5-12	Monkeys, dogs, rabbits, guinea pigs, rats	90 days	Continuous	Hematologic changes, weight gain, gross lung pathology	Bronchitis, broncho-pneumonitis, pneumonia, and foci of multinucleated cells noted in animals exposed at 12 ppm. No lung pathology observed in animals exposed at and below 5 ppm
1-14.8	Mice	1.9-14.8 ppm for 4 hr and 1, 2.3, 6.6 ppm for 17 hr	Continuous	Antibacterial activity of animals infected with radiophosphorus-labeled <u>Staphylococcus aureus</u>	Decreased bactericidal activity in animals infected then exposed to 7 ppm. Exposure at 2.3 ppm for 17 hr prior to infection also resulted in reduced bactericidal response

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
15 ± 2	Rats	1, 2, and 7 days	Continuous	Ultrastruc- tural changes of lung tissue	Bronchiolar epithe- lium was less columnar, brush cells increased in number, microvilli became smaller, and number of macrophages in- creased
3-16	Dogs	1 hr	Continuous	Microscopic changes of endothelial cells	Bleb formation, loss of pinocytic vesicles, and mitochondrial swelling. Exposure at 3 ppm resulted in bleb formation without other changes
5-16	Dogs and rabbits	1 hr	Continuous	Microscopic changes of capillary en- dothelium and alveolar epi- thelium	Exposure had greatest effect on capillary en- dothelium. Findings included bleb formation, endo- thelial cell organ- elles in the capillary lumens, and appear- ance of platelets and polymorphonuclear leukocytes in lumens of capillaries adjoining blebs

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
15-17	Rats	48 hr	Continuous	Macrophage division	Large increase in number of dividing macrophages, as well as total number of macrophages
15-25	Rats, guinea pigs, rabbits	2-hr expo- sures for 1 or 5 days	Continuous	Macro- and microscopic pulmonary changes	Pulmonary edema noted after one 2-hr exposure. Repair noted 2 weeks after exposure. Edema and inflamma- tion less severe after multiple 2-hr exposures than to single 2-hr exposure. Degree of morphologic change related to exposure concentration
25	Dogs	6 months	Continuous	Macro- and microscopic changes in pulmonary system	1 dog showed macro- scopic bullous em- physema. All dogs showed enlargement of alveoli

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
26	Dogs	191 days	15 ppm-cont. 40 ppm-int.: 1/2 hr every 2 hr for 4 1/2 hr	Macro- and microscopic changes in pulmonary system	1 dog showed bullous emphysema. Others showed a striking increase in the firmness of the lungs and emphysema, microscopically
8-40	Rabbits	3 hr	15 ppm-cont. 40 ppm-int.: 1/2 hr every 2 hr for 4 1/2 hr	Cellular dis- distribution in lung tissue	Significant increase in intraalveolar heterophiles from exposure at 8 ppm
10,22,36,45	Rats	Single 4-hr periods	Continuous	Microscopic changes in tracheal and lung tissue	Normal trachea and lungs 4-8 days after exposure
5-50	Rabbits	3 hr	Continuous	Phagocytic activity	Suppression of virus- induced resistance and phagocytic ac- tivity

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results
30-50	Guinea pigs	30-45 min	Continuous	Mortality due to inhaled acetylcholine	Exposure at 50 ppm resulted in signifi- cantly higher mortality in animals pretreated with nitrogen dioxide than in controls. No differences in mortal- ity between controls and pretreated groups at lower concentra- tions of nitrogen dioxide
20-70	Guinea pigs	30 min	Continuous	Antigen sensi- tization	Exposure at 70 ppm enhanced sensitization, 40 ppm and less did not
12.5-100	Rats	Until animals died or arbi- trary termi- nation of exposure	Continuous 24 hr/day, 7 days/wk	Microscopic changes in pulmonary system	Exposure at 100 ppm resulted in death within 24 hr. Rats exposed at 12.5 ppm had moderate hyper- trophy and hyperplasia of bronchial and bron- chiolar epithelium as well as irregular alveolar ducts and alveoli after 40 days of exposure

TABLE B-11 (Continued)

Concentration in ppm	Species	Duration of Exposure	Type of Exposure	Dependent Variable(s)	Results	
115-416	Rats, dogs, guinea pigs	5-60 min	Continuous	Mortality	<u>Time</u>	LC50
						<u>Rat</u>
					5 min	416 ppm
					15 min	201 ppm
					30 min	162 ppm
					60 min	115 ppm
						<u>Guinea Pig</u>
					15 min	315 ppm
					Threshold of toxicity approximately 25% of LC50 levels for rats. At these levels, dogs showed no gross or microscopic changes, rats showed some pulmonary edema	
					88-1445	Rats
2 min	1445 ppm					
5 min	833 ppm					
15 min	420 ppm					
30 min	174 ppm					
60 min	168 ppm					
240 min	88 ppm					

Source: Adapted from NIOSH 1976, Table XIII-10, p. 180-189.

TABLE B-12. SUMMARY OF DIESEL EMISSION LEVELS AND EXPOSURE LIMITS OF ALDEHYDES^a

Chemical	Concentration (mg/m ³)	Concentration (ppm)	Comments	Reference
ACETALDEHYDE				
			<u>Emission levels</u>	
	-	3.2	Engine not identified	Vogh 1969
	-	a) 4.8	a) Cetane No. 40	Iwai et al. 1976
	-	b) 2.6	b) Cetane No. 45	
	-	c) 0.5	c) Cetane No. 55	
			Direct injection. 12 L, V6 and V10, 500 rpm	
	0.4-3.3	-	1.4L, direct inject.	Springer and Baines 1978
	1.6-4.1	-	5.7L, direct inject.	
	-	0.56-0.76	No. 2 diesel fuel. Engine not identified	Creech et al. 1982
			<u>Exposure limits</u>	
	360	200	OSHA ^b 8-hr TWA	OSHA 1982
	270	150	ACGIH ^c STEL	ACGIH 1982
	180	100	ACGIH 8-hr TLV	ACGIH 1982
	-	-	No MSHA ^d standard	MSHA 1982
ACROLEIN				
			<u>Emission levels</u>	
	-	1.6-11.4	2 cycle, 6 cyl, 220 hp. No. 2 fuel, 50-cetane	Linnell and Scott 1962a
		2.9	Engine not identified	Vogh 1969
	0.5-0.8	0.2-0.3	1972 Mercedes Benz 220 auto	Smythe and Karasek 1973
	-	a) 7.5	a) Cetane No. 40	Iwai et al. 1976
	-	b) 4.3	b) Cetane No. 45	
	-	c) 2.1	c) Cetane No. 55	
			Direct injection. 12L, V6 and V10, 500 rpm	
	-	a) 8.0-8.4	4 cycle engine, idle	EPA 1978
	-	b) 2.1-2.8	4 cycle engine, half load	
	-	c) 3.7-3.9	2 cycle engine, idle	
	-	d) 4.1-5.0	2 cycle engine, full load	
			<u>Exposure limits</u>	
	0.25	0.1	OSHA 8-hr TWA	OSHA 1982
	0.8	0.3	ACGIH STEL	ACGIH 1982
	0.25	0.1	ACGIH 8-hr TLV	ACGIH 1982
	-	-	No MSHA standard	MSHA 1982

TABLE B-12 (continued)

Chemical	Concentration (mg/m ³)	(ppm)	Comments	Reference
ALDEHYDES (total)				
			<u>Emission levels</u>	
-	9-97		4 cycle, direct inject., nat. aspir.	Marshall and Fleming 1971
-	6-53		4 cycle, direct inject. turbo	Marshall and Fleming 1971
-	3-26		4 cycle precombustion, turbo	Marshall and Fleming 1971
-	a) 35		a) Cetane No. 40	Iwai et al. 1976
-	b) 22		b) Cetane No. 45	
-	c) 9		c) Cetane No. 55	
			Direct injection, 12L V6 and V10, 500 rpm	
-	a) 56.4-58.8		4 cycle engine, idle (range for No. 1 and No. 2 fuels)	EHA 1978
-	b) 13.8-17.3		4 cycle engine, half load	EHA 1978
-	c) 15.2-18.3		2 cycle engine, idle	EHA 1978
-	d) 25.3-30.3		2 cycle engine, full load	EHA 1978
-	1.3		5.7 L automobile (FTP)	Williams and Chock 1980
	4.2-4.7		Engine not identified	Creech et al. 1982
			<u>Exposure limits</u>	
-	-		No OSHA standard	OSHA 1982
10	-		Diluted exhaust	MSHA 1982
-	-		No ACGIH recommendation	ACGIH 1982
BENZALDEHYDE				
			<u>Emission levels</u>	
-	0.3		Engine not identified	Vogh 1969
0.2-1.5	-		1.4L, direct inject.	Springer and Baines 1978
0.3-0.6	-		1.4L, direct inject.	
-	0.05-0.07		Engine not identified	Creech et al. 1982
			<u>Exposure limits</u>	
-	-		No OSHA standard	OSHA 1982
-	-		No MSHA standard	MSHA 1982

TABLE B-12 (continued)

Chemical	Concentration (mg/m ³)	Concentration (ppm)	Comments	Reference
BUTYRALDEHYDE				
		<u>Emission levels</u>		
-	0.3		n-Butyraldehyde. Engine not identified	Vogh 1969
-	0.08-0.13		iso-Butyraldehyde. Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
-	-		No OSHA standard	OSHA 1982
-	-		No MSHA standard	MSHA 1982
CROTONALDEHYDE				
		<u>Emission levels</u>		
-	0.4		Engine not identified. Analytically not separable from valeraldehyde	Vogh 1969
0.2-3.2	0.7-0.6		1973 Mercedes Benz 220 auto	Smythe and Karasek 1973
-	a) 0.8		Cetane No. 40	Iwai et al. 1976
-	b) 0.5		Cetane No. 45	
-	c) 0.1		Cetane No. 55	
			Direct injection. 12L, V6 and V10, 500 rpm	
0.46-1.34	-		1.4L, direct inject.	Springer and Baines 1978
0.44-1.41	-		5.7L, direct inject.	
-	0.02-0.04		Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
6	2		OSHA 8-hr TWA	OSHA 1982
6	2		ACGIH 8-hr TLV	ACGIH 1982
18	6		ACGIH STEL	ACGIH 1982
-	-		No MSHA standard	MSHA 1982
FORMALDEHYDE				
		<u>Emission levels</u>		
-	18.3		Engine not identified	Vogh 1969
-	1.8-28.4		2 cycle, 6 cyl. 220 hp. No. 2 fuel, 50-cetane	Linnell and Scott 1962a
1.24-12.33	-		1.4L, direct inject.	Springer and Baines 1978
1.41-12.05	-		5.7L, direct inject.	
-	3.26-3.61		Engine not identified	Creech et al. 1982

TABLE B-12 (continued)

Chemical	Concentration (mg/m ³)	Concentration (ppm)	Comments	Reference
FORMALDEHYDE (cont.)				
		<u>Emission levels (cont.)</u>		
-	-	a) 18	a) Cetane No. 40	Iwai et al. 1976
-	-	b) 11.5	b) Cetane No. 45	
-	-	c) 5.1	c) Cetane No. 55, direct inject V6, V10, 500 rpm	
20-30	16-24		1973 Mercedes Benz 220 auto	Smythe and Karasek 1973
-	-	a) 32.3-34.9	4 cycle, idle	EHA 1978
-	-	b) 9.2-12.1	4 cycle, half load	EHA 1978
-	-	c) 10.4-12.6	2 cycle, idle	EHA 1978
-	-	d) 19.3-23.3	2 cycle, full load	EHA 1978
		<u>Exposure limits</u>		
-	-	a) 10	a) OSHA 30 min ceiling value	OSHA 1982
-	-	b) 5	b) OSHA ceiling value	
-	-	c) 3	c) OSHA 8 hr TWA	
-	-	-	No standard for underground mining	MSHA 1982
1.2	1.0		NIOSH 30 min ceiling, recommendation	NIOSH 1982
3.0	2		ACGIH ceiling (current)	ACGIH 1982
1.5	1.0		ACGIH ceiling (proposed)	ACGIH 1982
HEXALDEHYDE				
		<u>Emission levels</u>		
-	0.2		Engine not identified	Vogh 1969
0.05-0.09	-		5.7L, direct inject.	Springer and Baines 1978
-	0.01		Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
-	-		No OSHA standard	OSHA 1982
-	-		No MSHA standard	MSHA 1982
PROPIONALDEHYDE				
		<u>Emission levels</u>		
-	0.03-0.08		Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
-	-		No OSHA standard	OSHA 1982
-	-		No MSHA standard	MSHA 1982

TABLE B-12 (continued)

Chemical	Concentration (mg/m ³) (ppm)		Comments	Reference
TOLUALDEHYDE				
		<u>Emission levels</u>		
-		0.1	para-Isomer. Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
-		-	No OSHA standard	OSHA 1982
-		-	No MSHA standard	MSHA 1982
VALDERALDEHYDE				
		<u>Emission levels</u>		
-		0.01-0.03	iso-Isomer. Engine not identified	Creech et al. 1982
-		0.01-0.02	n-Isomer. Engine not identified	Creech et al. 1982
		<u>Exposure limits</u>		
-		-	No OSHA standard	OSHA 1982
175	50		ACGIH 8 hr TLV	ACGIH 1982
-		-	No MSHA standard	MSHA 1982

^aLiterature data, arranged alphabetically by compound. Engine type described in comments.

^bOccupational Safety and Health Administration.

^cAmerican Conference of Governmental Industrial Hygienists

^dMine Safety and Health Administration.

TABLE B-13. SUMMARY OF HEALTH EFFECTS OF ALDEHYDES^a

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Carcinogenicity:</u>						
Human	Acrolein				Insufficient data for assessment of carcinogenicity	IARC 1979
Hamster	Acet.	2500-1650 ppm	7 hr/d, 5 d/wk, 52 wk	inhalation	Nasal and laryngeal carcinomas formed	Feron et al. 1982
	Acrolein	9.2 mg/m ³ (4 ppm)	7 hr/d, 5 d/wk, 52 wk	inhalation	No significant increase in tumors of the respiratory tract	Feron and Kruyssse 1977
	Form.	10 ppm	5 hr/d, 5 d/wk, life-time	inhalation	No tumors of respiratory tract	Dalbey 1982
	Form.	30 ppm	5 hr/d, life-time	inhalation	No tumors of respiratory tract	Dalbey 1982
	Form.	0.1-2.5 µg/mL	24 hr	in vitro	Initiated transformation of C3H/10T _{1/2} cells by TPA	Boreiko et al 1980
Mouse	Acet.	10-100 µg/ml	24 hr	in vitro	Initiated transformation of C2H/10 T _{1/2} cells by tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA)	Abernethy et al. 1982
	Form.	2.1-14.1 ppm	6 hr/d, 5 d/wk, 18 mo	inhalation	Not tumorigenic	Swenberg et al. 1980
Rat	Form.	2.1 ppm, 5.6 ppm, 14.1 ppm	6 hr/d, 5 d/wk, 18 mo	inhalation	Hyperplastic and metaplastic lesions of nasal epithelium seen at 2.1 and 5.6 ppm and significant number of nasal carcinomas seen at 14.1 ppm	Swenberg et al. 1980
<u>Cardiovascular Effects:</u>						
Rat	Acet.	0.5-30 µg/ml	1 min intervals	inhalation	≥3.0 µg/ml resulted in significant increases in SP. 12 and 30 µg/ml increased heart rate significantly	Egle 1972
	Propion.	3.0-200 µg/ml	1 min intervals	inhalation	≥10.0 µg/ml resulted in significant increase in BP. ≥20 µg/ml increased heart rate significantly.	Egle 1972
<u>Hematological Effects:</u>						
Hamster	Acet.	4560 ppm	6 hr/d, 5 d/wk, 90 days	inhalation	Females showed slightly increased erythrocyte counts and slightly decreased leukocyte counts. Serum enzymes normal except for slightly raised alkaline phosphatase	Kruiysse et al. 1975

TABLE B-13 (continued)

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Hematological Effects</u> (continued)						
Rat	Acet.	5000 ppm	6 hr/d, 5 d/wk, 4 wk	inhalation	Increased neutrophils and decreased lymphocytes	Appleman et al. 1982
<u>Kidney:</u>						
Hamster	Acet.	4560 ppm	6 hr/d, 5 d/wk, 90 days	inhalation	Increase in relative weight, but no pathological changes. Urine reduced in volume and darker in color. Glutamic- oxalacetic acid transaminase activity increased.	Kruyssen et al. 1975
Rat	Acrolein	>100 mg/m ³	30 min	inhalation	Hyperemia	Skog 1950
	Croton.	>100 mg/m ³	30 min	inhalation	Hyperemia and hemorrhages	Skog 1950
	Form.	>600 mg/m ³	30 min	inhalation	Perivascular edema	Skog 1950
	Propion.	>32000 mg/m ³	30 min	inhalation	Hyperemia	Skog 1950
<u>Lethality:</u>						
Human	Acet.	4570 mg/kg	-	-	LD50	Dreisbach 1980
	Acrolein	7 mg/kg	-	-	LD50	Dreisbach 1980
	Croton.	6 mg/kg	-	-	LD50	Dreisbach 1980
Rat	Acet.	24 g/m ³ (13,300 ppm)	4 hr	inhalation	LD50	Appleman et al. 1982
	Acrolein	18.4 mg/m ³ (8 ppm)	-	inhalation	Lethal to 2-4 of 6 animals	Carpenter et al. 1949
	Acrolein	4.9 ppm	6 hr/d, 5 d/wk, 13 wk	inhalation	Lethal to 6 of 12 animals	Feron et al. 1978
	Form.	250 ppm	4 hr	inhalation	Lethal to 2-4 of 6 animals	Carpenter et al. 1949

TABLE B-13 (continued)

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Liver:</u>						
Rat	Acet.	>14000 mg/m ³	30 min	inhalation	Perivascular edema	Skog 1950
	Acrolein	>100 mg/m ³	30 min	inhalation	Hyperemia	Skog 1950
	Butyr.	>66000 mg/m ³	30 min	inhalation	Slight focal hyperemia; degenerative vacuolation of hepatocytes	Skog 1950
	n-Butyr.	1000 ppm	12 x 6 hr	inhalation	No toxicity	Gage 1970
	Croton	>100 mg/m ³	30 min	inhalation	Hyperemia and hemorrhages	Skog 1950
	Form.	>600 mg/m ³	30 min	inhalation	Hyperemia, perivascular edema, and necrosis	Skog 1950
	Propion.	>32000 mg/m ³	30 min	inhalation	Hyperemia	Skog 1950
	Propion.	1300 ppm	6 x 6 hr	inhalation	Liver cell vacuolation	Gage 1970
<u>Lungs and Upper Respiratory Tract:</u>						
Hamster	Acet.	a) 330 ppm	6 hr/d,	inhalation	a) No toxic effect	Kruyssen et al. 1975
		b) 1340 ppm	5 d/wk,			
		c) 4560 ppm	90 days		b) Slight hyper- and metaplastic changes in tracheal epithelium c) Necrosis, inflammatory changes and hyper- and metaplastic changes in nasal cavity, larynx, bronchi and lung epithelium.	
Rat	Acet.	>14000 mg/m ³	30 min	inhalation	Lethal levels resulted in hemorrhages, hyperemia and intraalveolar and perivascular edema	Skog 1950
	Acet.	a) 400 ppm	6 hr/d,	inhalation	a) Slight degeneration of nasal olfactory epithelium b) Slight to moderate degeneration of nasal epithelium c) Degeneration of nasal epithelium and larynx and trachea d) Severe degeneration of nasal, larynx, and tracheal epithelium	Appelmann et al. 1982
		b) 1000 ppm	5 d/wk,			
	c) 2200 ppm	4 wk				
	Acrolein	>100 mg/m ³	30 min	inhalation	Hyperemia, hemorrhages, edema and possibly degenerative changes in the bronchial epithelium	Skog 1950

TABLE B-13 (continued)

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Lungs and Upper Respiratory Tract (continued)</u>						
Rat, Rabbit, Hamster	Acrolein	0.4, 1.4, 4.9 ppm	6 hr/d, 5 d/wk, 13 wk	inhalation	Histopathological changes (hyper and metaplasia of epithelium) seen in all species at 4.9 ppm and in rats at 0.4 ppm	Feron et al. 1978
Rat	Butyr.	>66000 mg/m ³	30 min.	inhalation	Perivascular interstitial edema and changes in the walls of the venae in the form of swelling and vacuolation of smooth muscle cells.	Skog 1950
	Croton.	>100 mg/m ³	30 min	inhalation	Hyperemia and hemorrhages but no edema	Skog 1950
	Form.	>600 mg/m ³	30 min	inhalation	Lethal levels resulted in hemorrhages and intraalveolar and perivascular edema.	Skog 1950
	Propion.	>32000 mg/m ³	30 min	inhalation	Lethal levels resulted in bronchitis and bronchopneumonia	Skog 1950
<u>Mutagenicity:</u>						
Bacteria (E. coli)	Acet.	0.1%	-	-	Increased the spontaneous mutation frequency by 4.5 times	Igali and Grazso 1980
Bacteria (S. typhimurium)	Acet.	0.01-2.5 µmol/plate	-	-	Nonmutagenic with and without metabolic activation.	Pool and Wiessler 1981
	Acrolein	a) 3 µmoles/plate b) 0.3 µmoles/plate	-	Ames assay	a) Highly toxic b) Not mutagenic to TA98, TA100, TA1535 and TA1537 in spot test	Florin et al. 1980
	Acrolein	0.05-0.15 µmol/1 ml	90 min	Ames assay	Mutagenic to TA100 without metabolic activation. S-9 mix eliminated mutagenicity	Lutz et al. 1982
	Benz.	3 µmole/plate	-	Ames assay	Not mutagenic to TA98, TA100, TA1535 and TA1537 with and without metabolic activation	Florin et al. 1980
	Butyr.	3 µmole/plate	-	Ames assay	Not mutagenic to TA98, TA100, TA1535, and TA1537 with and without metabolic activation	Florin et al. 1980
	Croton.	0.5-1.5 µmole/2 ml	90 min	Ames assay	Mutagenic to TA100 without S-9 mix. S-9 mix reduced mutagenicity.	Neudecker et al. 1981; Lutz et al. 1982
	Form.	0-8 µg/mL	120 min	-	Weak mutagen to strain TM 677	Boreiko et al. 1980
	Propion.	3 µmole/plate	-	Ames assay	Not mutagenic to TA98, TA100, TA1535, and TA1537 with and without metabolic activation	Florin et al. 1980

TABLE B-13 (continued)

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Mutagenicity (continued)</u>						
Human	Form.	10-500 ppm	24-48 hr	<u>in vitro</u>	Dose-dependent increase in sister chromatid exchanges in lymphocytes	Obe and Beek 1979
Hamster	Acet. Form.	2.5-15 ppm 1-4 ppm	24 hr 24 hr	<u>in vitro</u> <u>in vitro</u>	Dose-dependent increase in sister chromatid exchanges in ovary cells	Obe and Beek 1979
Rat	Acet.	a) 10^{-4} - 10^{-2} M b) 10^{-5} - 10^{-3} M	12 hr, 24 hr, 48 hr	<u>in vitro</u>	a) Dose-dependent increase in micronuclei in skin fibroblast cell cultures b) Dose-related increase in metaphases with chromosomal aberrations	Bird et al. 1982
<u>Nervous System:</u>						
Mouse	Acet.	a) 2.2 mg/kg b) 2.7 mg/kg c) 100 mg/kg		iv	a) ED ₅₀ for depression of fine motor activity b) ED ₅₀ for depression of gross motor activity c) Profound behavioral depression	Holtzman and Schreider 1974
Rat	a) Acet., propion. and butyr. b) Form., croton., and acrolein	Lethal doses	30 min	inhalation	a) Strong narcotic effect b) Mild narcotic effect	Skog 1950
<u>Pulmonary Function:</u>						
Human	Acrolein	0.09-0.30 ppm	-	inhalation	Respiratory frequency decreased with increasing concentration	Weber-Tschopp et al. 1977
	Form.	0.45 mg/m ³ (0.36 ppm)	8 hr work day	inhalation	Pulmonary effects suggestive of bronchoconstriction	Alexandersson et al. 1982
	Form.	>1 mg/m ³ (>0.8 ppm)	5 hr	inhalation	Resistance to breathing	Andersen 1978
Mice	Acrolein	1.7 ppm	10 min	inhalation	50% reduction in resp. rate	Kane and Alarie 1977
	Form.	3.1 ppm	10 min	inhalation	50% reduction in resp. rate	Kane and Alarie 1977

TABLE B-13 (continued)

Species	Aldehyde	Dose	Exposure	Route	Effects	Reference
<u>Sensory and Respiratory Tract Irritation</u>						
Human	Acet.	50 ppm	15 min	inhalation	Eye irritation	Henschler (no date)
	Acet.	25 ppm	15 min	inhalation	Eye irritation, sensitive persons	Henschler (no date)
	Acet.	200 ppm	15 min	inhalation	Threshold effect for nose and throat irritation	Henschler (no date)
	Acet.	134 ppm	30 min	inhalation	Threshold effect for respiratory tract irritation	Henschler (no date)
	Acrolein	2.5 mg/m ³ (1 ppm)	5 min	inhalation	Causes lachrymation and eye, nose, and throat irritation	Sim and Pattie 1957
	Acrolein	0.30 ppm	10 min	inhalation	Acute irritation	Weber-Tschopp et al. 1977
	Acrolein	a) 1-2 ppm b) 0.5 ppm c) 0.5 ppm	5 min 5 min 12 min	vapor vapor vapor	Severe eye irritation Mild eye irritation Severe eye irritation	Stephens et al. 1961
	Butyr.	690 mg/m ³ (234 ppm)	30 min	inhalation	No irritation	Sim and Pattie 1957
	Croton.	12 mg/m ³ (4 ppm)	10 min	inhalation	Irritation of eye, nose, and upper respiratory tract	Sim and Pattie 1957
	Form.	17.3 mg/m ³ (12 ppm)	30 min	inhalation	Eye and nasal irritation	Sim and Pattie 1957
	Form.	a) 4-5 ppm b) 1-4 ppm	5 min 5 min	vapor vapor	Severe eye irritation Mild eye irritation	Stephens et al. 1961
	Propion.	324 mg/m ³ (133 ppm)	30 min	inhalation	Only mildly irritating	Sim and Pattie 1957
Mice	Acrolein	0.85-3.4 ppm	10 min	inhalation	In the presence of 10-140 ppm SO ₂ a ratio-dependent antagonistic effect occurred	Kane and Alarie 1979
<u>Teratogenicity:</u>						
Rat	Acet.	50-100 mg/kg/day	10th-12th day of gestation	ip	Teratogenic	Sreenathan et al. 1982
Mouse	Acet.	0.1 ml of 1% or 2% in saline/25 g body weight	7th, 8th, 9th day of gestation	iv	10 day old embryos exhibited abnormalities of the CNS	O'Shea and Kaufman 1979
Chicken	Acrolein	0.05 µmol/egg	-	injection	LD ₅₀ , injected into air space of egg. No teratogenic effects.	Kankaanpaa et al. 1979
Rat	Acrolein	50-250 µM	10.5-12.5th day of gestation	in culture	No teratogenic effects	Schmid et al. 1981
Dog	Form.	125, 375 ppm	4-56th day of gestation	oral	No malformations	Rurni and Ohder 1973

*Table arranged alphabetically by effect.

TABLE B-14. SUMMARY OF STATE^a AND FEDERAL
AIR QUALITY STANDARDS AND MONITORING METHODS FOR SULFATES

State	Standard	Monitoring Method
Alabama	None	-
Alaska	None	None
Arizona	None	Barium sulfate turbidimetric analysis
Arkansas	None	Technicon autoanalysis of TSP samples
California	25 µg/m ^{3b}	Barium sulfate turbidimetric method. Automated methylthymol blue.
Colorado	None	None
Connecticut	None	Turbidimetric
Delaware	None	Colorimetric analysis of rainwater
Florida	None	None
Georgia	None	None
Hawaii	None	-
Idaho	None	-
Illinois	None	Ion chromatography of TSP samples
Indiana	None	Ion chromatography; methylthymol blue autoanalysis
Iowa	None	None
Kansas	None	Ion chromatography of TSP samples
Kentucky	None	Methylthymol blue autoanalysis of TSP
Louisiana	None	None
Maine	None	Methylthymol blue autoanalysis
Maryland	None	Methylthymol blue analysis of TSP samples
Massachusetts	None	Analysis of TSP samples
Michigan	None	Ion chromatography
Minnesota	None ^c	None
Mississippi	None	None
Missouri	None	-
Montana	None	-
Nebraska	None	None
Nevada	None	Barium sulfate turbidimetric
New Hampshire	None	Nephelometry of TSP samples

TABLE B-14 (continued)

State	Standard	Monitoring Method
New Jersey	None	Ion chromatography of rainwater
New Mexico	None	None
New York	None	Turbidimetric
North Carolina	None	None
North Dakota	4 $\mu\text{g}/\text{m}^3$ ^d 12 $\mu\text{g}/\text{m}^3$ ^e	Automated methylthymol blue method
Ohio	None	Methylthymol blue method for TSP
Oklahoma	None	None
Oregon	None	Chromatographic analysis of cellulose acetate filter extracts
Pennsylvania	10 $\mu\text{g}/\text{m}^3$ ^f 30 $\mu\text{g}/\text{m}^3$ ^b	Colorimetry - methylthymol blue
Rhode Island	None	Barium sulfate - turbidometric
South Carolina	None	Automated methylthymol blue method
South Dakota	None	None
Tennessee	None	None
Texas	None	-
Utah	None	None
Vermont	None	Colorimetric
Virginia	None	None
Washington	None	Colorimetry - barium chloranilate
West Virginia	None	Ion chromatography of TSP samples
Wisconsin	None	Ion chromatography of TSP samples
Wyoming	None	-
Federal	None	-

^aInformation obtained through personal contacts with appropriate state agencies.

^b24-hr average.

^cAcid deposition standard will be implemented in 1986.

^dMaximum annual arithmetic mean.

^e24-hr concentration not to be exceeded 1% of the time.

^f30-day average.

TABLE B-15. SUMMARY OF HEALTH EFFECTS OF SULFATES

Species	Compound	Dose (aerosol size)	Exposure	Route	Effects	Reference
<u>Lethality:</u>						
Guinea pig	Sulfuric acid	a) 30 mg/m ³ (0.8 μm) b) 109 mg/m ³ (0.4 μm)		inhal.	a) LC ₅₀ b) 50% mortality not reached. Higher concentrations increased particle size.	Wolff et al. 1979
Guinea pig	Sulfuric acid	22.1 ppm (<2 μm)	2.75 hr	inhal.	Lowest lethal dose	Treon et al. 1950
Guinea pig	Sulfuric acid	100 mg/m ³	-	inhal.	LC ₅₀	Schwartz et al. 1979
Mouse	Sulfuric acid	140 ppm (<2 μm)	3.5 hr	inhal.	Lowest lethal dose	Treon et al. 1950
Rabbit	Sulfuric acid	374 ppm (<2 μm)	3.5 hr	inhal.	Lowest lethal dose	Treon et al. 1950
Rat	Sulfuric acid	178 ppm	7 hr	inhal.	Lowest lethal dose	Treon et al. 1950
<u>Hematology/Blood Chemistry:</u>						
Human	Sulfuric acid	100 μg/m ³	4 hr	inhal.	No significant changes indicative of tissue damage	Chaney et al. 1980
Mice	Sulfuric acid	125-154 mg/m ³	10-14 days	inhal.	Temporary elevation in WBC, PCV and plasma protein Neutropenia and lymphophilia as exposure progressed	Schwartz et al. 1979
Rat	Sulfuric acid	6.3, 6.6 mg/m ³	6, 13 wk	inhal.	Metabolic alkalosis	Lewkowski et al. 1979
<u>Mutagenicity:</u>						
Bacteria	Ammonium sulfate	2.-10%	2-4 days	Ames assay	Not mutagenic	Litton Bionetics Inc. 1975
Yeast	Ammonium sulfate	2.5-10%	3-5 days	-	Not mutagenic	Litton Bionetics Inc. 1975
<u>Pulmonary Effects:</u>						
Human	Ammonium bisulfate	4-47 μg/m ³	2 hr/d, 2-3 days	inhal.	Few if any significant changes in pulmonary function were seen in normal, sensitive, or asthmatic subjects	Avol et al. 1979
Human	Ammonium sulfate	117-337 μg/m ³	2 hr/d, 2-3 days	inhal.	Few if any significant changes in pulmonary function were seen in normal, sensitive, or asthmatic subjects	Avol et al. 1979
Donkey	Ammonium sulfate	≤2000 μg/m ³ (0.3-0.6 μm)	1 hr	inhal.	No effect on bronchial clearance, resistance, compliance, or deposition	Schlesinger et al. 1978

TABLE B-15 (continued)

Species	Compound	Dose (aerosol size)	Exposure	Route	Effects	Reference
<u>Pulmonary Effects</u> (continued)						
Rat	Ammonium sulfate	0.01-1.0 μmole		intra-tracheal, in vitro	1 μmole caused a reduction in respiratory volume probably as a result of the release of histamine stores.	Charles et al. 1977
Rat	Sodium sulfate	1 nmole	30 min		Cobalt, mercury, cadmium, and nickel increased sulfate adsorption substantially; iron and zinc to a lesser degree and manganese not at all.	Charles et al. 1977
Human	Sulfuric acid	93-111 $\mu\text{g}/\text{m}^3$	2 hr/d 2-3 days	inhal.	Few if any significant changes in pulmonary function were seen in normal, sensitive, and asthmatic subjects.	Avol et al. 1979
Human	Sulfuric acid	110 $\mu\text{g}/\text{m}^3$	1 hr	inhal.	Transitory alterations in mucociliary clearance	Leikauf et al. 1981
Human	Sulfuric acid	1000 $\mu\text{g}/\text{m}^3$	1 hr	inhal.	Decrease in mucociliary clearance	Leikauf et al. 1981
Human	Sulfuric acid	100 $\mu\text{g}/\text{m}^3$ (0.1-0.3 μm)	4 hr	inhal.	No adverse effects on pulmonary function	Kerr et al. 1981
Human	Sulfuric acid	1.0 mg/m^3	10 min	inhal.	No significant adverse pulmonary effects	Sackner et al. 1978
Human	Sulfuric acid	233-1107 $\mu\text{g}/\text{m}^3$ (0.91-0.93 μm)	2 hr	inhal.	No significant change in pulmonary function	Horvath et al. 1982
Human	Sulfuric acid	0.35-5 mg/m^3 (1 μm)	5-15 min	inhal.	77% average retention of acid. Lowest dose produced shall lower rapid breathing	Amdur et al. 1952b
Human	Sulfuric acid	108 xg/m^3 (0.5 xm)	4 hr	inhal.	No effect	Horstman et al. 1982
Donkey	Sulfuric acid	194-1364 $\mu\text{g}/\text{m}^3$	1 hr	inhal.	Decrease in bronchial clearance rate. No effect on pulmonary resistance, dynamic compliance, or regional deposition.	Schlesinger et al. 1978

TABLE B-15 (continued)

Species	Compound	Dose (aerosol size)	Exposure	Route	Effects	Reference
<u>Pulmonary Effects (continued)</u>						
Guinea pigs	Sulfuric acid	0.1-1 mg/m ³ a) 0.3 μm b) 1.0 μm	1 hr	inhal.	a) Increase in pulmonary flow resistance and decrease in pulmonary compliance. b) Observed effects not as large as those seen at 0.3 μm	Amdur et al. 1978a
Guinea pig	Sulfuric acid	a) 1.2 mg/m ³ b) 14.6, 24.3, 48.3 mg/m ³ (1.0 μm)	1 hr	inhal.	a) No pulmonary function changes b) 8 of 27 animals showed a dose-independent increase in total pulmonary resistance and decrease in dynamic compliance.	Silbaugh et al. 1981a
Guinea pig	Sulfuric acid	32.6 mg/m ³ (1.0 μm)	4 hr	inhal.	Bronchiolar epithelial desquamation. Lung lesions were prevented by pretreatment with atropine.	Brownstein 1980.
<u>Respiratory Irritation:</u>						
Human	Sulfuric acid	233-1107 μg/m ³ (0.92 μm)	2 hr	inhal.	Throat and respiratory irritation, but no adverse changes in pulmonary function.	Horvath et al. 1982
Human	Sulfuric acid	a) 4.1-39 mg/m ³ b) 2.9-39 mg/m ³ (1 xm) c) 11.5-38 mg/m ³ (1.5 xm)	a) 10 min b) 60 min c) 30-60 min	inhal.	a) Coughing, bronchoconstriction, rales b) Coughing, bronchoconstriction, rales (62% rel. humidity) c) Severe coughing, lacrimation and rhinorrhea (91% rel. humidity)	Sim and Pattle 1957
<u>Susceptibility to Infections:</u>						
Mouse	Ammonium sulfate	<5.3 mg/m ³	3 hr	inhal.	No effect on mortality or survival time following exposure to infectious bacteria.	Ehrlich 1980
Mouse	Zinc ammonium sulfate	≥2.1 mg/m ³	3 hr	inhal.	≥2.1 mg/m ³ resulted in significant increase in mortality and reduction in survival time following exposure to infectious bacteria.	Ehrlich 1980
Mouse	Zinc sulfate	≥1.2 mg/m ³	3 hr	inhal.	Significant increase in mortality and reduction in survival time following exposure to infectious bacteria.	Ehrlich 1980

TABLE B-15 (continued)

Species	Compound	Dose (aerosol size)	Exposure	Route	Effects	Reference
<u>Teratogenicity:</u>						
Mice	Sulfuric acid	5-20 mg/m ³	7 hr/d 6-15th day of gestation	inhal.	Not teratogenic	Murray et al. 1979
Rabbit	Sulfuric acid	5-20 mg/m ³	7 hr/d 6-18th day of gestation	inhal.	Not teratogenic	Murray et al. 1979

LIST OF ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
A/F	air-to-fuel ratio
Al	aluminum
AMER	acceptable maximum emission rate
bhp	brake horsepower
Ba	barium
bsfc	brake specific fuel consumption
C	carbon
Ca	calcium
CHO	aldehyde group
°C	degree Celsius
CaSO ₄	calcium sulfate
cfm/hp	cubic feet per min per rated horsepower
CO	carbon monoxide
CO ₂	carbon dioxide
Cr	chromium
Cu	copper
dB	decibel
dBA	decibel-adjusted
DF	diesel fuel
DF-A	diesel fuel - arctic grade
DF-1	diesel fuel - winter grade
DF-2	diesel fuel - No. 2 grade
DF-2 CONUS	diesel fuel No. 2, continental U.S.
DF-2 OCONUS	diesel fuel No. 2, outside continental U.S.

DNPB	2,4-dinitrophenylhydrazine
DOAS	diesel odor analysis system
EGR	exhaust gas recirculation
EPA	U.S. Environmental Protection Agency
°F	degree Fahrenheit
Fe	iron
ft ³	cubic feet
g	gram
gal	gallon
g/bHP hr	grams per brake horsepower hours
g/cm ³	grams per cubic centimeter
g/hr	grams per hour
g/hr/hp	grams per hour per rated horsepower
g/kg	grams per kilogram
g/km	grams per kilometer
g/mi	grams per mile
g/mL	grams per milliliter
H	hydrogen
Hb	hemoglobin
HbCO	carboxyhemoglobin
HC	hydrocarbons
H ₂ O	water
H ₂ S	hydrogen sulfide
H ₂ SO ₄	sulfuric acid

hp	horsepower
HPLC	high precision liquid chromatography
hr	hour
in ³	cubic inches
iv	intravenous
kW	kilowatt
L	liter
lb	pound
lbm	pound-mass
lbm/lb-mole	pound-mass per pound mole
lb/hr	pound per hour
lb-ft	pounds-feet
LCO	liquid chromatography - oxygenates
LD50	lethal dose - 50% of test animals
MBTH	3-methyl-2-benzothiazolone
MetHb	methemoglobin
mg/kg	milligrams per kilogram
mg/m ³	milligrams per cubic meter
µg	microgram
µg/m ³	micrograms per cubic meter
µm	micron
µmol/L	micromoles per liter
mi/gal	miles per gallon
min	minute
mL	milliliter
Mn	manganese
mol	moles

MSHA	Mine Safety and Health Administration
N	nitrogen
Ni	nickel
N-m	newton-meter
NaSO ₄	sodium sulfate
NDIR	nondispersive infrared absorption spectroscopy
NIOSH	National Institute of Occupational Safety and Health
NO	nitric oxide
NO ₂	nitrogen dioxide
NO _x	nitrogen oxides
O ₂	molecular oxygen
O ₃	ozone
OSHA	Occupational Safety and Health Administration
%	percent
Purpald	4-amino-3-hydrazino-5-mercapto-1,2,4-triazole
PAH	polycyclic aromatic hydrocarbons
Pb	lead
PM	particulate matter
ppm	parts per million
RH	relative humidity
rpm	revolutions per minute
S	sulfur
Si	silicon
SO ₂	sulfur dioxide
SO ₃	sulfur trioxide
SO ₄ ²⁻	sulfate
STEL	short-term exposure limit

THC	total hydrocarbons
Ti	titanium
TIA	total intensity of aroma
TLV	threshold limit value
TWA	time weighted average (concentration)
UV	ultraviolet
vol %	volume percent
wk	week
wt %	weight percent
Zn	zinc

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