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HEALTH AND SAFETY IMPLICATIONS OF DIESEL LOCOMOTIVE EMISSIONS.(U)
APR 78 M S SANDERS, J M PEAY

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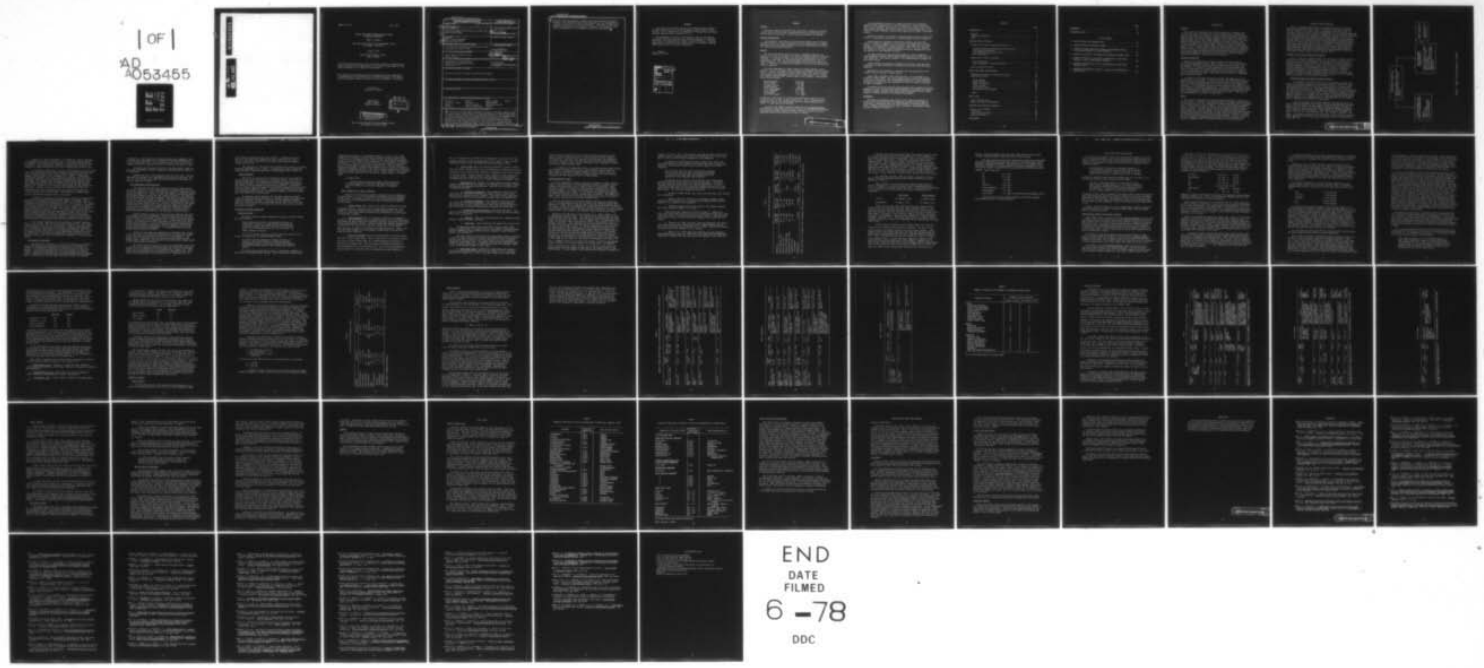

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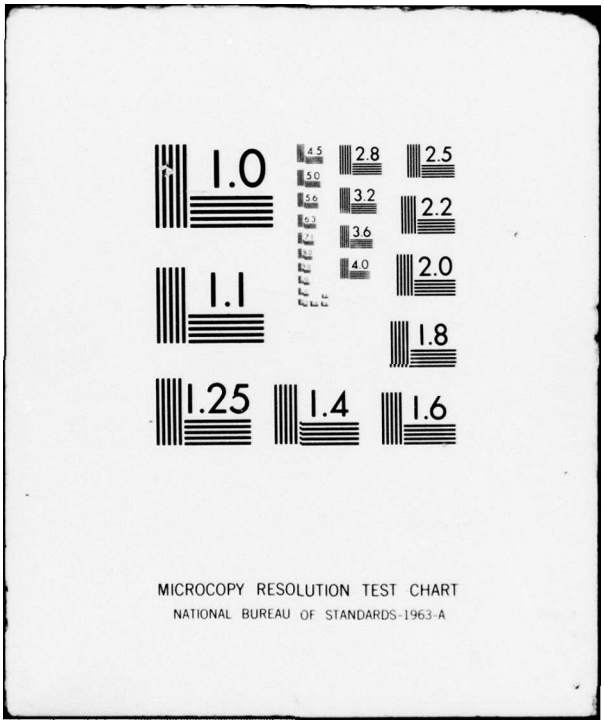
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NPRDC TR 78-17

April 1978

HEALTH AND SAFETY IMPLICATIONS OF DIESEL LOCOMOTIVE EMISSIONS

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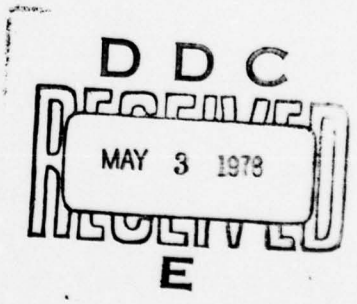

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San Diego, California 92152

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SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: NPRDC-TR-78-17
2. GOVT ACCESSION NO.:
3. RECIPIENT'S CATALOG NUMBER:

4. TITLE (and Subtitle): HEALTH AND SAFETY IMPLICATIONS OF DIESEL LOCOMOTIVE EMISSIONS
5. TYPE OF REPORT & PERIOD COVERED: Final Report
6. PERFORMING ORG. REPORT NUMBER:

7. AUTHOR(s): Mark S. Sanders (NPRDC)
James M. Peay (NWSC)
8. CONTRACT OR GRANT NUMBER(s):

9. PERFORMING ORGANIZATION NAME AND ADDRESS:
Navy Personnel Research and Development Center
San Diego, California 92152
10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: N0016477WR30092

11. CONTROLLING OFFICE NAME AND ADDRESS:
Naval Weapons Support Center
Crane, Indiana
12. REPORT DATE: April 1978
13. NUMBER OF PAGES: 62

14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office):
Department of Transportation
Federal Railroad Administration
Washington, D. C.
15. SECURITY CLASS. (of this report): UNCLASSIFIED
15a. DECLASSIFICATION/DOWNGRADING SCHEDULE:

16. DISTRIBUTION STATEMENT (of this Report):

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report):

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Pollution	Health	Sulfur Oxides	Safety
Locomotive engine	Railroad cars	Hydrocarbons	
Diesel	Carbon Monoxide	Particulate Matter	
Emissions	Oxides of Nitrogen	Caboose	

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

A review of the published literature was made to determine whether there are health and/or safety effects of long-term exposure to low concentrations of diesel emissions within the ranges reported in actual railroad operations. No consistent evidence was found linking low concentrations of diesel emissions to long-term health effects or short-term respiratory function. Evidence was found linking emissions to eye irritation. Interviews with union officials and operating crews, letters from union members, union file

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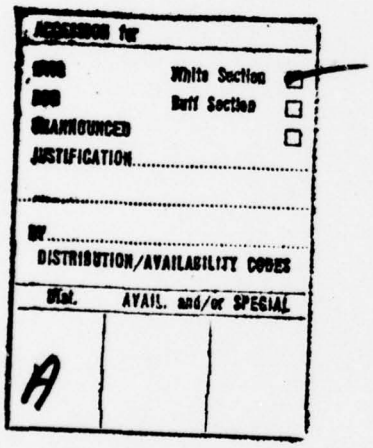
material, and miscellaneous locomotive and caboose inspection reports pointed to the conclusion that diesel emissions are not a widespread or frequent problem in the railroad environment. There may be short-term, infrequent occurrences of burning eyes, headache, and nausea, but any safety consequences of such symptoms could not be determined.

FOREWORD

This effort was performed jointly by the Navy Personnel Research and Development Center and the Naval Weapons Support Center, Crane, Indiana. It was sponsored by the Department of Transportation, Federal Railroad Administration, Washington, D. C.

Acknowledgement is made of the cooperation and time afforded by both union officials and members and railroad companies in supplying information and permitting the conduct of field interviews. Further, appreciation is extended to Mr. Robert Clarke of the Federal Railroad Administration for his technical assistance.

J. J. CLARKIN
Commanding Officer



SUMMARY

Problem

Ever since diesel locomotives were introduced to American railroads in the 1940s, there has been a continuing interest in measuring the resultant levels of pollution to which railroad personnel are exposed.

Purpose and Approach

The purpose of this effort was to determine whether there are health and safety effects of long-term exposure to low concentrations of diesel emissions within the ranges reported in railroad operations by reviewing the available published literature.

Results

The health and safety effects of diesel locomotive emissions are mediated by a host of variables that can be classified under the following headings: (1) locomotive engine and operating characteristics, (2) environmental conditions, (3) crew compartment characteristics, and (4) human parameters. The relationship between levels of diesel exhaust and types of locomotives is complex and depends on the duty cycle and specific emission compound.

The major components of diesel exhaust are carbon dioxide, carbon monoxide, oxides of nitrogen, sulfur oxides, hydrocarbons, and particulate matter. From the published literature specifying actual levels of pollution during tunnel operations, worst-case (5 tunnel runs per day) 8-hour time weighted average (TWA) exposure levels were computed. The computed values are as follows:

Carbon Monoxide	35.10 ppm
Nitric Oxide	23.10 ppm
Nitrogen Dioxide	.87 ppm
Sulfur Dioxide	1.00 ppm
Total Hydrocarbons	13.90 ppm
Total Aldehydes	15.7 ppm
Acetaldehyde	2.5 ppm
Particulate Matter	1.7 mg/m^3

In addition, peak levels of acrolein and formaldehyde reported in the literature were .04 and .20 ppm, respectively. All of these values are below limits set by the Occupational Safety and Health Administration (OSHA) for long-term 8-hour exposure.

A review of the Federal Railroad Administration Accident Bulletin published from years 1968 through 1973 revealed no serious train accidents attributable to diesel fumes or exhaust, and only 28 cases per year of operating crewmen being overcome by fumes or exhaust.

Four epidemiologic studies on railroad personnel all revealed no unusual health consequences of long-term exposure to diesel emissions. Short-term, infrequent incidences of eye irritation, however, were noted. Some evidence surfaced that certain people may be more susceptible to the effects of diesel emissions, specifically those over 45 years of age and/or smokers.

Laboratory studies of reactions to diesel exhaust showed no short-term effects on respiratory function but a high potential for eye irritation.

Surprisingly few laboratory studies have assessed the long-term effects on humans of exposure to low levels of the components of diesel exhaust. Carbon monoxide at worst-case 8-hour TWA levels has been found detrimental to people with coronary disease. Other than that, however, the results of low level carbon monoxide exposure are contradictory with the majority of tests showing no effect.

Nitrogen dioxide has been shown with animals to cause changes in their pulmonary system at levels around those expected in worst-case railroad operations. The results, however, can be generalized to humans only with great caution.

Sulfur dioxide concentrations expected in worst-case railroad operations have not shown impairment of respiratory functions from short-term exposures.

Hydrocarbons and aldehydes at the worst-case concentrations are believed to pose no health or safety threat.

Diesel odor, although a short-term irritant, is not believed to be a serious health hazard. The safety implications of nausea and headache could not be ascertained from the published literature. There have been virtually no studies that have assessed the effect of nausea or headache on human performance.

Interviews with union officials and operating crews, letters from union members and union file materials, and miscellaneous locomotive and caboose inspection reports indicate that diesel exhaust emissions are not a widespread or frequent problem in the railroad environment.

Conclusion

The overall conclusion of the study is that, based on computed expected worst-case 8-hour TWA diesel emission exposure levels, there is no unusual or widespread health or safety hazard to railroad operating crews. However, there may be short-term, infrequent occurrences of burning eyes, headache, and nausea.

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INTRODUCTION

Problem

Ever since the introduction of diesel locomotives to American railroads in the 1940s, there has been a continuing interest in measuring the resultant levels of pollution to which railroad personnel are exposed (Berger & McGuire, 1946; Railroad Commission of the State of California, 1946; Grewels, 1954; Clark, 1958a; Thompson, 1972, 1973; Apol, 1973; Scott Research Laboratories, 1972, 1974; Belgea, 1974; Hobbs, Walter, Hard, & Devoe, 1976; Battigelli, 1963a, 1963b, 1965). Also, this interest has not been confined to the United States (Prokhorov, Novikova, Pervukhina, & Ryzhoua, 1973). Almost without exception, these studies, often conducted under the worst of conditions, showed pollution levels below the criteria set by such regulatory agencies as the Occupational Safety and Health Administration (OSHA) and the National Institute of Occupational Safety and Health (NIOSH) for long-term, 8-hour exposures.

Purpose and Approach

Despite the accumulation of objective data, both unions and the Federal Railroad Administration (FRA) continue to receive a small number of sporadic complaints concerning diesel emissions in locomotive cabs and cabooses. In response to these complaints, the FRA initiated this study to investigate the possible health and safety effects of exposure to low concentrations of diesel emissions that are within the ranges reported in railroad operations. This effort took the form of a comprehensive literature review.

In addition, an attempt was made to evaluate the magnitude of the problem as it exists today. To accomplish this, interviews were held with union officials and operating crews in several areas of the United States. This included the northwest part of the United States where tunnels are prevalent. The information gleaned from the interviews was interwoven into the literature review where appropriate. In addition to interviews, several data sources were made available by the unions (United Transportation Union and the Brotherhood of Locomotive Engineers) and the FRA. These are discussed separately in Section 5 of this report.

Scope

This report is divided into 5 main sections in addition to this introductory section. Section 2, which deals briefly with diesel emission studies, was included to define the constituents of diesel emissions and to establish their representative levels in order to focus on their health and safety effects. Section 3 reviews the literature on health and safety effects of diesel emissions. Section 4 deals with diesel emission odors and their implications for health and safety. Section 5 deals with the interview data and data supplied by the unions and the FRA, which were analyzed to assess the relative magnitude of the diesel emission problem. Section 6, the last section, contains recommendations concerning the role of crews, unions, railroad management, and the government control of diesel pollution.

RAILROAD DIESEL EMISSIONS

There is a considerable quantity of literature published on diesel engines and emissions. Much of this literature, fortunately, falls outside the scope of the present study. For example, this review does not include a discussion of the various methodologies for detecting and measuring the constituents of diesel emissions, methods for control of diesel emissions (for a good discussion of control, see Patterson & Henein, 1972), or the precise engineering relationships between engine parameters and emissions. A great quantity of literature exists on the emissions of gasoline engines. This data is irrelevant, however, due to fundamental differences between diesel and gasoline engines. By their very nature as a compression ignition rather than spark ignition device and their operation at relatively high compression ratios and fuel lean mixtures, diesel engines generate exhaust products significantly different from those associated with gasoline engines (Moran, Taylor, Stoub, & Wheeler, 1977).

A Model of the Railroad Emission Situation

Figure 1 presents a simplified model of the railroad emission situation. Each box in the model represents factors that influence the health and safety effects of diesel emissions. The purpose of this study was not to analyze in detail the effects of all of the factors outlined in Figure 1. A few illustrative references, however, will be cited to illustrate the extreme complexity of the problem of trying to specify the makeup of typical diesel exhaust and its health and safety effects. To quote Battigelli (1963b), "A frank statement should say that the question concerning the characterization of typical diesel polluted air has no answer. There is probably no typical diesel pollution."

Locomotive Engine and Operating Characteristics

The quantity of emissions will vary as a function of engine type. There are five basic categories of locomotives (Kircher, 1975): (1) two-stroke supercharged switcher, (2) four-stroke switcher, (3) two-stroke supercharged road service, (4) two-stroke turbocharged road service, and (5) four-stroke road service. When measuring emissions in terms of kilograms per 1000 liters of fuel, Kircher (1975) found differences between the five types of locomotives. The differences, however, were not consistent across emission products. For example, the two-stroke supercharged switch engine had the highest level of emissions for hydrocarbons (HC), but the lowest level of emissions for oxides of nitrogen (NO_x).

Hare and Springer (1972) report emissions (in grams per hour) for three locomotives--an EMD 12-567 switch engine, an EMD 16-645E-3 line-haul engine, and a GE 7FDL16 line-haul engine. Using a standard line-haul or switch yard duty cycle, they found large differences between the engines on all emission products measured. In all cases, the GE 7FDL16 gave off higher levels of HC, carbon monoxide (CO), NO_x, aliphatic aldehydes, and smoke. In addition to quantitative differences, the GE locomotive produced three light hydrocarbons (C_2H_6, C_2H_2, and C_3H_6) that were not detected in either EMD unit.

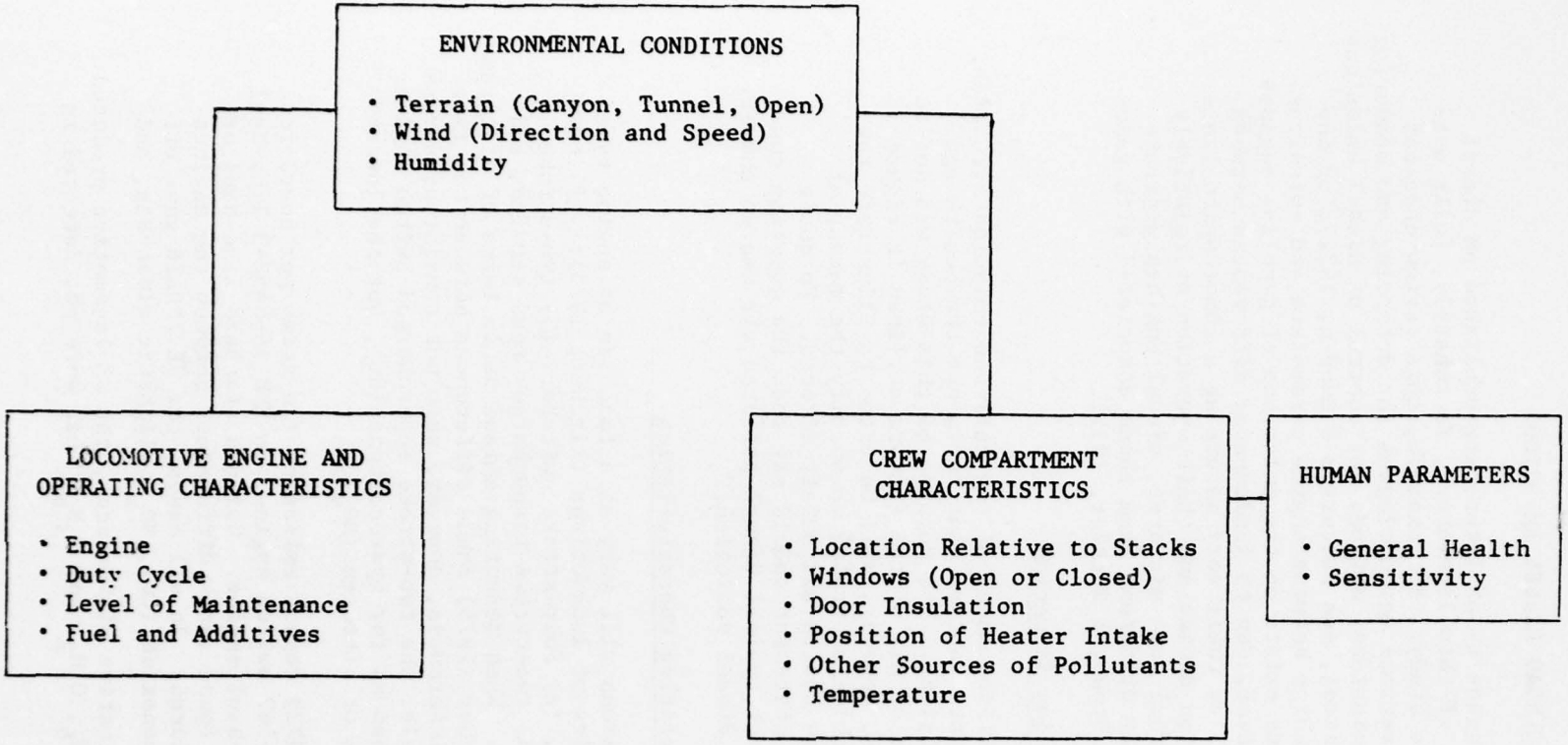

Figure 1. Model of railroad emission situation.

Moran et al. (1977) and Hobbs et al. (1976) also report differences in emissions as a function of engine type. In contrast, however, Rounds and Pearsall (1956) found little variation in odor between different makes of engines. Odor determinations, however, are subjective and may be insensitive to small variations in the level of exhaust above a given value.

In addition to engine type, the duty cycle (amount of time and transitions from one speed-load condition to another) also affects the level and type of emissions (Moran et al., 1977; Rounds & Pearsall, 1956; Hare & Springer, 1972; Kircher, 1975; and Hobbs et al., 1976). To complicate the situation further, the relative emission levels given off at different speed-load conditions are not consistent across emission products or engine types. For example, Hare and Springer (1972) report that, at idle, the GE 7FDL16 engine gives off 1.6 times more CO than the EMD 16-645E-3 engine, but, in notch 8, the GE engine actually gives off less CO than the EMD engine. Further, with NO_x, at idle, both the EMD and GE give off almost identical levels of emission, but at notch 8, the GE gives off 1.3 times as much NO_x as does the EMD. This indicates a complex engine type by load-speed by emission product interaction.

In addition to engine type and duty cycle, the level of maintenance has an effect on emission levels (Hedlund, Ekberg, & Mortstedt, 1967; Stokinger, 1975; Patterson & Henein, 1972; Hobbs et al., 1976). The exact relationship between level of maintenance (i.e., the condition of the engine) and the type and amount of emissions is not clear. Generally, an engine in good condition will give off less smoke than an engine in poor condition (Southwest Research Institute, 1971). In contrast, Rounds and Pearsall (1956) found engine condition had comparatively little effect on odor levels. Pattle, Stretch, Bukgess, Sinclair, and Edington (1957) found greater toxicity of emissions from an engine (single cycle) with a good injector than from the same engine with a worn injector. Apparently, the worn injector resulted in less NO_x emission than obtained with the good injector.

Fuel and fuel additives are also a moderator of diesel emissions (Moran et al., 1977). There are three grades of diesel fuel. From least to most volatile, they are 1-D, 2-D, and 4-D. There are two basic classes of fuel additives--cetane-type octane improvers and smoke suppressants. Here, as elsewhere, there appear to be complex interactions. Hare (1975), for example, found the effect of a smoke suppressant depended on the type of engine used. It decreased the percentage of organic solubles in the particulate matter for a two-cycle engine but increased it in a four-cycle engine.

Environmental Conditions

Referring back to Figure 1, it can be seen that once the emission escapes the engine, usually through the stacks, it is influenced by a variety of environmental conditions that principally act to dilute the emission. The terrain over which the train is traveling is a major influence. Tunnels, particularly, and, to a lesser extent, steep narrow canyons, increase the concentration of emissions reaching the crew compartments. Characteristics of the tunnel will also influence the emission

concentration. Such things as the length, grade, bore, alignment, type of lining, and ventilation must be considered (Railroad Commission of the State of California, 1946). Even the direction the train is traveling through a tunnel will influence the time it takes for the smoke to clear.

Wind direction and speed can direct emissions toward or away from the crew's compartment, thereby increasing or decreasing their exposure to pollutants.

Humidity may act to hold emissions down over the train. This was mentioned by one member of an operating crew during the interviews. He said, "if the wind were just right and the air was heavy, you might get a little smoke in the cabin."

Crew Compartment Characteristics

The interviews revealed that locomotives that are operated with the stacks forward of the crew compartment tend to produce more pollution in the crew compartment. This is in contrast, however, to objective results reported by Hobbs et al. (1976), in which they found no significant difference between longhood forward and cab forward locomotives. All longhood forward locomotives, when driven forward, result in the stacks being ahead of the crew. This, of course, is obvious. Less obvious, however, is the fact that forward cab locomotives, when run in the reverse direction, also present the stacks ahead of the crew. Further, crews riding in the second or third unit of a multiple-unit consist are also behind the stacks and, hence, are more susceptible to exhaust emissions. Emissions from the locomotive in the caboose depend on the length of the train as well as other environmental conditions and crew compartment characteristics.

Whether windows are open or closed obviously influences the level of pollutants in the crew compartment. Exposure level and time is drastically reduced in the caboose if windows are closed while the locomotive is in the tunnel and opened immediately after it leaves the tunnel (Clark, 1958a). The condition of the door insulation also is a factor in determining pollution levels. A few of the people interviewed indicated that, on the older locomotives and cabooses, the door insulation is worn and the air can blow into the compartment. It is possible that the air may contain diesel emission pollutants.

During the interviews, one operating crew mentioned that, on some of the older units of one particular manufacturer, the heater air intake in the locomotive was positioned at the rear of the locomotive. Crew members felt that emissions from the stack were often sucked into the heater intake. Apparently, this problem has been corrected on newer models of this manufacturer's locomotives.

The actual level of pollutants in the crew compartment is not only a function of the locomotive diesel emissions, but also of the types and amounts of other pollutants in the crew compartment. Hobbs et al. (1976) lists several potential sources of pollutants in locomotives (e.g., oil leaks, toilets, batteries, and electricals) and in cabooses (e.g., oil

fired heaters, refrigerated cars, and toilets). In addition, one can add oil in the brake lines (brakes are vented inside the cab, and, if oil is in the line, it will give off an odor), and cigarette smoking, a major source of CO.

The temperature in the cab can also influence the toxicity of various substances (Keplinger et al., 1959). It appears that toxicity rises both below and above an optimum temperature of approximately 26° C (78.8° F).

Human Parameters

The effect of a given level of pollution will not be the same for all individuals (with the exception of megalethal doses). There are individual differences in the response to pollutants as there are with any other human response. The general health of the individual will moderate the effects of the pollutants to which he/she is exposed. In addition, there is evidence that some people are particularly susceptible to the effect of diesel exhaust (Randolph, 1954). Susceptible persons may develop rhinitis, coughing, headache, nausea, and abdominal cramps, as well as "drunkenness sans alcohol" and disorientation.

It should be readily apparent from the model depicted in Figure 1 that the railroad emission situation is a complex problem with many intervening and moderating variables. Although all the studies of emission levels in locomotive cabs and cabooses show overall low levels, it is possible that, on occasion, all the conditions will be right, and a crew will be severely "gassed."

Composition of Diesel Emissions

State-of-the-Art

Battigelli (1963a) probably summarized the nature of diesel exhaust best when he wrote:

> Diesel exhaust may be more scientifically described as a mixture at the source of approximately 98 percent air containing a higher than normal fraction of CO and water (H_2O), and an inconspicuous portion of an extremely com-
>
> complex combustion mixture.

It is this "complex combustion mixture" which has been the focus of most investigations. To quote Stokinger (1975):

> The almost unending complexity of diesel emissions in respect of (sic) their chemical consituents has been increasingly revealed as even more sensitive and specific procedures have been used for their analysis and identification.

The myriad factors depicted in Figure 1 undoubtedly contribute to the problem of analyzing diesel exhaust. There is, however, another major

problem--unreliability of the analysis procedures. Pattle et al. (1957) comment on the variation of their results within conditions between days. In addition, an illuminating study carried out by the Coordinating Research Council of New York (1975) further highlights the unreliability of emission measurement. A multicylinder diesel engine and two samples of bottled gas were circulated among 15 participating laboratories. Each lab measured exhaust hydrocarbons by methods that complied with the Society of Automotive Engineers recommended practices. The results showed "substantial" differences among labs on both the bottled samples and the engine exhaust. The standard deviations between labs were 10 to 22 percent, respectively, of the grand means.

In summary then:

> . . . despite analytic advances, chemical characterization of diesel emissions is still far from complete, either as to type or amount of the various compounds. (Stokinger, 1975)

Major Components of Diesel Emissions

Despite the aforementioned problems associated with the analysis of diesel exhaust, it is still possible to delineate the major components of the exhaust. Actually, the major source of controversy and continued discovery is in the determination of the types of hydrocarbon compounds in the exhaust.

1. Carbon Dioxide (CO_2) results from complete burning of fuel and is heavier than air. Its major effect on the body is stimulation of the respiratory centers resulting in an increased breathing rate. As long as there is sufficient oxygen available in the environment, CO_2 will cause no significant problems.

2. Carbon Monoxide (CO) is a colorless, tasteless, and odorless gas that is not produced if fuel is completely burned. It is the most widely distributed and most commonly occurring air pollutant (National Air Pollution Control Administration, 1970b). It has also been the focus of the majority of research in the area of air pollution. Unlike CO_2, CO more readily takes the place of oxygen on the blood's hemoglobin molecules. Hence, even with sufficient oxygen in the environment, a high level of CO can still cause an hypoxic condition.

3. Oxides of Nitrogen (NO_x) are primarily made up of nitric oxide (NO) and nitrogen dioxide (NO_2). Nitric oxide (NO) is a colorless, odorless gas, while nitrogen dioxide (NO_2) is a reddish-orange-brown gas with a characteristic pungent odor. NO_2 is corrosive and highly oxidizing and may be physiologically irritating and toxic. Most of the NO_x produced by diesel engines is, fortunately, NO with comparatively small amounts of

NO_2. NO is subsequently oxidized in the atmosphere to the more toxic NO_2. Normally, however, at low NO concentrations of 1.2 mg/m^3 (1 ppm) or less, oxidation proceeds slowly (Environmental Protection Agency, 1971).

4. Sulfur oxides (SO_x) are composed principally of sulfur dioxide (SO_2) and sulfur trioxide (SO_3), with SO_2 dominating in combustion pollution. The amount of SO_x emitted during combusion is dependent on the sulfur content of the fuel burned. Sulfur content of fuels, however has been reduced due to federal and air standards so that there should be no toxicologically detrimental amounts of SO_x given off in diesel combustion (Stokinger, 1975).

5. Hydrocarbons (HC) represent the most complex portion of diesel exhaust. Hydrocarbons can be thought of as composed of two classes, simple hydrocarbons and oxygenated hydrocarbons. There are five principle types of simple hydrocarbons:

a. Paraffins (or Alkanes). Each compound in this group ends with "ane," and the prefix indicates the number of carbon atoms in the molecule (e.g., methane, octane, propane). This class is also classified as the heavy fraction and is associated primarily with raw unburned fuel.

b. Olefins (or Alkenes). Each compound in this group has the ending "ene" or "diene" depending on the number of double bonds it has. This class is also classified as the light fraction associated with partially burned ("cracked") hydrocarbons not present in the raw fuel.

c. Naphthenes or Cycloparaffins (also called Cyclanes). The name of compounds in this family are preceded by "cyclo" and have an ending "ane" (e.g., cyclohexane).

d. Aromatics. These are benzene derivatives. Examples include benzene, toluene, styrene, and xylene.

e. Acteylenes. These compounds have names with a suffix "yne."

The oxygenated hydrocarbons identified in diesel exhaust (Nala, Padrta, & Samson, 1968) fall generally into five classes: aldehydes (which include formaldehyde and acrolein), ketones, carboxylic acids, alcohols, phenols, and nitrophenols.

The actual number of hydrocarbons and oxygenated compounds contained in diesel exhaust is uncertain. Goretti and Liberti (1972), for example, reported over 400 compounds. Others have isolated over 1000 compounds (O'Donnell & Dravnieks, 1970). It appears that the major classes of hydrocarbons in diesel exhaust are the aromatics, phenols, and aldehydes (Moran et al., 1977; Karasek, Smythe, & Laub, 1974).

6. Particulate matter is defined as those solids and aerosols that exist after dilution with clean air and cooling to 90° F, excluding unbound water (Moran et al., 1977). The most common type of smoke from

diesel engines is dark black or hot smoke consisting of unburned carbon particles, almost always less than .5 micron in size (Stewart, Morgan, & Dainty, 1975). Toxic materials, most often hydrocarbons, are absorbed by the carbon soot particles. The particles then carry the toxic materials to the more sensitive tissues of the respitatory tract (National Air Pollution Control Administration, 1969).

In addition to black smoke, there is also blue and white smoke (Southwest Research Institute, 1971). Blue smoke contains unburned engine oil that reaches the combustion chamber because of worn pistons, rings, etc. White smoke is made up of droplets of unburned liquid fuel and is usually associated with the start-up or idle of some engines.

Railroad Emission Levels

The major components of diesel emissions with health or safety implications, as outlined above, are carbon monoxide, oxides of nitrogen, sulfur oxides, particulate matter, and a complex array of hydrocarbons. There have been a series of studies that have attempted to measure these diesel emission products. The latest, and probably most comprehensive, analysis was done by Hobbs et al. (1976). In the report, the authors also summarize the previously reported railroad emission level literature. The Hobbs et al. report, then, will serve as the basis for defining the levels of pollution that could be experienced by railroad operating crews.

There are two aspects of pollution exposure which must be considered; that is, the peak exposure level and the 8-hour time weighted average (TWA). Hobbs et al. (1976) list the maximum peak concentrations of emission products they found, as well as those reported in the previous literature. In all cases, the values were obtained under the worst possible conditions. All were measured during tunnel trips, usually in the longest tunnels in the United States.

Several of the peak levels cited by Hobbs et al. (1976), however, are of questionable practical value. The peak levels for hydrocarbons and alde- of questionable practical value. The peak levels for hydrocarbons and aldehydes, for instance, came from a study by Belgea (1974). The particular peak values (50 ppm and 75 ppm, respectively), however, were obtained under improbable conditions. These pollution levels were measured in the caboose on the fourth trip through a tunnel that had not been purged of pollution from the three previous trips through it. The next highest pollution levels for hydrocarbons (34 ppm) and aldehydes (25 ppm) reported by Belgea (1974) were obtained under more probable circumstances; that is, in the caboose through a tunnel that had not been purged from one, rather than three previous trips. For this reason, the 34 ppm and 25 ppm values will be used in worst-case calculations to be discussed shortly. Further, the peak value cited by Hobbs et al. (1976) for NO_2 (6 ppm from Thompson, 1973) may be an error. Thompson (1973), in the text of his report, cites the 6 ppm maximum figure for NO_2 but, in his data summary in the same report, the maximum value is listed at only 5 ppm. Further, this 5 ppm (or 6 ppm) figure was obtained with the train stopped for one hour in the longest tunnel in the United States. The next highest value obtained was 4.0 ppm on a standard run

through the tunnel. Due to the confusion regarding the peak value and the somewhat unusual conditions under which it was obtained, the 4.0 ppm value will be used in the worst-case calculations to be discussed.

In addition to specifying peak values, Hobbs et al. (1976) also calculated 8-hour TWA values based on their own tunnel and nontunnel data. From all of the data they collected and reviewed, Hobbs et al. (1976) concluded:

> The worst-case data from this study and the published work of others indicate that the breathing environment of railroad operating crews is acceptable within the guidelines of the published OSHA standards.

The purpose of the current review, however, was to investigate possible health and safety effects of long-term exposure to low levels of diesel pollution characteristic of railroad operations. In order to establish characteristic levels, worst-case assumptions were used to calculate 8-hour TWA levels for those pollution products for which necessary data existed. The following assumptions were made:

1. During an 8-hour period, five tunnel runs were made, each lasting 20 to 30 minutes.

2. Exposure levels in tunnels were the highest probable values reported in the literature (i.e., Belgea's second highest values) whether measured in the caboose or the locomotive.

3. Nontunnel exposure levels were based on the highest nontunnel TWA values reported by Hobbs et al. (1976).

These worst-case values are presented in Table 1. Hobbs et al. (1976) also calculated 8-hour TWA values for locomotives running five tunnel trips per 8 hours. These values are also shown in Table 1. The worst-case value shown in Table 1 differs from those of Hobbs et al. (1976) for two reasons.

1. Hobbs et al. (1976) used only their peak exposures from locomotive, while our worst-case calculations used the peak exposure reported by previous literature as well as Hobbs et al. (1976), regardless of whether they were found in the locomotive or the caboose.

2. Hobbs et al. (1976) based their TWA value on only locomotive peak nontunnel data, while our worst-case calculations used peak TWA values regardless of whether they were recorded in the caboose or the locomotive.

Table 1

8-Hour TWA Railroad Exposure Data

Substance	Worst-Case 8-Hour TWA	Hobbs et al. 8-Hour TWA	Thompson 8-Hour TWA	OSHA 8-Hour TWA Limit	ACGIH 8-Hour TWA Limit
Carbon Monoxide (CO)	35.1 ppm	35.1 ppm	9.4	50.0 ppm	50.0 ppm
Nitric Oxide (NO)	23.1 ppm	22.0 ppm	—	25.0 ppm	25.0 ppm
Nitrogen Dioxide (NO_2)	.87 ppm	.03 ppm	0 ppm	5.0 ppm	5.0 ppm
Hydrocarbons Total (HC)	10.7 ppm	4.94 ppm	--	No Standard	No Standard
Aldehydes--Total	5.3 ppm	.09 ppm	—	No Standard	No Standard
Acetaldehyde	--	--	2.5 ppm	--	100.0 ppm
Particulate	.71 mg/m^3	.07 mg/m^3	2.5 mg/m^3*	15 mg/m^3	10 mg/m^3

*This value may be too high; a more reasonable value would be 1.7 mg/m^3. See text for discussion.

Thompson (1973) also calculated 8-hour TWA railroad exposure values for several substances based on the assumption of a 1-hour exposure to the maximum concentration he had found and 7 hours of low level exposure. Table 1 contains these values. Thompson's particulate 8-hour TWA is unusually high. This is due to his use of .95 mg/m^3 as the 7-hour low-level exposure value. Thompson, however, does not say how he arrived at that value. From actual measurements, Hobbs et al. (1976) computed the maximum nontunnel TWA to be only .16 mg/m^3. Recalculating Thompson's 8-hour TWA using the .16 mg/m^3 figure instead of .95 mg/m^3 yields an 8-hour TWA value of 1.7 mg/m^3. Without justification for the .95 mg/m^3 value, we are forced to accept the lower 1.7 mg/m^3 8-hour TWA for particulate matter.

For comparison purposes, OSHA and American Conference of Governmental Industrial Hygienists (ACGIH, 1975) 8-hour TWA limits are also presented in Table 1.

Other studies in the literature have reported peak levels for specific aldehydes, but, without nontunnel railroad background levels, it is not possible to compute 8-hour TWA values. These peak levels reported in the literature and the ACGIH 8-hour TWA limit are given below for comparison:

	Peak (ppm)	ACGIH Standard
Acrolein	.04 (Belgea, 1974)	.1 (ACGIH, 1975)
Formaldehyde	.20 (Apol, 1973)	2.0 (ACGIH, 1975)

Again it can be seen that these worst-case short duration peak exposures are well below the acceptable 8-hour exposure levels. If the necessary data were available and 8-hour TWA levels were calculated for these hydrocarbons, the 8-hour TWA would be even lower than the peak levels shown above. In support of the literature showing relatively low levels of specific hydrocarbons in railroad diesel exhaust is a study by Hancock, Applegate, and Dodd (1970). They found higher levels of aromatic hydrocarbons on leaves in a control area than they did on leaves along a railroad right-of-way.

Sulfur oxides, principally sulfur dioxide (SO_2), have also been reported in the railroad tunnel exposure literature. The level of SO_2 is a function of the sulfur content of the fuel burned, however. Clark (1958a) reported the highest peak level of 8 ppm for 3 minutes using X-6 diesel fuel in tunnel operations, but only .8 ppm was reported using X-3 fuel. Other investigations have reported virtually no SO_2 at all (Apol, 1973; Thompson, 1972). A worst-case 8-hour TWA value, however, can be calculated from the data reported by Clark (1958a). Assuming five 22-minute tunnel trips per 8-hour day, using the highest tunnel concentrations with X-6 fuel (which produces 10 times more SO_2 than does X-3 fuel), and using the lowest tunnel concentration found by Clark (.18 ppm) as the

expected nontunnel exposure level, an 8-hour TWA yields a value of only 1.0 ppm. The ACGIH (1975) 8-hour TWA limit on SO_2 is 5 ppm.

The next section of this report reviews the literature on the health and safety implications from repeated exposure to pollution levels characteristic of railroad operations. This study will take the highest 8-hour TWA values from Table 1 as characteristic of the major components of railroad diesel exhaust, along with the SO_2 8-hour TWA calculations from Clark (1958a). These values are:

CO	35.1	ppm
NO	23.1	ppm
NO_2	.87	ppm
SO_2	1.0	ppm
Total HC	13.9	ppm
Total Aldehydes	15.7	ppm
Acetaldehyde	2.5	ppm
Particulate	1.7	mg/m^3 (recalculated from Thompson, 1973)

The next section of this report focuses on literature relating these exposure levels to health and safety.

HEALTH AND SAFETY IMPLICATIONS

The literature reviewed in this section deals with exposure to the low levels of diesel emissions or the specific products of diesel emission set forth in Section 2 of this report. Continuous exposure studies are not included, for, according to the National Institute for Occupational Safety and Health (1976):

> An extrapolation from data on continuous exposure to the intermittent exposure characteristics of the occupational setting cannot be correctly performed on the assumption that effective concentration x time is constant.

In addition, animal studies have been deemphasized, for, according to the National Air Pollution Control Administration (1969):

> The use of laboratory animals in toxicological experiments is more straightforward, but the obvious anatomical and metabolic differences between animals and man require the exercise of considerable caution in applying the results of animal exposures to human health criteria.

In support of this, Tyler, McLaughlin, and Canada (1967) reviewed comparative micro- and macroscopic anatomy of blood in humans and in many of the common experimental animal species. On the basis of their review, they concluded that the rat, the most common experimental species, was an inadequate model for the study of human emphysema. They felt the horse was the closest model to man.

Further, this review does not discuss epidemiologic studies of general population exposures to ambient pollutants except where railroad workers are the focus of the study.

Epidemiologic Studies of Railroad Personnel

Devoe (1976) reports a search of the FRA Accident Bulletin for the 6 years from 1968 to 1973. During that period, there were no train accidents attributed (primary cause) to fumes from internal combustion engines or appurtenances (Code 2113). To check on diesel emissions as a contributing cause of accidents, Devoe reviewed 90 detailed reports of serious train accidents. In only two of the reports did crew members claim the presence of fumes as a contributing cause. In both cases, however, the accident examiners ruled that the claims were invalid.

Incidences of operating personnel being overcome by fumes in train service accidents would be included in any of the three Accident Bulletin classifications (operating locomotive, operating rail motor car, and not elsewhere classified). Devoe (1976) reports a yearly average of 28 incidences in these classifications from 1968 through 1973 (range 15 to 48 per year).

In addition to the FRA Accident Bulletin, there have been four epidemiologic studies dealing with railroad personnel. Two studies dealt with locomotive repairmen (Apol, 1973; Battigelli, Mannella, & Hatch, 1964) and two studies focused on operating crews (Clark, 1958b; Kaplan, 1959).

Apol (1973) carried out a hazard evaluation of the diesel run and repair building and a medical evaluation of the employees of the Union Pacific Railroad Company, Pocatello, Idaho. Engines were brought into the service building (52 feet long by 60 feet wide by 35-40 feet high) and serviced. The engines were often left running during this time. At the time of the environmental evaluation, six line-haul engines and four switch engines were in the building. All the end doors and side windows were open, and 10 ceiling exhaust fans were operating. Environmental measurements made over a 20-hour period revealed the following range of values for the various components of diesel emissions:

Acrolein	< .015 ppm to < .04 ppm
Formaldehyde	.015 ppm to .07 ppm
NO_2	.01 ppm to .06 ppm
NO_x	.03 ppm to .26 ppm
SO_2	All less than .01 ppm
Particulates	.09 mg/m^3 to .26 mg/m^3
CO	1 ppm to 15 ppm

Compared to levels reported in the literature for nontunnel through-freight operations (Hobbs et al., 1976), these values are comparable but are lower than would be experienced in tunnel operations.

Apol's medical evaluations of 117 employees (out of a total population of 125) were based on results of a chest x-ray, forced expiratory spirogram, and a health questionnaire. The questionnaire data revealed 31 of the 114 males as having symptoms of bronchitis (27%). As pointed out by Apol, however, the prevalence of chronic bronchitis among industrial and nonindustrial populations ranges from 5 to 40 percent. The 27 percent figure found here, therefore, is not unusual.

Twelve (12) abnormal spirograms were identified. Based on a Los Angeles Breathmobile study of 10,000 industrial workers, Apol indicates that a 7.2 abnormal reading would be expected for this particular employee population. Applying a Chi Square test to the data revealed that the obtained number of abnormal spirograms is most likely a chance deviation from the expected level. The chest x-rays revealed no pneumoconiotic lesions. These results led Apol to conclude that, "excessive chronic respiratory disease probably does not exist among those surveyed in this plant."

Although no unusual acute health effects were found, private interviews with three employees revealed general comments concerning occasional burning eyes, headaches, and offensive odors. Apol states that the eyes of one employee interviewed were watering and red. In fact, for one 30-minute period (out of 20 hours of time sampling), Apol himself noted eye irritation, manifested by slight burning. Apol concluded that, "Eye irritation may occur for short periods of time . . . . This condition is not predictable and does not appear frequently."

It appears, therefore, that continued long-term exposure to low levels of diesel exhausts and fumes, which occasionally can cause eye irritation, do not seem to lead to abnormal occurrences of chronic respiratory diseases.

Battigelli et al., in 1964, reported a medical evaluation of 210 engine repairmen (mean time on job, 9.16 years) from three railroad shops situated in the area of Pittsburgh, Penssylvania. In addition to this group, another group of 153 railroad yard workers not exposed to diesel emissions was also sampled. Battigelli et al. (1964) did not give details about the nonexposed group's work environment. It seems unlikely that yard workers would not be exposed to diesel fumes and exhaust; however, their exposure would probably be less than that experienced by the locomotive repairmen.

The exposure conditions of the repairmen were obtained from 350 atmophere samples taken over a period of several months. The maximum concentrations found are listed below; 8-hour TWA values were not calculated.

NO_2	1.8 ppm maximum
Acrolein	.1 ppm maximum
Aldehydes	1.7 ppm maximum
SO_2	4.0 ppm maximum
HC	5.0 ppm maximum

All subjects were processed through a physical exam, chest x-ray, electrocardiogram, spirometry, medical history, and questionnaire. There were essentially no differences between exposed and "control" groups, with respect to complaints about dyspnea, cough, or sputum. Nor were there any differences uncovered during the physical examinations, electrocardiograms, or spirometry. Further, comparing those with short exposure history in the exposed group with those having longer exposure histories also revealed no systematic differences that could be attributed to exposure to diesel emissions. Battigelli et al. (1964) found, however, that smoking cigarettes did have a significant effect on both complaint rate and pulmonary function. Fortunately, the exposed and control groups were matched on smoking behavior (packs per day x years).

So, once again, as with Apol (1973), we find no adverse health effects associated with long-term exposure to diesel emissions.

Clark (1958b) conducted a comprehensive medical analysis of head end locomotive crews and caboose crews at the beginning and end of trips. The logic was that head end locomotive crews were in front of the exhaust while caboose crews were behind and thus exposed to more pollution. All the trips involved tunnels. Clark reports his data in terms of the proportion of man-trips (men x trips) that showed symptom changes from the beginning to the end of the trip. There are several problems with this type of data. First, the effects of long-term exposure to diesel exhaust is not being measured; rather, only short-term (trip length) exposure effects

are being measured. Second, a person can contribute several data points to the analysis equal to the number of trips he made. Each person, however, did not make the same number of trips (average 1.6 trips per person in the head end crews and 2.9 trips per person for the caboose crews). Therefore, each person is not contributing the same data, and the data points are not independent. This lack of independence precludes the use of inferential statistics, making it impossible to determine what the probability is that the differences obtained occurred by chance.

In many of the objective medical tests conducted (distance, acuity, depth perception, blowmeter test of nasal patency, maximum breathing capacity, pulse rate, blood pressure, respiratory pattern, color of nasal mucosa, nasal secretion), there was virtually no difference between the head end and caboose crews in terms of the percentage of man-trips for which there was an increase in symptoms from the beginning to the end of a trip. Only three measures (vital capacity, throat color, and lung rales) showed differences between head end and caboose crews. For vital capacity, 28 percent of the head end crew man-trips showed a decrease in inspiratory vital capacity from the beginning to the end. This is compared to 34 percent of the caboose man-trips showing a decrease. This difference was not apparent if only those under 45 years of age were considered, but it was more pronounced for those workers over 45 years of age (28% and 39%, respectively). Expiratory vital capacity, a second measure of vital capacity, also showed differences between head end and caboose crews for those over 45 years of age. For the head end crews, only 16 percent of the man-trips showed a decrease in expiratory vital capacity from the beginning to the end of the trip, compared to 28 percent for caboose crew man-trips. In like manner, timed vital capacity for those over 45 years old also showed differences of 18 versus 25 percent for head and caboose, respectively.

With respect to throat color, 39 percent of the head end crew man-trips revealed increased redness from the beginning to the end of the trip, compared to 46 percent of caboose crew man-trips. In the case of rales in the lungs, again Clark found that the difference between head end and caboose crews was most marked in those people over 45 years old. Twenty-two (22) percent of the head end crew man-trips showed increases in lung rales, whereas 30 percent of the caboose crew man-trips showed an increase.

All of these differences between head end and caboose crews on the three measures (vital capacity, throat color, and lung rales) are small and probably are not statistically significant. In fact, based on this data and his own observations, Clark concluded:

> There appears, however, to be no important amount of acute health damage and no definite difference between the experience of those riding in the head engine and the caboose engine of the freight trains from either the combustion products of diesel locomotives or from the dust arising out of the use of track sanders.

Recall, however, that this study is assessing short-term health effects of differing degress of exposure. One explanation for the lack of clear cut differences may be that the exposure levels of the head end crews are sufficient to cause physiological changes in the course of 1 day equal to those found for caboose crews. It may be, then, that the difference in exposure levels between head end and caboose is not large enough to produce differences in short-term physiological change measures.

Clark also solicited complaints and subjective opinions from the crew members at the beginning and end of each trip. The following are the percentage of man-trips in which there was an increase in the specific complaint for head end and caboose crews:

	Head End	Caboose
Discomfort to eyes	28%	57%
Discomfort to nose	24%	46%
Throat discomfort	9%	30%
Chest discomfort	8%	22%

In addition, on 4 percent of the head end crew man-trips and 16 percent of the caboose man-trips, the "feeling of well-being" declined from the beginning of the trip to the end. The subjective complaints show a clear and marked pattern of short-term adverse effects in the caboose crews. Clark points out, however, that there was no relationship between the objective physiological changes and the subjective complaints. This seems to indicate that complaints may not be an accurate indicator of actual health effects.

The last epidemiologic study done on railroad employees was carried out by Kaplan in 1959. Kaplan reviewed 6506 death reports of Baltimore and Ohio railroad employees and former employees from the years 1953 through 1958. Of those, Kaplan found 818 malignancies of organs. Of those 818, 154 were lung and/or bronchial tumors. It was this sample of 154 respiratory tumors that made up the sample data.

Three groups of employees were defined based on the degree of exposure to diesel fumes and exhaust likely to occur on their job.

1. High-exposure Group: Engineers, firemen, motormen, hostlers, road brakemen, yard helpers, yard foremen, conductors, switchmen, yardmasters, and trainmasters.

2. Medium-exposure Group: Manual labor in shop and roundhouses, carmen, machinists, crane operators, welders, and painters.

3. Low-exposure Group: Clerks, janitors, agents, and bridge inspectors.

It is interesting to compare this exposure classification to that used by Battigelli et al. (1964). Battigelli et al. considered locomotive repairmen (Kaplan's medium-exposure group) as the "exposed group" and yard workers (Kaplan's high-exposure group) as the "control."

Kaplan computed for each group their respiratory tumor death rate per 100,000 people and compared that to the expected death rate based on American Cancer Society data. In all cases, the death rate was actually lower than the expected rate, as shown below:

	Actual	Expected
High exposure	49	56
Medium exposure	67	93
Low exposure	38	42

As a percentage of expected death rate, the highest rate is the low-exposure group (89% of expected) followed by the high-exposure (87% of expected) and medium-exposure (72% of expected) groups. Kaplan notes that the high-exposure group made up 31.4 percent of the population and accounted for 31.8 percent of the lung cancer deaths. He concludes, "Our statistical studies fail to reveal any relationship between noxious fumes associated with railroad work and primary lung carcinoma."

In summary, then, all the epidemiologic studies done with railroad personnel, despite different methodologies and designs, have found essentially no widespread health hazards associated with diesel emissions. There seems to be evidence, however, that some types of people, especially smokers and those over 45 years old, may be more susceptible to the effects of pollution than others.

These epidemiologic studies, by their very nature, lack experimental control. Exact exposure dosages and histories are unknown, and other contaminating variables are not always eliminated. It is possible that in such uncontrolled realistic situations, subtle effects of low levels of diesel emissions may be overlooked. To explore such subtle effects, therefore, we now turn to controlled laboratory studies of exposure to low levels of diesel exhaust and the separate components of diesel exhaust. Unfortunately, however, all the laboratory studies to date have focused on short-duration exposures. There have not been any laboratory experiments that have systematically studied exposures to low levels of any pollutant over periods characteristic of occupational exposures (8 hours per day for 5 or 10 years). With long-term exposure to low levels of pollution, one may ask whether there is an acclimatization process or whether any detrimental effect becomes progressively more severe.

Laboratory Studies

Diesel Exhaust

One study (Battigelli, 1965) was found that investigated, in a controlled laboratory situation, the effects of diesel exhaust on human

subjects. Volunteers were exposed for short periods of time up to 1 hour in duration to one of three dilutions of diesel exhaust produced by a single-cylinder, 4-cycle, 7-horsepower diesel motor. The dilutions are shown in Table 2. A comparison of these dilutions to the worst-case 8-hour TWA figures for rail operations from Section 2 shows that dilutions B and C are very similar to the worst-case values for CO, NO_2, and acrolein, but are lower in total aldehydes and total hydrocarbons.

Pulmonary resistance was measured at 15-minute intervals throughout the exposure. For none of the dilutions, there were no significant differences in pulmonary resistance throughout the exposure. The largest percentage change occurred in the most dilute condition (A). Further, the point of maximum effect was not consistent between conditions. Dilution A showed its maximum effect after 60 minutes exposure, while dilution B peaked after 30 minutes and C after 45 minutes. The lack of statistically significant differences and irregular patterns of effect are due in part to the variability between subjects exposed to the same conditions. Mean pulmonary resistance ranged from -.234 to +.130, while the standard error of the means ranged from .08 to .65. The corresponding range of standard deviations would be .019 to 2.12. Battigelli concluded, "Inhalation of exhaust at each and every one of the three levels so far employed has not produced any change which could be considered significant in terms of pulmonary resistance." Further, Battigelli noted that none of the 13 volunteers complained during the breathing test.

To further test the effects of these three dilutions of diesel exhaust, Battigelli applied the gases directly to the eyes of the subjects for up to 10 minutes. All of the concentrations were sooner or later objectionable to the subjects. None, however, discontinued their exposure to dilution A, while over half did to dilution C. Battigelli had the subjects rate the subjective irritation of each dilution on a 4-point scale:

0 - No subjective irritation
1 - Some (threshold)
2 - Conspicious but tolerable
3 - Intolerable

The mean score (and peak score) for each concentration is as follows:

A - .86 (2)
B - 1.47 (3)
C - 2.65 (3)

It appears from this study that eye irritation may be the primary immediate response to diesel emission rather than gross pulmonary changes.

Table 2

Dilutions Used by Battigelli (1965)

Substance	Dilution A		Dilution B		Dilution C	
Nitrogen Dioxide	1.3	(.2-2.0)*	2.8	(2-4)	4.2	(1.5-7.0)
Sulfur Dioxide	.1	(.12-.40)	.5	(.2-1.0)	1.	(.5-2.8)
Carbon Monoxide	<20.	(<20)	30.	(20-50)	55.	(50-80)
Carbon Dioxide	1000.	(900-1500)	9000.	(8000-12000)	11000.	(9000-15000)
Oxygen	20.5	(20.5)	20.	(20)	19.5	(19.5)
Total Aldehydes	<1.0	(<1.0)	<1-2	(<1-2)	1-2	(1-2)
Acrolein	< .05	(<.05)	< .05	(<.05)	<.05	(<.05)
Formaldehyde	< .1	(<.0)	< .1	(<.1)	<.1	(<.1)
Hydrocarbons	<2.0	(<2.0)	2.5	(2-4)	3.2	(2-6)

*Mean (range), Pulmonary Resistance

Carbon Monoxide

There is extensive documentation of the fact that high concentrations of CO can cause many physiological and pathological changes and, ultimately, death. The effects of exposure to levels of CO less than 100 ppm are not so well documented, however (National Air Pollution Control Administration, 1970b).

The principle toxic properties of CO arise because the affinity of human hemoglobin is 210 times greater for CO than for oxygen (O_2). Thus, CO will take the place of O_2 in the blood leading ultimately to tissue anoxia. Hemoglobin saturated with CO is called carboxyhemoglobin (COHb). The critical variable in relating CO exposure to physiological and performance changes is the percent blood concentration of COHb. The percent COHb of the blood reaches an equilibrium at any given CO atmospheric concentration after 4 to 8 hours (Smith, 1968). A rough approximation of the percent COHb for exposures of less than 100 ppm CO for 4 or more hours is given in Goldsmith and Landau (1968) as:

$$\% \text{ COHb} = \text{CO ppm} \times .16$$

Assuming an 8-hour TWA exposure of 35 ppm for railroad operating crews (see Section 2) and applying this formula yields an estimated COHb of 5.6 percent. According to the National Air Pollution Control Administration (1970b), to get the equilibrium COHb percentage, .5 must be added to account for normal physiological "background" levels of COHb. The resultant value is then 6.1 percent COHb. This is almost the same as the median value of COHb (5.9%) found for moderate cigarette smokers who inhale between one half to two packs per day.

The review of physiological and performance effects of CO will focus on studies resulting in COHb levels up to 6 percent.

One finding relating to low-level exposure to CO that seems to be fairly widely accepted (Goldsmith & Aronow, 1975; National Air Pollution Control Administration, 1970b) is that CO is especially dangerous to people with coronary disease. In a review of the literature, Goldsmith and Aronow (1975) state that angina pectoris occurs following exposure to 50 ppm of CO after just 90 minutes with patients prone to angina attacks. Aggravation of intermittent claudication occurs after exposure to 50 ppm of CO for 120 minutes. Anginal patients also show depressed forcefulness of heart contractions after exposure to CO. Accordingly, employees with suspected or confirmed cardiovascular disease should avoid trips where CO levels are apt to be high (e.g., tunnel trips).

Human studies dealing with the performance effects of low levels of CO are summarized in Table 3. As can be seen, the results are often contradictory with the majority of tests showing no effect of CO below COHb levels of 6 percent. This is more readily apparent in Table 4, which lists the dependent variables tested across all studies listed in Table 3, and the number of tests that either showed or did not show an effect of CO at COHb levels below 6 percent. Most of the dependent variables have not been researched extensively, and, of the nine variables with more than

one test, only brightness threshold and vigilance show consistent detrimental effects of low COHb levels. In all other cases, the results are either contradictory or show no effect of low COHb levels. It is difficult, from the published literature, to make any firm conclusion regarding the railroad safety implications of exposures to low levels of CO. Horvath, Dahmos, and Hanlon (1971) felt that persons might become less effective in coping with unexpected events or might be liable to perform routine tasks in an inefficient manner. This may be true, but the evidence in the literature on this point is far from conclusive.

Table 3

Summary of Human Laboratory Studies on Performance Effects of Low-level Exposure to Carbon Monoxide

Study	CO Levels (ppm)	Exposure Time	% COHb	Dependent Variables	Findings
McFarland et al. (1944)	---	Tied to COHb	5 to 20	Threshold brightness for visual discrimination of a flash of light	Changes at COHb as low as as 5%. Light had to be more intense to be discriminated.
Schulte (1963)	100 ppm	Tied to COHb	up to 20	Reaction time, static steadiness.	No effect of up to 20% COHb.
				Choice discrimination tests of cognitive ability.	Increases in errors and completion time at COHb of 5%.
Beard & Werheim (1967)	0, 50, 100, 175, 250	4 hours	---	Time discrimination of tones 1 sec ± .375 in duration.	Decrement in percent correct discrimination after 90 min at 50 ppm.
Mikulka et al. (1969)	0, 50, 175	3 hours	.96, 2.98, 6.64	Time estimation, tracking performance, vestibular function.	No effect of CO exposure on performance.
Beard & Grandstaff (1970)	0, 50, 150, 250	1 to 2-1/2 hours	---	Estimation of 10 sec time intervals.	No effect of CO exposure.
				Estimation of 30 sec time intervals.	Significant impairment with 50 ppm and 64 minutes exposure.
				Relative brightness threshold, critical flicker fushion, visual acuity.	Significant impairment with COHb below 5%.
Hanks (1970)	0 to 100	4 hours	---	Tracking task, visual pursuit task.	No effect of CO.

Table 3 (Continued)

Study	CO Levels (ppm)	Exposure Time	% COHb	Dependent Variables	Findings
Stewart, Newton, Hosko, & Peterson (1970)	<1, 25, 50, 100	8 hours	up to 13	Time estimation, reaction time, steadiness, manual dexterity.	No effect of CO.
O'Donnell, Mikulka, Heinig, & Theodore (1971)	50, 125, 200, 250	3 hours	3 to 12	Time estimation, tracking.	No effect of CO.
O'Donnell, Chikos, & Theodore (1971)	75, 150	9 hours	5.9, 12.7	Time estimation, mental arithmetic, tracking.	No effect of CO.
Horvath, Dahmas, & Hanlon (1971)	0, 26, 111	2-1/4 hours	up to 6.6	Vigilance task, brightness discrimination.	No effect at 26 ppm, decrement at 111 ppm after 10 hours.
Baretta, Dodd, Fisher, Hermann, Hosko, Newton, Peterson, & Stewart (1972)	<1, 25, 50, 100, 200, 500, 1000	Tied to COHb	2 to 23	Visual evoked response, reaction time, manual coordination, time estimation.	No effect with COHb less than 12%.
Groll-Knapp, Wayne, Hauck, & Haider (1972)	50, 100 150	2 hours	3, 5.4, 7.6	Auditory vigilance.	More stimuli to be missed at all 3 CO levels.
Beard & Grandstaff (1970)	---	90 minutes	up to 6	Vigilance, velocity estimation.	Decrement at COHb of 2.5%.
				Problem solving, digit span, arithmetic computations, spatial perception.	No effect up to 6% COHb.
McFarland (1973)	0, 700	Tied to COHb	<4, 11 17	Simultaneous complex reaction time, dark adaptation, glare recovery, peripheral vision, depth perception, steering wheel reversals in actual driving task.	No effect with COHb less than 17%.

Table 3 (Continued)

Study	CO Levels (ppm)	Exposure Time	% COHb	Dependent Variables	Findings
Stewart, Peterson, Baretta, Bachand, Hosko, & Hermann (1973)	<2, 50, 100, 200, 500	up to 5 hours	up to 20	Time perception/estimation with 4 difference methods.	No effect of CO.
Putz, Johnson, & Setzer (1976)	5, 35, 70	4 hours	1, 3, 5	Tracking easy task, visual peripheral monitoring detection, auditory monitoring.	No effect of CO.
				Tracking difficult task, reaction time to peripheral stimuli.	Impairment at COHb below 5%.

Table 4

Summary of Studies on CO Exposure by Dependent Variable Used

Dependent Variable	Number of Tests Showing:	
	Effect of CO[a]	No Effect of CO[a]
VISUAL		
Brightness Threshold	3	0
Critical Flicker Fusion	1	0
Visual Evoked Response	0	1
Visual Acuity	1	0
Dark Adaptation	0	1
Glare Recovery	0	1
Peripheral Vision	0	2
Depth Perception	0	1
Visual Pursuit Task	0	1
MANUAL		
Steadiness	0	2
Vestibular Function	0	1
Manual Dexterity	0	2
Tracking Performance	1	6
COGNITIVE		
Reaction Time	1	4
Cognitive Abilities	1	4
Choice Discrimination	1	0
Spatial Perception	0	1
Auditory Monitoring	0	1
Velocity Estimation	1	0
Vigilance	3	0
Time Discrimination/Estimation	2	10

[a]At or below approximately 6 percent COHb.

Oxides of Nitrogen

Unfortunately, there are vitually no carefully controlled studies of workers exposed to low concentrations of oxides of nitrogen. Further, there is a deficiency of studies investigating the chronic effects in experimental animals of exposures to NO_x at schedules and concentrations representative of occupational environments (National Institute for Occupational Safety and Health, 1976). There also are few primary references in the literature as to the effects of NO_x upon the eyes or mucosae other than that in the lower respiratory tract (NIOSH, 1976).

The two principle review documents on the effects of NO_2 are those published by the National Institute of Occupational Safety and Health (1976) and the Environmental Protection Agency (1971). Unless otherwise stated, these documents served as the primary source for the discussion on NO_2 effects. NIOSH cites only four NO_2 experimental studies involving human subjects. Of the four, two employed concentrations above those considered to be worst-case 8-hour TWA values for operating railroad personnel (i.e., NO = 23 ppm, NO_2 = .87 ppm). The two studies using more reasonable values investigated the effects of NO_2 on chronic bronchitis patients and healthy males (Von Niedling & Krekeler, 1971; Von Niedling, Krekeler, Fuchs, Wagner, & Koppenhagen, 1973). In both cases, no effects were noted below 1.5 ppm of NO_2.

The animal studies that have been done have focused mainly on the more toxic NO_2. NIOSH, in fact, only lists four animal studies investigating the effects of NO. All four, however, used concentrations (range 175 to 20,000 ppm) far in excess of those experienced in railroad operations. In the same report, NIOSH lists 62 animal studies on NO_2. Of those 62 studies, only 26 investigated NO_2 at concentrations below 2 ppm. Of these 26 low-level exposure studies, 11 used continuous multiday exposure schedules rather than intermittent or short-term (8 hours or less) exposures. The 15 intermittent or short-term exposure studies are summarized in Table 5 as taken from NIOSH (1976, pp. 183-189).

Although, at the outset of this section, it was indicated that animal studies would not be emphasized, the virtual lack of human data on the effects of NO_x makes it necessary to review them. Recall, however, that generalizations from animals to humans is tenuous and should be done with caution.

In general, it appears that concentrations in the range expected under worst-case railroad conditions cause some micro- and macroscopic changes in the pulmonary system of a variety of laboratory animals and increases the likelihood of mortality from deliberate infection of klebsiella pneumoniae. Whether like changes would occur in humans exposed to similar concentrations is unknown, however.

Table 5

Summary of Effects of Low-level Intermittent or Short-term Exposure to NO_2 in Experimental Animals

Concentration (ppm)	Species	Duration	Type of Exposure	Dependent Variables	Results	Reference
1	Dogs, mice, rabbits, guinea pigs, rats, hampsters	up to 18 minutes	6 hrs/day, 5 days/wk	Macro/microscopic changes of the pulmonary system.	Dogs exposed at 1 ppm for 1 year had moderately dilated alveolar ducts and sacs which contained some edematous fluid and an occasional macrophage. After 18 months, some thickening of alveolar septa and chronic inflammatory cells were noted.	Wagner, Duncan, Wright, & Stokinger (1965)
2	Rats	43 days	1 hr/day	Microscopic changes in lung tissue.	No increase in lung weight, loss of cilia, hypertrophy, and focal hyperplasia noted after 3 days of exposure.	Stephens, Freeman, & Evans (1972)
1.9 - 7.0	Mice	4 hours	—	Antibacterial activity.	No decreased bacterial activity in animals exposed to less than 7.0 ppm.	Goldstein, Eagle, & Hoeprich (1973)
1.5, 2.5, 3.5	Mice	2 hours	—	Mortality due to pneumonia	No significant increase below 3.5 ppm.	Ehrlich, Henry, & Fenters (1970)
.5 - 3.5	Mice	9 minutes	6 hrs/day, 5 days/wk	Mortality due to pneumonia.	Significant increase in mortality in animals exposed intermittently for 1 month at .5 ppm.	Ehrlich (1966)
.1 - 2	Mice	3-1/2 - 7 min	.5 ppm with 1 hr peaks at 2 ppm 5 days/wk, or .1 ppm with 3 hr peaks of 1 ppm 5 days/wk.	Cellular morphology of lungs and alveolar macrophages.	Cell counts, macrophage viabilities at isolation and oxygen consumption of macrophages unaffected. In vitro phagucytic activity reduced in animals exposed at .5/2 ppm for 3.5 or 7 months. Changes in morphology of of macrophages noted in animals exposed at .5/2 ppm. No changes in .1/11 ppm groups.	Aranyi & Port (1974)
.5 - 2.0	Mice	up to 40 weeks	5 days/wk at .5 ppm with 1 hr peaks at 2.0 ppm	Immune response.	No difference between experimental and control animals in HI antibody titers. SN titers significantly depressed in .5/2 animals. Significant increase in IgA, IgM, IgG, and IgG2 immunoglobin levels in .5/2 animals.	Ehrlich, Silverstein, Maigetter, & Fenters (undated)

Note: From National Institute for Occupational Safety and Health. Criteria for a recommended . . . . Occupational exposure to oxides of nitrogen. HEW Publication No. (NIOSH) 76-149, March 1976.

Table 5 (Continued)

Concentration (ppm)	Species	Duration	Type of Exposure	Dependent Variables	Results	Reference
1	Guinea pigs	180 days	8 hrs/day	Macro- and microscopic scopic changes in lung. Hematologic urinary and immunologic changes.	Evidence of chronic respiratory disease such as bronchitis bronchopneumonia, extravasation of blood in lungs, and fuci of emphysema. Urinary hydroyproline and acid mucopolysac-charides were increased. Decreased serum proteins, immunoglobulins and weight gain.	Kosmider, Ludyga, Misiewicz, Drodz, & Sagan, 1972)
1	Rabbits	1 hour	—	Changes in protein structure of of lung tissue.	Peak shift in absorbance spectrum immediately after exposure, but returned to normal 24-48 hours after exposure.	Buell, Tokiwa, & Mueller, 1966)
1	Rats	1-6 days	4 hrs/day	Changes in lung lipid structure.	Absorption spectra indicative of dienne conjugation.	Thomas, Mueller, & Wright (1967)
.5 to 1	Rats	1 hr at 1 ppm 4 hrs at .5 ppm	—	Changes in mast cells of lung.	Exposure at 1 ppm resulted in loss of cytoplasmic granules, rupture, and reduction in number of mast cells. Exposure at .5 ppm resulted in degranulation of mast cells.	Thomas et al. (1967)
.2 - 1 in combination with .2-2 ppm NO	Dogs	4.5 years	16 hrs/day	Cardiovascular changes.	No significant effects.	Block, Lewis, Busch, Orthoefer, & Stara (1972)
.1 - 1 in combination with .1-2 ppm NO	Dogs	18 months	16 hrs/day	Pulmonary function.	No change in single breath CO diffusing capacity, dynamic pulmonary compliance, or total pulmonary resistance.	Vaughan, Jennell, & Lewis (1969)
.5	Mice	3-12 months	6, 18 hrs/day	Alveolar size.	Lung alveoli expanded in all mice exposed, compared to controls.	Blair, Henry, & Ehrlich (1969)

Note: From National Institute for Occupational Safety and Health. Criteria for a recommended standard . . . . Occupational exposure to oxides of nitrogen. HEW Publication No. (NIOSH) 76-149, March 1976.

Table 5 (Continued)

Concentration (ppm)	Species	Duration	Type of Exposure	Dependent Variables	Results	Reference
.5	Mice	1-2 months	6, 18 hrs/day	Mortality rate of bacterial clearance, lactic dehydrogenase (LDH) resulting from exposure to pneumoniae.	Reduced rate of clearance, LDH snift from anaerobic to aerobic bands. Increase in mortality for 6 month exposure.	Ehrlich & Henry (1968)

Note. From National Institute for Occupational Safety and Health. Criteria for a recommended standard . . . . Occupational exposures to oxides of nitrogen. HEW Publication No. (NIOSH) 76-149, March 1976.

Sulfur Dioxide

Here, as with the effects of oxides of nitrogen, little data exists on the effects of SO_2 on humans. Studies use small sample sizes, with a study reporting a sample size of 10 being considered a major contribution. Accepting our worst-case 8-hour TWA SO_2 railroad exposure as 1.0 ppm, we find only a handful of studies which assess the effects of such low concentrations on humans.

Sim and Pattle (1957) found little change in airway resistance (constriction) below 5 ppm for their maximum exposure time of 60 minutes. They found that subjects with histories of allergy showed evidence of airway constrictions. Frank, Amdur, Worcester, and Whittenberger (1962) found only 2 out of 11 subjects showing a significant increase in flow resistance at exposure of 1 ppm for 30 minutes. One of the two subjects who showed a significant increase in flow restriction had the highest nonexposure resistance, suggesting prior respiratory difficulties. Interestingly, Frank, Amdur, and Whittenberger (1964) found that when SO_2 is administered twice with a 15-minute period of clean air between exposures, the response to the second exposure is less than the response to the first. This suggests that adaptation to repeated exposure may occur.

Tomono (1961) reported that the lowest level of SO_2 that could induce broncho-constriction in healthy males was 1.6 ppm. In support of this, Burton, Corn, Gee, Vassallo, and Thomas (1968) found no significant increase in resistance or dynamic lung compliance in subjects exposed to 1 ppm for 30 minutes. Anderson, Lundquist, et al. (1974), in addition, found no change in nasal mucous flow rate from exposure to 1 ppm for 6 hours.

It seems, therefore, that SO_2 concentrations expected in worst-case railroad operations are probably of no consequence to respiratory functioning. However, it must be pointed out that the studies upon which this conclusion is based all involved short-term exposures.

One effect of SO_2 which has found consistent support in the literature (Dubrovskaya, 1957; Bushtueva, 1961) is that low concentrations (below 1 ppm) cause an increase in sensitivity to light during dark adaption. The effect can only be seen under tightly controlled laboratory conditions, however. The practical significance of this phenomenon is indeterminate.

Particulate Matter

Particulate matter may elicit a pathological or physiological response in the respiratory system in at least three ways. First, particles may be intrinsically toxic. According to the National Air Pollution Control Administration (1969), however, carbon black, the principle particulate matter in diesel exhaust, "produces little major damage to the respiratory

system." This is especially true at the low 8-hour TWA concentrations anticipated in worst-case railroad operations (1.7 mg/m^3).

Second, the presence of inert particles may interfere with the clearance of other airborne toxic materials. The relationships here can be complex. For example, Pattle and Burgess (1957) found that, with mice and guinea pigs, the prior inhalation of smoke actually reduced the toxicity of SO_2, but the simultaneous inhalation of smoke and SO_2 increased the toxicity.

Third, particles may act as a carrier of toxic materials. It appears that, with diesel emissions, this is the main attribute of particulate matter. The materials absorbed on the particles are largely hydrocarbons. More will be said concerning hydrocarbons in section 3.2.6.

The principle danger, in railroad operations, from particulate matter does not appear to be respiratory in nature. In fact, Stokinger (1975) concluded, after a review of the literature, that:

> It is wholly possible that the effects of heavy cigarette smoking in underground diesel workers will have more serious effects, including cancer, than inhaling comparatively dilute diesel smoke.

Hydrocarbons and Aldehydes

The most authoritative summary source on the toxic effects of hydrocarbons and aldehydes is that prepared by the National Air Pollution Control Administration (1970c). Unless otherwise indicated, the major conclusion concerning hydrocarbons and aldehydes comes from this source.

The National Air Pollution Control Administration categorizes hydrocarbon compounds into three classes. Aliphatic hydrocarbons are biologically and biochemically inert. Except for some anesthetic properties at extremely high concentrations (over 500 ppm), they produce no detectable, functional, or subclinical alternations. Alicyclic hydrocarbons, the second class, act as an anesthetic and central nervous system depressant. They are, however, of low toxicity and do not tend to accumulate in body tissues. Here again, high concentrations are required before effects are observed.

The third class, aromatic hydrocarbons, is biochemically active with no effect on respiratory function or eye irritation being found below 25 ppm. Some aromatic hydrocarbons, however, are considered carcinogenic, especially benzo (a) pyrene (also called 3-4 benzprene). Cottini and Mazzone (cited in Falk, Kotin, & Mehler, 1964) applied a 1 percent benzene solution of benzo (a) pyrene daily to the skin of 26 humans. They observed erythema, pigmentation, desquamation, and vearucae formation with infiltration. After cessation of treatment, however, the damage was reversed. Kotin, Falk, and Thomas (1955) painted the skin of mice three times per week with a solution of diesel extract containing the following aromatic hydrocarbons: pyrene; 3, 4, benzprene; 1, 2, benzprene; 1, 12 benzperylene; anthanthrene; and coronene. The mice showed immediate widespread systematic reactions including liver and lung damage and skin tumors. In the same

year, however, Clemo and Miller (1955) reported that diesel bus smoke extract did not cause cancer in mice. Battigelli (1963a) states that studies that have tried to cause 3-4 benzprene to form cancer in organs other than the skin, using procedures more comparable to human exposure, specifically inhalation, have failed systematically.

It appears that even under our worst-case railroad exposure condition of 10.7 ppm hydrocarbon, there is little or no health or safety effects anticipated from hydrocarbons. Part of the 10.7 ppm will be made up aliphatic and alicyclic hydrocarbons, which are harmless at even 10 times the worst-case concentration. What small part of the 10.7 ppm is made up of aromatic hydrocarbons seems, from the published literature, to pose no health effects.

Aldehydes, a class of oxygenated hydrocarbons, contain many individual compounds that differ in toxicity. The worst-case railroad concentration (5.3 ppm) contains unknown proportions of various aldehydes.

Unfortunately, as with the other pollutants discussed, there have been almost no studies on the long-term effects of aldehydes. Much of the available information on the toxicity of aldehydes pertains either to the effects from single acute exposures of animals, or to industrial exposures to high concentrations. The principle effect of low concentrations of aldehydes is irritation of the mucous membranes of the eyes and upper respiratory tract, particularly the nose and throat. Many of the aldehydes, however, are irritating only at concentrations above 200 ppm (e.g., propionaldehyde, butyraldehyde, isobutyraldehyde); others are just detectable at 50 ppm (e.g., acetaldehyde). The two aldehydes that are most irritating and have been the focus of most of the studies dealing with aldehydes are formaldehyde and acrolein.

Although in Section 2, the worst-case 8-hour TWA values were not computed for formaldehyde and acrolein, peak values reported in the literature were given (.04 ppm acrolein, .20 ppm formaldehyde). Even at 10 times these concentrations, neither formaldehyde nor acrolein pose any health or safety hazard.

The principle effect of formaldehyde on humans appears to be irritation of the mucous membranes of the eyes, nose, and upper respiratory tract. Symptoms include lacrimation (watering eyes), sneezing, coughing, dyspnea, a feeling of suffocation, rapid pulse, headache, weakness, and fluctuations in body temperature. Several reports indicate that irritation of the eyes and upper respiratory tract can first be detected at formaldehye levels of .01 to 1 ppm. On the other hand, Fasset (1963) reported no discomfort until the level reached 2 or 3 ppm, at which time a very mild tingling sensation was detected in the eyes, nose, and posterior pharynx. He also reported that some tolerance occurs with larger exposures, thus suggesting that repeated 8-hour exposures are possible.

Acrolein is even more toxic than formaldehyde. Its vapors are extremely irritating to the eyes and respiratory tract. Symptoms from inhalation include lacrimation, swelling of the eyelids, shortness of breath, pharyngitis, laryngitis, bronchitis, oppression in the chest, somnolence,

and asthma. Fortunately, however, these effects occur only at concentrations many times that of the peak railroad exposure level of .04 ppm. Concentrations as low as .25 ppm have caused moderate eye and nose irritation. Lacrimation results when concentrations reach .67 ppm.

Summary

The published literature on the long-term intermittent exposure to low levels of diesel exhaust is meager. What little there is, however, points consistently to the conclusion that such exposure histories probably pose no adverse health effects. Exposure to diesel exhaust can cause eye irritation that may have some safety implications as well as being a short-term irritant to those working in the situation.

One manifestation of diesel exhaust, which to this point has been ignored, is its characteristically bad odor. Although not a health hazard in the traditional sense, odors can be quite unpleasant and irritating to people. The following section of this report will review what little is currently known about diesel odor.

DIESEL ODOR

Cause of Diesel Odor

Effective chemical or physical methods for the measurement of odor have not been developed, and, therefore, the human nose must play a significant role in all odor studies. This adds a subjective component to odor analysis. The Turk kit has become the standard for odor studies (Matula, 1973). The kit contains a number of different standard odors classified as (a) burnt/smoky, (b) oily, (c) pungent/acid, and (d) aldehydic/aromatic.

There have been many studies attempting to isolate the odor components of diesel exhaust (e.g., Spindt, Barnes, & Somer, 1971; Linnell & Scott, 1962; Dietzmann, Springer, & Stahman, 1972; Caragay, Funkhouser, Kendall, Leonardos, & Levins, 1971), but two are seen as the most comprehensive and probably the most accurate. The two studies are often referred to as the Arthur D. Little study (Levine, 1972) and the Illinois Institute of Technology (IIT) study (O'Donnell & Dravnieks, 1970).

The IIT study reported that only about 100 compounds, out of over 1000 compounds isolated from diesel exhaust, were odor relevant. Based on the IIT work, several classes of compounds have been eliminated as important odorants. Lower series alcohols, simple alkyl benzenes, and alkanes were found generally at concentrations below their high odor thresholds. The odor relevant compounds and their specific odor characteristics isolated by IIT are listed in Table 6 as taken from Matula (1973).

The Arthur D. Little (ADL) study characterized diesel odor into two distinct components: (a) oily-kerosene and (b) smoky-burnt. ADL has reported more progress on determining the classes of compounds responsible for the oily-kerosene odor than for the smoky-burnt odor. Table 7 lists the relevant compounds isolated, contributing to each odor characteristic, by ADL as adapted from Matula (1973). ADL concludes that neither the most abundant exhaust aldehydes, sulfur compounds, nor nitrogen compounds contributed to diesel odor.

A comparison of Tables 6 and 7 reveals little in common. This is due to differences in experimental procedure; the multitude of variables that affect diesel odor components such as engine type and condition, load, and fuel; the subjectivity involved in odor determination; and the unreliability of the techniques used to isolate hydrocarbons. About all that can be said is that many compounds contribute to diesel odor, but, at the present stage of knowledge, only tentative lists can be generated.

One thing is certain: Diesel exhaust can be odorous, and some people are more sensitive to the odor than are others (Randolph, 1954). Further, although no evidence in the literature was found, it is suspected that, over a period of time, there probably is an adaptation to the odor. This is a common experience of people working on odorous jobs.

Table 6

Compounds Identified by IIT as Contributing to Diesel Odor (Matula, 1973)

Compound	Elemental Composition	Odor Characteristics
Ethylbenzene	C_8H_{10}	Pungent
p-Xylene	C_8H_{10}	Pungent
m-Xylene	C_8H_{10}	Pungent
C_4-substituted benzene	—	Pungent-fuel
Trimethylbenzene	C_9H_{12}	Pungent
p-Ethylstyrene	$C_{10}H_{12}$	Unpleasant-burnt
C_5-substituted benzene	—	Pungent-burnt
Allyltoluene	$C_{10}H_{12}$	Burnt-pungent
C_6-substituted benzene	—	Burnt-pungent
Dimethylcumene	$C_{11}H_{17}$	Strong-burnt-pungent
Naphthalene	$C_{10}H_8$	Naphthalene
Methylnaphthalene	$C_{11}H_{10}$	Naphthalene
Methylindan	$C_{10}H_{12}$	Burnt-fuel
Dimethylindan	$C_{11}H_{14}$	Burnt-fuel
Methyltetralin	$C_{11}H_{14}$	Burnt-rubber
Possibly 1,7-octadiyne	C_8H_{10}	Pungent-burnt
Substituted cyclohexane and cyclohexene	—	Pungent-burnt
Cyclic olefin or alkyne	—	Unpleasant-burnt
Trimethylthiophene	$C_7H_{10}S$	Strong-foul
C_2-substituted benzothiophene	—	Burnt
Ethanal	C_2H_4O	Sweet
n-Propanal	C_3H_6O	Sweet
n-Butanal	C_4H_8O	Unpleasant-aldehyde
n-Pentanal	$C_5H_{10}O$	Unpleasant-aldehyde
n-Hexanal	$C_6H_{12}O$	Aldehyde
n-Heptanal	$C_7H_{14}O$	Citrus-aldehyde
n-Octanal	$C_8H_{16}O$	Aldehyde
Benzaldehyde	C_7H_6O	Cherry-pungent
C_2-substituted benzaldehyde	—	Pleasant-sweet
Ethylbenzaldehyde	$C_9H_{10}O$	Sweet-floral
Tolualdehyde	C_8H_8O	Pleasant-floral
Acetone	C_3H_6O	Pleasant
Possibly acetoin and methylallylketone	$C_4H_8O_2$	Strong-foul
Acetophenone	C_8H_8O	Pleasant-floral
Methylacetophenone	$C_9H_{10}O$	Pleasant-sweet

Table 7

Compounds Identified by Arthur D. Little as Contributing to Diesel Odors

Compound or Structure Class[a]	Elemental Composition or C-range	Odor Characteristics
OILY-KEROSENE ODOR:		
Indan and tetralin compounds		
Methylindan	$C_{10}H_{12}$	Irritation
Tetralin	$C_{10}H_{12}$	Rubbery sulfide
Dimethylindan	$C_{11}H_{14}$	Kerosene
Methyltetralin	$C_{11}H_{14}$	Naphthenate
Dimethyltetralin	$C_{12}H_{16}$	Kerosene
Trimethylindan	$C_{12}H_{16}$	Kerosene, irritation
Alkyltetralin	$C_{12}H_{16}$	Kerosene
Trimethyltetralin	$C_{13}H_{18}$	Naphthenate
Alkyltetralin	$C_{13}H_{18}$	Irritation, kerosene, pungent/acid
Indene, acenaphthene, and benzothiophene compounds		
Alkylindene	$C_{12}H_{14}$	Heavy oil
Naphthalene compounds		
Monomethyl	$C_{11}H_{10}$	Feel (mothballs), irritation
Alkyl-benzene compounds		
—	$C_{10}H_{14}$	Rubbery
—	$C_{10}H_{14}$	Musty oily
—	$C_{10}H_{14}$	Tarry
—	$C_{11}H_{16}$	Strong
—	$C_{11}H_{16}$	Oily metallic
—	$C_{11}H_{16}$	Oily
SMOKY-BURNT ODOR:		
Alkenone	$C_5 - C_{11}$	Oxidized oily
Furan	$C_6 - C_{10}$	Irritation, pungency
Dieneone	$C_9 - C_{12}$	Sour, oxidized oily
Furfural	$C_6 - C_7$	Burnt, oily
Methoxybenzene[b]	$C_8 - C_9$	Smoky, pungency
Phenol[b]	$C_7 - C_{12}$	Burnt, smoky, particle size, pungency
Benzaldehyde[b]	$C_7 - C_{10}$	Burnt, smoky, metallic, pungency
Benzofuran	$C_8 - C_9$	Particle size, smoky
Indanone[b]	$C_9 - C_{18}$	Metallic, smoky, sour
Indeneone	$C_9 - C_{10}$	Linseed-oily, sour
Naphthol[b]	$C_{10} - C_{14}$	Smoky, burnt
Naphthaldehyde	C_{11}	Smoky

[a] Includes hydroxy and methoxy derivatives.

[b] Most abundant classes.

Health and Safety Consequences

The primary result of exposure to malodors is nausea and headache. From Section 3, we saw there was probably no long-term health effects from exposure to diesel exhaust. Nausea and headache, although in the short-term are unpleasant, probably present no real health hazard. The question then becomes, what effect does nausea and headache have on performance that might be related to railroad safety? Unfortunately, there is vitually no data in the published literature related to this. A computerized literature search was conducted (Lockheed Data Systems) of 13 bibliograhic data bases (including ERIC, BIOSIS, NTIS, Psychological Abstracts, Dissertation Abstracts, Enviroline, Pollution Abstracts, and APTIC) containing over 4,000,000 references, retrieving all items that even mentioned the terms nausea or headache in the title, keywords, or abstract. Although almost 1000 items were identified, only two were even remotely relevant. All the other studies dealt with the causes or the treatment of nausea or headache. In essence, all the studies treated nausea or headache as dependent variables. No study treated them as independent variables in order to assess their effects on performance. The only two studies that are of any value to the problem at hand were studies that were assessing the effects of altitude on a variety of dependent variables, including performance variables and nausea and headache.

The first of these studies, Fine and Kobrick (1969), found that increases in visual acuity and brightness sensitivity from sea level to altitude were inversely related to severity of headache at altitude. This means that increases were smallest when headaches were most severe. Unfortunately, we do not know whether the severity of the headache itself caused the reduced visual acuity and sensitivity, or that both are simply symptoms of mild hypoxia at altitude.

The second study (Carver & Winsmann, 1968), fortunately, yields a less ambiguous conclusion. Abrupt exposure to an elevation of 13,000 feet did not affect aspects of physical proficiency other than running proficiency, nor did it affect cognitive functioning. It did produce headache, dizziness, and nausea. What this means is that, even with headache, dizziness, and nausea present, there was no effect on physical or cognitive performance.

It appears that, based on published literature, vitually nothing can be said about the safety implications of headache or nausea.

INTERVIEW AND OTHER DATA OBTAINED

Extent of the Problem

During the course of this study, several sources of data were made available which could shed some light on the extent of the diesel emission problem in railroad operations. Each of these sources are discussed in this section, but, before proceeding, several words of caution are in order. All of the sources of information were collected in an unsystematic and potentially biased fashion. In all cases, the suppliers of the information knew that diesel emissions and other environmental pollutants were the focus of the study. Further, in all cases, no attempt was made (a) to randomly or representatively sample from a population, or (b) to ascertain the population size or characteristics in order to convert the sample to a proportion of the population. In short, the data sources to be discussed are methodologically lacking and afford only a limited basis for making projections to populations. Nevertheless, all the sources together paint a somewhat consistent picture of the diesel emission problem. Together they suggest that, all in all, it is, in terms of frequency and extent, a relatively minor problem.

Interviews

As part of this project, interviews were held with six union officials from both the United Transportation Union (UTU) and the Brotherhood of Locomotive Engineers (BLE) and ten operating train crewmen.

All interviewees were told that the purpose of the interview was to gather information regarding environmental irritants in operating crews' jobs. Specific things were listed to focus the discussion such as noise, vibration, fumes and odors, and temperature. If the respondents did not mention fumes or odors, they were specifically questioned about them.

The six union officials, all of them in positions that would be in the channel through which complaints would be filed, did not feel that exhaust fumes were a very big problem. Representative quotes included, "once in awhile, not all the time," "not ordinarily a big problem," and "not to an obnoxious extent." One official, with over 30 years of operating experience, had never personally experienced a bad fume problem.

Several recurring themes were noted in the union officials' interviews. Noise was considered a big problem, and, in several interviews, it was hard to get the official to discuss fumes as a problem. Poor maintenance, especially on older units, was consistently given as the crux of the problem. One official said of the railroad companies, "If they maintained their equipment as well as they should maintain their track, we wouldn't have any problem," He also indicated that the "men are sometimes their own worst enemy" because they fail to complain to the union. It was acknowledged that the very nature of the industry makes maintenance a problem. Being a "moving industry," an engine or caboose on which a complaint has been registered may be out of state the next day, making it difficult to affect repairs. Most of the officials, on balance, however, felt that the railroad companies did a good job of responding to complaints and work order requests.

The interviews with the operating crews revealed the same general picture as was seen with the union officials. One engineer with 30 years of experience said he had to switch engines due to fume problems "maybe six times in his 30 years." Two other engineers and a conductor, all with over 30 years' experience, said they have never had any bad experiences with smoke or fumes.

Letters and Union Files

During the course of this study, the United Transportation Union published a short note to their membership (UTU News, April 23, 1977) requesting them to send information concerning environmental irritants to our project office. This was not a planned part of the project, and the article was not initiated by the project manager. We did, however, ask both the UTU and BLE to send us any relevant information from their files, which they did.

The UTU and BLE have a combined membership of almost a quarter of a million, with the bulk of those belonging to the UTU (approximately 200,000). A total of 14 letters were received in response to the UTU News article. Only 10 of them mentioned exhaust, fumes, or odors. The union files contained a total of 23 items dating back to 1975, in the case of the UTU, and 1973, for the BLE. Of these 23 items, only 11 mentioned fumes, exhaust, or odor as a problem.

This small number of complaints may be due, in part, to a general reluctance to complain among union members or to a feeling that complaining really has little impact on things. There is some evidence in support of this. As part of the study carried out by Hobbs et al. in 1976 on railroad diesel emissions, articles were published in the UTU News (8 March 1975) and the BLE publication Locomotive Engineer (7 February 1975) requesting information relevant to fumes and other irritants in locomotive cabs and cabooses. In addition, the unions sent relevant information from their files. A total of only 84 items (letters, union file items, etc.) were received, 19 from the BLE and 65 from the UTU. A few of these related severe disabling experiences with exhaust and fumes, but most were more on the order of general complaints. Besides engine exhaust, several other sources of pollution and odor were mentioned. These are (a) fumes from cab and/or caboose heaters, (b) oil spills on floor, (c) oil particles in brake valve exhaust, (d) toilet odors, (e) tobacco smoke, and (d) refrigerator car exhaust.

One is forced to conclude from these data sources that diesel fumes and odors are not a widespread problem but can become acute on occasion.

Inspection Reports

Some of the union officials we interviewed supplied us with locomotive inspection reports and/or caboose reports covering a short period of time during their jurisdiction. We have no idea how representative the samples are, or whether they represent all the reports filed during that period or only a select sample.

Ninety-six (96) locomotive inspection reports, representing 86 different locomotives and covering a 3-month period, revealed only three incidents in which oil or fuel leaks were mentioned. The three most common complaints were broken or missing sun visors; dirty, odorous, and/or broken toilets; and grease around brake valve and speed recorder (with no mention of odor, however).

A 2-month sample of caboose reports for one railroad yielded only six reports, out of a total of 42, mentioning oil fumes from stoves. The most common complaints were dirty, odorous, and/or broken toilets (32 reports) and general overall dirtiness (29 reports).

Caboose reports (28) covering a 10-month period from another railroad showed only three reports mentioning fumes or smoke from stoves. The most common complaints were again dirty, odorous, and/or broken toilets (11 reports) and no lights (12 reports).

From these inspection reports, it appears that the major sources of pollution are dirty, odorous toilets rather than exhaust or oil fumes.

The evidence from all three data sources--interviews, letters and union files, and inspection reports--points to the conclusion that diesel exhaust emissions are not a widespread or frequent problem in the railroad environment.

CONCLUSIONS

From the published literature reviewed and the data sources analyzed, we must conclude that diesel emissions, as experienced by operating railroad crews, are not a widespread or frequent problem and, except in rare circumstances, probably present no long-term health hazard. Short-term, infrequent occurrences of burning eyes, headache, and nausea may occur but the safety consequences of such symptoms could not be estimated.

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